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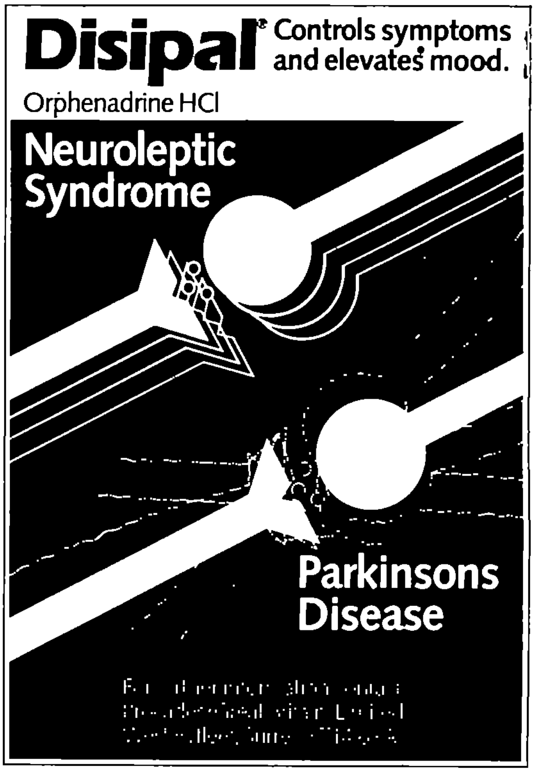

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Kraepelin's work on psychiatric nosography and systematics still continues to be of topical interest nowadays. Psychiatric diagnosis throughout the world is based on his studies, which was also of outstanding significance for the development of psychiatric research. Current areas of investigation, such as psychopharmacology, psychiatric epidemiology, transcultural psychiatry and psychiatric genetics were all either founded, inspired or decisively supported by Emil Kraepelin.

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This book is a comprehensive report of the present state of knowledge in the main areas of schizophrenia research. It presents a critical evaluation of this knowledge with the aim of formulating new working hypotheses and research strategies in the search for the causes of the disease.

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This monograph compares the biographies of young adult inmates with a random sampling of young men from the general population and considers the results in the light of other studies of similar nature, primarily from the Anglo-American literature. For the practitioner, such an approach allows a differential assessment of the individual offender in the context of his social relationships with conclusions regarding prognosis and therapeutic effects.

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This volume asks if there are ways to intervene early in a child's life that might reduce, at a reasonable cost, the probability of his becoming a serious delinquent. The results of some small-scale social experiments have been published suggesting that certain kinds of preschool education and parent training might have desirable and lasting effects.

Psychological Treatment of Mental Illness

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1987. Approx. 12 figures. Approx. 245 pages. (Research in Criminology). Hard cover DM 98,–. ISBN 3-540-96483-5

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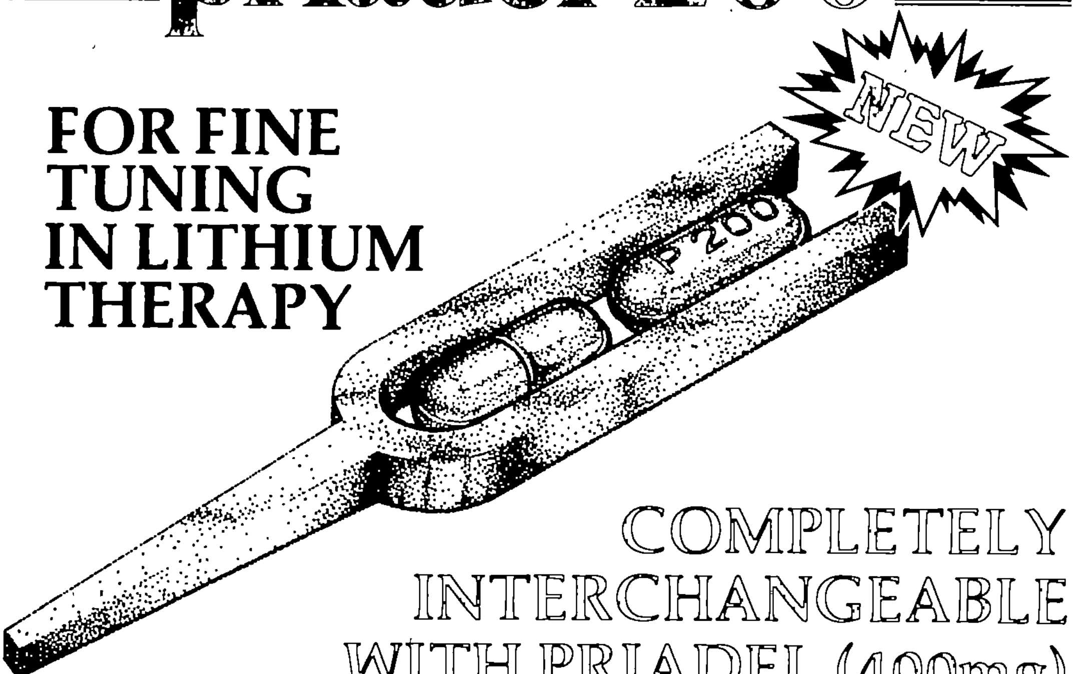

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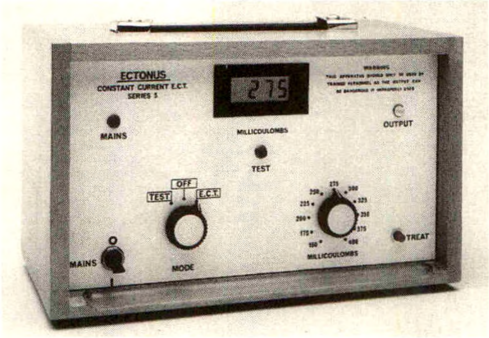

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British Journal of Psychiatry (1987), **151**, 1–8

Assessing the Functional Value of Relatives' Knowledge about Schizophrenia: A Preliminary Report

C. BARROWCLOUGH, N. TARRIER, S. WATTS, C. VAUGHN, J. S. BAMRAH and H. L. FREEMAN

An instrument for assessing and evaluating what relatives know about schizophrenia was evaluated as both a pre- and a post-test for an educational programme. The Knowledge About Schizophrenia Interview (KASI) places emphasis on the functional value of the reported knowledge rather than on the recall of information; it is quick, easy to administer, can be rated reliably, and has face-validity for the relative. The educational programme increased scores from pre-test to a post-test one week after the programme. Relatives with high criticism ratings on the Camberwell Family Interview had lower scores at both tests. Relatives of less chronic patients showed lower scores at pre-test and acquired significantly more information from the programme, while relatives of more chronic patients were less influenced by the information sessions.

Several recent studies (e.g. Berkowitz *et al*, 1984; McGill *et al*, 1983) have reported on giving information about schizophrenia to relatives, generally as part of larger psychosocial interventions that attempt to reduce relapse rates of patients suffering from schizophrenia who return to live with their families (Goldstein, 1981; Barrowclough & Tarrier, 1984). It can be argued that both relatives and people suffering from schizophrenia should have, as their right, access to information about the condition; but in addition, informing carers about schizophrenia may in fact contribute to an improvement in the condition.

The index of Expressed Emotion (EE) has been important in studying schizophrenics' families and relapse (Leff & Vaughn, 1985). Relatives are measured as being "high-EE" or "low-EE" on the basis of the number of critical comments and the extent of emotional over-involvement or hostility shown to the patient during a recorded, semi-structured Camberwell Family Interview (CFI) (Vaughn & Leff, 1976*b*). A number of studies have found an association between relapse of schizophrenia within nine months of discharge and the EE of the key relative residing with the patient (Brown *et al*, 1972; Vaughn & Leff, 1976*a*; Vaughn *et al*, 1984). Patients returning to live with high-EE relatives had significantly higher relapse rates than those going to low-EE relatives, independent of all other factors assessed.

It has been suggested (Vaughn & Leff, 1981) that high-EE relatives tend to feel that patients could control their symptomatic behaviour, and take a relatively unsympathetic view of the illness, whereas low-EE relatives believe that the patients suffer from a legitimate illness and cannot control certain behaviour. Since Brown *et al* (1972) suggested that a major contribution to high-EE was lack of knowledge by the relative about schizophrenia, it has been hypothesised that educating relatives, by giving them information about schizophrenia, might lead to reduced criticism and hostility and a lowering of EE (Berkowitz *et al*, 1984; McGill *et al*, 1983). However, the study of Berkowitz *et al*, failed to support the hypothesis that high-EE relatives know less about schizophrenia than low-EE relatives.

Tarrier & Barrowclough (1986) have discussed some of the issues involved in giving relatives information about schizophrenia and have questioned the utility of measuring acquired information *per se*. This article describes the development of an instrument designed to assess the *functional value* of the relative's knowledge about schizophrenia, which was part of a larger study investigating the effects of psychological intervention in families, including an educational component.

Method

Patients

All patients admitted to the acute wards of a large psychiatric hospital and the psychiatric ward of a general hospital within one health district were screened. Patients were included within the study if they met the following criteria:

1. Having a diagnosis of schizophrenia elicited by use of the Present State Examination (Wing *et al*, 1974) by a trained interviewer (JB, CB, NT, SW, HF).
2. Age on admission 16–65 years.

3. Residing with someone (e.g. parent, spouse, sibling, friend, partner) for at least three months prior to admission, and returning to this residence on discharge.
4. Having no evidence of an organic condition to explain the psychopathology.

Relatives

On the patient being recruited into the study, the relatives were contacted and the CFI was carried out and audiotaped by a trained interviewer (CB, NT, SW). Relatives were allocated to the education programme in accordance with the stratified random design of the larger intervention study; 24 relatives (related to 17 patients) were assessed and given the education programme. Relevant characteristics of these relatives and patients can be seen in Table I. All relatives allocated to the education programme received both sessions described below.

The education programme

Relatives were given a hospital appointment to attend an education session during the first week after discharge (Session 1) and another one week later (Session 2). Two relatives preferred the sessions to be given at home; this was arranged.

Session 1

The relative was interviewed and audiotaped, using the *Knowledge about Schizophrenia Interview* (KASI): if more than one relative of the patient attended, each relative was interviewed separately. All relatives aged 16 years or above who lived with the patient were encouraged to attend.

After the pre-test, relatives were given an information booklet (Barrowclough *et al*, 1986); the content of the booklet was worked through with one of three interviewers (CB, NT, SW). Particular attention was paid to:

(i) areas of information highlighted as being 'incorrect' or not known during the pre-test
(ii) describing the particular patient's psychotic symptoms, elicited by the PSE.

If more than one relative attended, the education part of the session was conducted with all the relatives together. The patient was interviewed simultaneously but separately, and given the same information as the relative(s). Questions from relatives were encouraged and prompted, taking into account information from the pre-test interview and including such questions as: "You said earlier that you thought X might be better off without medication. What do you think about that now?" or "You said earlier that you didn't think stress had anything to do with Y's illness; what do you feel about this now?".

In the latter part of the session, patient and relative(s) were seen together, and further questions encouraged.

At the end of the session, the relatives were given an appointment for Session 2, one week later; they were asked to take the booklet away with them and to read it thoroughly. (The booklet is written in simple terms and has been assessed by the Flesch reading scale (Flesch, 1948) as being understandable to 75% of the population.) Relatives were told that the main function of the subsequent session was to answer any further questions that they might have.

Thus Session 1 consisted of three stages:

(i) KASI pre-test (relatives tested individually)
(ii) information giving and question/feedback (relatives together, patient separately)
(iii) further questions/feedback (relatives and patient together).

TABLE I

Characteristics of 17 schizophrenic patients and 24 relatives with whom they lived

Patients (n = 17)	
Male/Female	7M/10F
Living with:	
Parents	11
Spouse/partner	5
Child	1
Age (years):	
Mean	37
Median	39
Range	16–60
Number of previous hospital admissions:	
Mean	3.2
Median	3
Range	0–13
First admission	*n* = 2
Years since onset:	
Mean	6.6
Median	4
Range	0–24
Relatives (n = 24)	
Relationship to patient:	
Mother	9
Father	6
Spouse/partner	6
Daughter	2
Sibling	1
EE status	High 20/ Low 4
Reason for high-EE rating:	
Emotional over-involvement (EOI)	*n* = 5
> 5 critical comments (CC)	*n* = 3
EOI + CC	*n* = 3
EOI + CC + hostility	*n* = 3
CC + hostility	*n* = 6
Current/previous employment	
Unskilled	14
Skilled manual	2
White-collar	7
Professional	1

Session 2

There were two stages: (i) *Questions/feedback*, during which the relatives and the patient were seen together, and asked if they had any further questions or if there was any further issue they wished to discuss; and (ii) *KASI (post-test)* during which the relative(s) were interviewed individually and audiotaped.

This procedure for the educational programme—two sessions with a one-week interval—was adopted because:

- (i) Relatives occasionally became worried, or misunderstood the information they were given. An early second appointment allowed an opportunity to discuss any such problems.
- (ii) KASI assessment one week after the education session was thought to be more likely to assess longer-term information retention than a test given immediately after the education session.
- (iii) The one-week period allowed relatives and patients time to discuss the information and to read the booklet.
- (iv) The possibility of the outcome evaluation (KASI post-test) being affected by factors other than the education programme was minimised.

The KASI interview

The KASI and its rating criteria were designed in accordance with the following objectives:

1. Items should assess not just the presence or absence of information about schizophrenia, but the effects of that information on the relative's behaviour.
2. The items should be phrased simply, and the absence of academic or technical knowledge should not be penalised in the scoring.
3. Administration and scoring of the interview should be relatively quick and required the minimum of training.

As with comparable instruments (e.g. Berkowitz *et al*, 1984; McGill *et al*, 1983) the following aspects of the schizophrenic condition were covered:

1. Diagnosis
2. Symptomatology
3. Aetiology
4. Medication
5. Course and prognosis
6. Management

Each topic was covered in a different section which could be scored independently. Most questions were structured so as to require a "Yes/No/Don't know" choice, but the section, on management contained open questions. In selecting items, the following questions were borne in mind:

1. What information might have a beneficial influence on the relative's behaviour in relation to the patient?
2. Conversely, what perceptions, attitudes, and reported behaviour might have undesirable effects on the relative's behaviour in relation to the patient?

The interview was developed to permit the allocation of the relative's responses for each section to one of four categories:

1. *Negative value*: Relative reports information which may lead to potentially detrimental actions in regard to the patient's management.
2. *Neutral value*: Relative knows very little or nothing, but reports no information which would probably result in detrimental management.
3. *Positive value*: Relative reports information which may lead to potentially valuable actions in regard to the patient's management.
4. *Positive value and additional correct information*: Over and above the information for category 3, the relative demonstrates wider knowledge about the assessed aspect of the condition.

Some examples of how these criteria were applied are given in the Appendix at the end of this paper. Further details of the interview and the scoring criteria may be obtained from the authors (Barrowclough *et al*, 1985).

For each of the six sections of the KASI, the relative's responses were allocated a score on the 1–4 scale described above. Both interviews for all 24 relatives were scored by CB. Ten interviews were then selected randomly and scored independently by NT for reliability calculations.

Results

Reliability

The degree of inter-rater agreement between the two independent raters (CB and NT) for each of the six sections of the KASI is shown in Table II. These data are based on the two independent raters scoring ten randomly selected audiotaped interviews with ten different relatives (60 sections of interview). It was concluded that the scores obtained were of acceptable reliability and that this finding might be generalised from the random sample to the total population of audiotapes scored by CB.

1. KASI scores before/after education

A Wilcoxon matched-pairs test was performed on the data for all 24 subjects, comparing pre-test and post-test scores

TABLE II

Inter-rater agreement for each of the six sections in the audiotaped KASI

Section of interview	*Percentage agreement*	*Weighted κ coefficient*
1. Diagnosis	80%	0.70 ($P<.01$)
2. Symptomatology	80%	0.66 ($P<.01$)
3. Aetiology	100%	1.00 ($P<.01$)
4. Medication	80%	0.64 ($P<.01$)
5. Course and prognosis	90%	0.78 ($P<.05$)
6. Management	80%	0.66 ($P<.05$)

TABLE III

KASI scores before and after the educational programme. Relatives' responses were rated as 1 (negative), 2 (neutral), 3 (positive) or 4 (positive + well-informed)

Section of KASI	*Pre-Test Scores ($n=24$)*				*Post-Test Scores ($n=24$)*				*Wilcoxon pairs test (z scores)*	*McNemar test (χ^2)*
	Median	*Range*	*Score 1 or 2*	*Score 3 or 4*	*Median*	*Range*	*Score 1 or 2*	*Score 3 or 4*		
1. Diagnosis	2	1–4	$n=16$	$n=8$	3	1–4	$n=3$	$n=21$	3.3 ($P<0.0005$)	15.08 ($P<0.001$)
2. Symptomatology	2	1–3	$n=13$	$n=11$	3	1–4	$n=7$	$n=17$	2.4 ($P<0.008$)	7.2 ($P<0.01$)
3. Aetiology	2	1–2	$n=24$	$n=0$	2	2–4	$n=14$	$n=10$	2.93 ($P<0.002$)	12.1 ($P<0.001$)
4. Medication	3	1–4	$n=6$	$n=18$	3	1–4	$n=3$	$n=21$	1.52 ($P<0.06$)	3.2 ($P<0.08$)
5. Course & prognosis	2	1–4	$n=20$	$n=4$	3	1–4	$n=11$	$n=13$	2.93 ($P<0.002$)	11.11 ($P<0.001$)
6. Management	3	1–4	$n=11$	$n=13$	3	1–4	$n=5$	$n=19$	2.4 ($P<0.008$)	6.13 ($P<0.02$)
Total KASI score	13.5	7–18	$n=90$	$n=54$	17	8–21	$n=43$	$n=101$	3.85 ($P<0.0001$)	44.18 ($P<0.001$)

and using a one-tailed test of probability (we made the *a priori* assumption that there would be either an increase or no change in knowledge). The results (*z* scores) are shown in Table III. Scores for sections 1,2,3,5, and 6 increased significantly between tests, as did the total score (the sum of all the section scores). Scores on section 4 (medication) showed an increase which approached statistical significance.

A McNemar test was also performed to compare scores on pre- and post-test, for each section and for total scores; change from a score of 1 or 2 to 3 or 4 was tested. The results are presented in Table III, which shows a significant change from category 1 or 2 to 3 or 4 for total scores and for all sections except medication. Whereas 31 sections received a rating of 1 at pre-test, only 11 were rated 1 at post-test; 15 relatives had at least one section given a score of 1 at pre-test, compared to only seven at post-test.

2. Relatives' characteristics and KASI scores

(a) Critical/non-critical relatives

For the purposes of this analysis, ratings of hostility and emotional over-involvement were ignored. Relatives were divided into those who expressed six or more critical comments during the CFI, and so were rated 'high-EE' ($n=15$); and those who expressed five or less critical comments ($n=9$). Three Mann-Whitney U tests were carried out to compare total scores between the two groups at pre-test and at post-test, and change scores:

Comparison	*z*	*P (two-tailed)*
Pre-test total score	1.99	0.05
Post-test total score	2.28	0.02
Change scores	0.64	NS

At pre-test, relatives with fewer critical comments had a significantly higher total score (median 14, range 10–18) than those with more critical comments (median 12, range 7–17). Similarly at post-test, scores were significantly higher for the less critical group (median 19, range 12–21) than for the more critical group (median 17, range 8–18).

(b) Emotional over-involvement (EOI)

Relatives were divided into those who scored 3 or more on EOI ($n=11$) and those who scored 2 or less ($n=13$). Hostility and critical comments were ignored for this analysis. Mann-Whitney U tests were carried out to compare the two groups on total scores (pre- and post-tests) and on change scores. No significant differences were found between the two groups.

(c) Hostile/non-hostile relatives

Relatives were divided into two groups, according to their hostility rating (hostile $n=9$, non-hostile $n=15$). EOI and critical comments were ignored for this analysis. Mann-Whitney U tests were carried out on the total scores for pre-test and post-test, and on change scores. No significant differences were found between the two groups.

(d) Relationship with patient

Relatives were divided into two groups according to whether they were the parents of the patient ($n=15$) or were related in another way (e.g. spouse, child, sibling: see Table I) ($n=9$). Mann-Whitney U tests were carried out on total scores at pre-test and post-test and on change scores. No significant differences were found.

3. Patients' characteristics and KASI scores

(a) Duration of illness

Relatives were divided into two groups, based on a median split of the number of years since the onset of the patient's illness (calculated from the first hospital admission). For the 'less chronic' patient–relative group, the median number of years since onset was 2.5, with a range of 0–3 years. For the 'more chronic group', the median was 7, with a range of 4–24 years. A Mann-Whitney U test was carried out on total scores at pre-test and post-test and on change scores:

Comparison	*z*	*P (two-tailed)*
Pre-test total scores	1.76	0.08
Post-test total scores	1.91	0.06
Change scores	2.71	0.007

Relatives of 'less chronic' patients had significantly higher change scores (median 6, range 0–10) than relatives of 'more chronic' patients (median 3, range -1–$+10$)

At pre-test, relatives of 'less chronic' patients had lower scores (median 13, range 7–18) than those of 'more chronic' patients (median 14, range 12–17); but at post-test, the position was reversed (less chronic: median 17.5, range 8–21; more chronic: median 15.5, range 12–18). These pre-test and post-tests comparisons just failed to reach significance.

(b) Number of hospital admissions

Relatives were divided into two groups, based on a median split of the number of times the patient had been admitted to hospital. A Mann-Whitney U test was carried out on total scores at pre-test and post-test and on change scores: no significant differences were found between the two groups.

(c) Remission of symptoms

Relatives were divided into two groups, depending on whether the patient experienced positive schizophrenic symptoms continuously, or had had remission of symptoms in between episodes of illness. Mann-Whitney U tests were carried out on the total scores for pre-test and post-test and change scores, and showed no significant differences between the groups.

4. Summary of results

(a) There was a significant increase in individual scores between tests. Total scores showed a significant increase, as did scores on all the different sections of the KASI except section 4 (medication), where the increase in scores closely approached significance. There was a significant change from responses of negative or neutral value to the patient to those of positive value, for all sections except medication.

(b) Relatives who were rated 'low on criticism' had significantly higher test scores at both pre-test and post-test. Relatives rated 'low' on hostility or emotional over-involvement did not show significant differences in test scores from those rated 'high'.

(c) Relatives of patients with more recent onset of illness showed lower scores at pre-test and higher scores at post-test: these differences closely approached significance. The change scores of these relatives were significantly higher.

(d) No significant differences in scores were found when relatives were grouped according to the patient's number of hospital admissions, the pattern of remissions, or the relative's familial relationship to the patient.

Discussion

This paper has described a method of assessing what relatives know about schizophrenia, and how this knowledge might be improved or changed through a relatively brief educational intervention. It is important, however, to emphasise the nature of the assessment: our concern was to categorise relatives' responses to questions about schizophrenia in terms of what significance those responses might have in either assisting or hindering the patient's recovery. In other words, our interest was in the functional value that the information potentially held, so that the relatives' responses were allocated either a negative, neutral, or positive functional value.

The results from this initial trial appear to be encouraging: relatives' knowledge (as assessed by the KASI) improved significantly after the information session. Moreover, there was a significant change in their responses, from functionally negative or neutral before the education sessions to positive afterwards. This result applied to five of the six different aspects of schizophrenia assessed and to the total score derived from all six sections of the KASI; in the case of the other section (Medication) there was improvement which closely approached statistical significance. This is evidence of the success of brief intervention in giving relatives information which might be used to assist the patients. Conversely, information which might lead to actions detrimental to the patients' recovery was significantly reduced.

A closer examination of the questions in the medication section, and relatives' responses to them, may explain why the trend towards improvement there did not reach significance. To receive a positive-value score (3), relatives were required to know some practical details about the administration of the medication; e.g. when, where and for how long it was to be given. They were also required to advocate that the patient remain on medication for as long as the doctor prescribed, when presented with other choices such as "for as long as the patient thinks she/he needs it" or "for as long as you think she/he needs it". Since most relatives had this knowledge already at pre-test, any improvement was probably limited by their initial scores. Questions about the prophylactic nature of medication (e.g. "If your relative seemed and felt completely better and decided to stop having the medication, what do you think would happen?") were contained in Section 5 of the interview ('Course and prognosis'), which showed significant improvement at post-test. Relatives could therefore agree that the doctor should decide how long the patient should receive the medication, yet make clear from their responses in Section 5 that they actually believed that the patient would be better off without it. According to the KASI, such negative responses were reduced at post-test.

The data from the relatives' responses were analysed according to the important dimensions of EE: frequency of criticism; hostility; and marked emotional over-involvement. Analyses of the data were performed taking each dimension into account independently of the other two. Only one analysis produced significant results: relatives who made six or more critical comments in the CFI tended to have significantly lower KASI scores at both pre-test and post-test. There was no significant difference between the critical and less critical groups in their change scores. These results suggest that the more critical relatives had more negative or neutral-value information about schizophrenia at pre-test, but that they benefitted as much as the less critical relatives from the education sessions. We therefore agree with Vaughn & Leff (1981) that one of the characteristic attitudes or response styles distinguishing high- from low-EE relatives is their attitude to the illness. High-EE relatives tend to doubt that the patient has a legitimate illness, and feel that he or she might do more to control the 'symptoms'. Of particular relevance to this conclusion are the responses of the relatives in our study to questions in Section 2 of the KASI, which refer to symptoms. Relatives were required to say whether the 'problems' they had reported with the patient were part of the person's illness or were associated with his or her natural self'; they were also required to state whether or not they thought the patient could control these symptoms. All four relatives in our study who were given a 'negative' score in this section (i.e. those who reported believing that most of the symptoms they observed were part of the patient's personality and/or within his or her control) were rated as critical and hostile. A larger group of relatives would be necessary to examine the statistical significance of this finding, however.

No differences were found between KASI scores when relatives were grouped on the basis of hostility or EOI ratings; but there were relatively few subjects, in the group analysis, and further investigation is necessary.

The analysis of relatives' scores in relation to the chronicity of the patients' illness produced some interesting results; chronicity was defined as the number of years since the onset of the illness, and the relatives were divided into two groups on this basis. The relatives of patients with relatively short histories had lower KASI scores at pre-test and greater change scores. These results suggest that the shorter the length of illness the less the relatives know, but the more receptive they are to acquiring information. This is consistent with an interaction model of how relatives take in information about schizophrenia (Tarrier & Barrowclough, 1986), which postulates that relatives develop an individual view of the patient's condition and bring to an education session opinions as to its causes, course, and consequences which affect their acceptance or rejection of alternative information offered to them. The longer the patient's illness, the more time the relative has had to formulate his own lay model of the illness, and the less influenced he is by professional opinion. We found no significant differences in knowledge between relatives of patients with comparatively few past hospital admissions and those with more, so one might speculate that contact with hospital psychiatric services did not influence the development of the relatives' attitudes. It is apparent that early education about the schizophrenic condition is important in influencing relatives' behaviour. Birchwood (1983) has suggested that negative family influences in schizophrenia develop as the condition becomes chronic.

If some relatives have an existing and well developed lay model of schizophrenia, it is not surprising that there may be no change in their views after a relatively brief educational intervention. Although analysis of group data indicated significant increases in information in the group as a whole, those relatives whose scores did not show improvement are important from both the research and clinical points of view. The scoring system of the KASI may be of particular use in indicating which aspects of schizophrenia are misconstrued by particular relatives; further information and advice may then be given to those most in need, and their responses re-evaluated until acceptable criteria are reached.

Berkowitz *et al* (1984) commented that relatives remembered only a fraction of what they had been told in their educational sessions. The differences in findings between their study and ours may be due to several factors, including differences in educational content and presentation, the assessment procedures used, and the time interval before retesting. Berkowitz *et al*, like McGill *et al* (1983), placed greater emphasis than us on imparting and assessing knowledge about schizophrenia *per se*, in contrast to our assessment of the potential functional value of the information in the context of the particular patient's illness. Although the education sessions reported by McGill *et al* did emphasise the symptomatology of the individual patient, this does not appear to have been reflected in the assessment procedures. Neither McGill *et al* nor Berkowitz *et al* differentiated between negative, neutral and positively valuable information. Consequently, a relative might be assessed as having given the 'correct' answer after an education session, while retaining beliefs which could lead to actions detrimental to the patient's management (see Tarrier & Barrowclough, 1986, for discussion).

The finding of Berkowitz *et al* that relatives were particularly lacking in knowledge about the aetiology of schizophrenia, and that they tended to retain their own versions of the causes of the illness, is partly supported by the results of the present study. The Aetiology section of the KASI contains an open question about causation, followed by a series of forced-choice questions regarding possible causes, and enquiries designed to investigate relatives' behaviour. At pre-test, all the relatives' responses were assessed as negative or neutral. Although there was a large and significant increase in scores at post-test, a substantial number remained in the neutral category, indicating that these relatives had learnt little about aetiology.

Conclusions

This paper has described the development and initial results of an evaluation procedure which aims to examine the information which relatives hold about schizophrenia, with a specific focus on the functional value of that information. The advantages of the instrument include ease of administration and scoring, reliability of evaluation, and sensitivity to change. The effectiveness of the educational programme has been demonstrated, although the longer-term maintenance of any improvement which the relatives showed in their knowledge has yet to be evaluated. The data will be reported when available.

Many relatives bring to the educational session well-developed and systematised knowledge of their own, and the difficulties in changing their beliefs and attitudes should not be under-estimated. Our preliminary results suggest that educational sessions are likely to have more impact on relatives of patients with a fairly short history of illness.

One of the advantages of the KASI may be the identification of relatives with beliefs and attitudes which can result in actions detrimental to the patients' well-being, and who consequently require further advice and support.

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Appendix

As described in the "Method" section of this paper, each relative's knowledge of schizophrenia and his/her corresponding attitude towards the patient was assessed as:

1 negative
2 neutral
3 positive
4 positive and well-informed

This assessment was done for each area of knowledge examined by the KASI interview. Examples to show how such assessments were made with regard to diagnosis and symptomatology are given below.

Rating relatives' knowledge of diagnosis

The section of the KASI dealing with diagnosis is concerned only with relatives' knowledge of the name of the condition and the recognition that schizophrenia is a serious mental illness which can affect all aspects of a person's life. The absence of such knowledge is not by itself considered to have direct negative functional value, and so would indicate a rating of 2 (neutral); but such ignorance combined with other erroneous information with potentially negative value would lead to the relative's allocation to category 1 (negative). Examples of negative statements would include "It's not a mental illness, it's just his personality" or "She's not mentally ill, she's a drug addict". It would be logical to suppose that in the first case the relative's denial of the patient's condition could lead to criticism of his behaviour; while the relative's attitude in the second case could lead to a failure to support compliance with prophylactic medication.

Some items in the diagnosis section were later removed because it was found that they were not after all helpful in categorising relatives' behaviour. These items included "Does schizophrenia mean the person has a split personality?" and "Are people with schizophrenia more likely to be violent?". Although strictly speaking, a negative response to these items would indicate knowledge of schizophrenia, it became clear that a positive reply had positive functional value to some relatives: the idea of a "split personality" was useful in distinguishing between the ill person who behaved in a way unlike his usual self and the healthy person. Similarly, the association of violence with schizophrenia was helpful to those people who had experienced their relatives behaving 'violently' while ill, in complete contrast to their normal selves.

Rating relatives' knowledge of symptoms

Our concern here was with relatives' attribution of the problems or symptoms they had previously reported in the CFI, as well as with their knowledge of the patient's symptoms.

To be allocated a score of 3 (i.e. to be rated 'positive'), the relative was required to attribute 60% or more of the problems or symptoms to the illness rather than to the person's natural self. The same criteria also had to apply to the relative's beliefs concerning the amount of control the patient had over the symptoms. In addition, relatives were required to identify at least *one* of the patient's diagnostic symptoms, and not to attribute this to the patient's personality or to regard it as being under his control.

The relatives' attribution of problems and their beliefs about control over symptoms were assessed through a forced-choice response; e.g. "You have mentioned that X's problems affected him by him talking to himself. Do you think this behaviour is part of X's natural self, or do you think it's part of X's condition?", and "Do you think Y could help or control talking to himself?". The relative was required to reply "Yes", "No" or "Don't know". A response of "don't know" for more than 60% of the items meant allocation to category 2 (neutral). Attribution mainly to the person's 'natural self' and/or 'own control' were allocated to category 1 (negative), on the grounds that such beliefs might have detrimental functional value; the relative might *blame* the patient for unusual or disruptive behaviour.

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Abnormal Auditory Brain-stem Responses in Hallucinating Schizophrenic Patients

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Abnormal auditory brain-stem responses (ABRs) were recorded in 10 out of 20 schizophrenic in-patients. The response abnormalities did not show any correlation to the degree of psychopathology, sub-group of schizophrenia, age, sex, or cerebral ventricular enlargement. Nor was there any correlation to previous neuroleptic treatment: a pathological ABR was recorded in 5 of the 8 patients who had never received such medication. A statistically significant relationship was found between ABR pathology and auditory hallucinations: 9 of the 11 patients who admitted having hallucinations exhibited brain-stem response abnormality, whereas ABR abnormality was recorded in only 1 of the 9 patients who denied having hallucinations. The data imply that brain-stem dysfunction is involved in the psychopathology of schizophrenia, and that interference with the auditory pathways in the brain-stem may induce auditory hallucinations in schizophrenic patients.

Schizophrenia has been found to be associated with many signs of brain dysfunction. Techniques including neuropathology, computed tomography (CT), cerebral blood flow and positron-emission tomography have contributed to the demonstration of cerebral abnormalities of various degrees in a large proportion of schizophrenic patients (for review see Seidman, 1983). Neurological so-called "soft signs" indicating brain dysfunction are also common in schizophrenia (Kennard, 1960; Pincus & Tucker, 1978): the frequency of such signs in schizophrenics has been reported to range between 36% and 75% (Hertzig & Birch, 1966; Mosher *et al*, 1971; Torrey, 1980). But although there is general agreement about the existence of brain dysfunctions in schizophrenia, opinions regarding their anatomical localisation differ. Much attention has been directed towards the limbic structures, but also towards the basal ganglia (Lindsky *et al*, 1979), corpus callosum (Rosenthal & Bigelow, 1972) and cerebellum (Health *et al*, 1982).

Several studies using different techniques have suggested the brain-stem to be the critical site for many of the symptoms observed in schizophrenic patients. The brain-stem cranial-nerve nuclei and tracts, together with the reticular formation, are responsible for sensory integration. Data indicating abnormal brain-stem function have been obtained from schizophrenic patients by testing proprioceptive mechanisms in the vestibular system (King, 1974), by continuous performance tests (Heaton *et al*, 1978), by eye tracking tasks (Holzman *et al*, 1976) and as evoked potentials on EEG (Shagass, 1975). Postmortem studies have reported brain-stem lesions of an encephalitic nature in schizophrenics (Fisman, 1975).

We present here a study which aimed to assess brain-stem function, using auditory brain-stem responses, in first-admitted as well as chronic residual schizophrenic in-patients. The data were correlated to age, sex, symptomatology, brain ventricular enlargement and medication.

Method

Patients

Twenty schizophrenic in-patients, 12 male and 8 female, participated in the study on an informed-consent basis. The median age was 27 years (range 19–44) and the median duration of illness was 3½ years (range 1 month–20 years). Eight of the patients had never been admitted to hospital before and had never received neuroleptic treatment. Diagnosis of schizophrenia was ascertained using the Research Diagnostic Criteria (RDC: Spitzer *et al*, 1977), according to which 7 cases were of the disorganised sub-type, 6 of the paranoid sub-type and 7 of the undifferentiated sub-type of schizophrenia.

Symptoms were scored using 33 items from the Comprehensive Psychopathological Rating Scale (CPRS: Åsberg *et al*, 1978). Special attention was focused on the presence or absence of auditory hallucinations. If an individual had experienced voices for at least one hour during the week preceding the brain-stem recording, auditory hallucinations were considered to be part of the symptomatology.

Nine patients had been on neuroleptic treatment for at least one month (range 1 month–16 years) when the brain-stem response was recorded. Data on the patients are presented in Table I.

TABLE I

Clinical characteristics, ventricular/brain ratios and auditory brain-stem responses of 20 schizophrenic patients

Patient number	*Sex*	*Age*	*Current neuroleptic treatment*[1]	*Total CPRS score*[2]	*Auditory hallucinations*	*Ventricular/brain ratio (VBR)*	*Auditory brain-stem responses (ABR)*
1	M	26	—	22.5	—	10.63	normal
2	F	25	neuroleptics	29.5	hallucinations	13.01	normal
3	M	19	—	20.0	—	9.60	normal
4	M	28	—	—	—	—	normal
5	F	29	neuroleptics	14.0	—	11.78	normal
6	M	30	neuroleptics	11.5	—	9.13	abnormal
7	M	22	—	10.5	hallucinations	12.81	abnormal
8	M	25	neuroleptics	22.5	hallucinations	6.84	abnormal
9	M	24	—	17.5	—	9.70	normal
10	M	32	neuroleptics	22.5	hallucinations	8.93	abnormal
11	F	20	neuroleptics	17.5	hallucinations	12.97	abnormal
12	F	41	neuroleptics	24.0	hallucinations	8.07	abnormal
13	M	44	neuroleptics	—	hallucinations	10.98	abnormal
14	M	31	neuroleptics	—	hallucinations	9.30	abnormal
15	F	21	—	18.0	hallucinations	—	normal
16	M	19	—	17.5	hallucinations	8.73	abnormal
17	F	33	—	37.5	hallucinations	—	abnormal
18	F	33	—	21.0	—	—	normal
19	M	35	—	10.0	—	—	normal
20	F	19	—	15.0	—	10.06	normal

1. Receiving neuroleptics for at least one month prior to ABR recordings.
2. Comprehensive Psychopathological Rating Scale: Åsberg *et al* (1978).

ABR recording technique

The recording arrangements used in this study are based on a widely accepted technique and the normative data used in our Department of Audiology are in close agreement with those reported by Rosenhamer *et al* (1978).

Before the recording session, pure tone audiometry was performed to detect any hearing loss that might influence the recording and analysis of the ABR.

During the recording session the subjects rested comfortably on a couch in a sound-proof and electrostatically shielded room. No sedatives were given. For auditory stimulation and recording brain-stem response a Nicolet Pathfinder II-system was used. The acoustic stimuli consisted of condensation clicks which were presented unilaterally via TDH-39 headphones at a rate of 21.3 clicks per second, a mode of stimulation that was found to produce a minimum of aliasing errors. No contralateral masking was used. The phones were fed with unfiltered square-wave pulses of 100 µs duration. The evoked potentials were picked up with gold-plated surface electrodes applied to the forehead (position Fp_z) and over the mastoid processes, and recorded between the forehead and the ipsilateral ear (with regard to stimulation). The electrode on the contralateral side was connected to ground. The impedance of each electrode pair was checked to be less than 5 kΩ.

The auditory brain-stem responses to 2000 clicks were amplified, filtered and, after static artifact rejection, recorded and averaged in a 10 ms window opened by the stimulus signal. The responses were recorded when stimulating each ear at intensity levels of 90dB, 70dB, 50dB and 0dB ref nHL (normal Hearing Level) (i.e. dB above the laboratory reference zero level). To check the replicability of an averaged response, two averages were recorded at each stimulation level and superimposed. All averaged responses were stored in computer files for digital filtering, processing, analysis and further follow-ups. The input signal from the patient was continuously monitored, to check for disturbances on the recording due to motoric hyperactivity, dislocation of electrodes, etc.

An ABR-pattern normally consists of a series of positive/negative waves within the first 10 ms after the onset of acoustic stimulation. Forehed (vertex) positive peaks are recorded as upward deflections and designated by the roman numerals I, II, III, IV, V (Jewett & Williston, 1971).

On analysis an ABR-peak was looked upon as significant only if its absolute latency (from onset of stimulation) was replicated with a latency difference not exceeding 0.1 ms. The absolute latencies and amplitudes of accepted peaks were measured, as well as identifiable inter-peak intervals I–III, I–V and III–V. Abnormalities such as absence of peaks, pathology of absolute latencies or inter-peak intervals, right–left differences and abnormal stimulus–response functions were judged according to our normative data and the hearing of the subject.

Doubtful abnormalities were disregarded. The auditory brain-stem responses were thus classified as pathological only if one or more peaks were lacking; or if the right/left difference in latencies for the identified peaks exceeded 0.2 ms at a stimulation level of 70dB, or if the latency for peak V exceeded 6.3 ms on either side.

Computed tomography

Lateral ventricular size was determined from CT scans by use of an EMI CT 1010 Scanner: slices were taken, without contrast enhancement, every 10 mm from vertex to foramen magnum. The slice showing the ventricles at their largest was selected, and the area of the lateral ventricles and of the intracranial space in the chosen cut was measured with a planimeter on the transparent film. The ventricular area was then divided by the intracranial area to provide the ventricular/brain ratio (VBR), which has been shown to correlate well with computer-derived ventricular volume (Penn *et al*, 1978). Four measurements were performed for each cut, and the mean values are presented in Table I.

Results

Ten out of the 20 patients studied exhibited abnormal auditory brain-stem responses. Two pathological brain-stem responses are presented in Figures 1 and 2: Figure 3 shows a normal ABR-pattern for comparison. The normal response pattern (patient no. 5) shows the peaks I-V, clearly identifiable, with normal latencies and normal amplitude ratios,

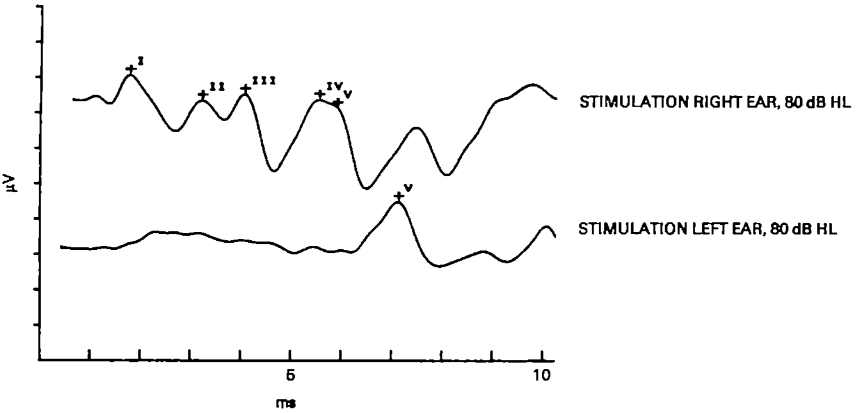

FIG. 1 Abnormal auditory brain-stem response with only a pathological delayed peak V on stimulation left. No ABR abnormality on equivalent stimulation right (patient No. 12).

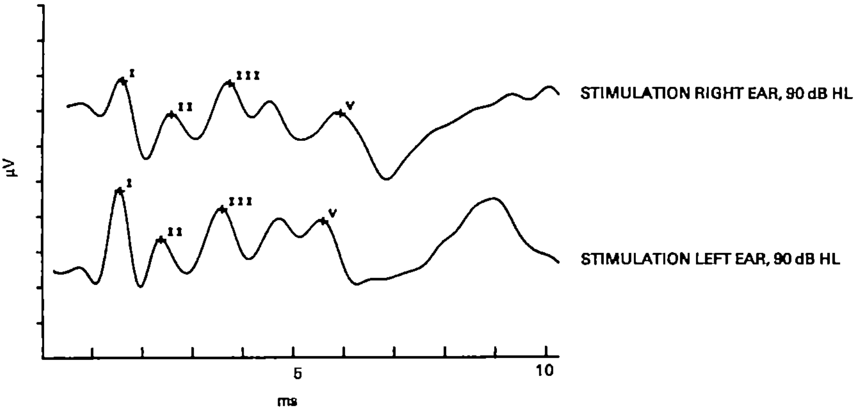

FIG. 2 Abnormal auditory brain-stem response in the form of pathological right–left latency differences (patient No. 8).

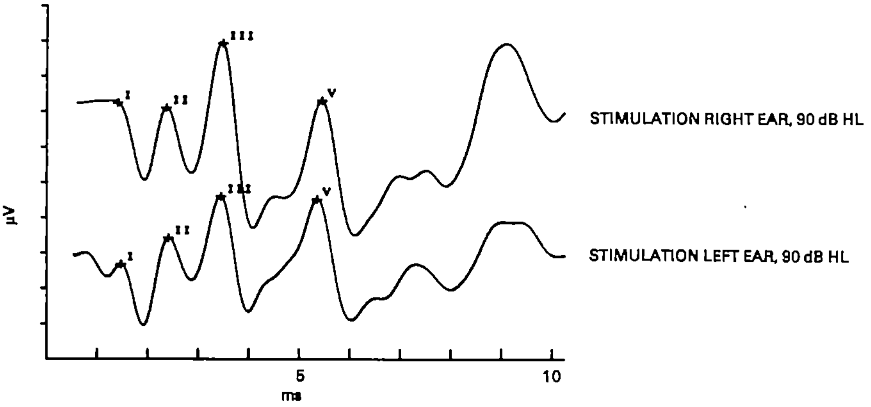

FIG. 3 Normal auditory brain-stem response in a schizophrenic patient (patient No. 5).

and without any significant right/left differences. Figure 1 (patient no. 12) demonstrates a normal response to stimulation of the right ear, but left-ear stimulation produced only a peak V with an abnormal absolute latency. In Figure 2 there is a significant right/left difference in latency of the peak II–V (patient no. 8).

There was no statistically significant difference between patients with normal *vs* abnormal ABR with regard to age ($t = 1.19$, NS), degree of psychopathology as assessed by total CPRS scores ($t = 0.53$, NS), or signs of cerebral atrophy as indicated by VBR ($t = 1.10$, NS). Nor was there any significant difference between the sexes: 7 of the 12 males (58%) and 3 of the 8 females (38%) exhibited abnormal brain-stem responses. The distribution of abnormal *vs* normal brain-stem responses within each sub-group of schizophrenia did not yield any differences between sub-groups (disorganised 3:4, paranoid 3:3 and undifferentiated 4:3).

Of the eight first-admission drug-naive patients, five (63%) displayed abnormal brain-stem response.

Although no relationships were seen between ABR and degree of psychopathology or sub-group of schizophrenia, a significant relationship was found between the presence of auditory hallucinations and abnormal brain-stem responses. Of the eleven patients who admitted having auditory hallucinations, nine had abnormal brain-stem responses. Among the nine patients who denied having hallucinations, only one had abnormal responses (Fisher's exact probability test; $P < 0.01$).

Discussion

Auditory brain-stem evoked response recording is a computerised, non-invasive tool for tracing brain-stem pathology that may cause disturbances of the auditory pathways.

The pattern of the ABR in a normal adult population is very stable, both in and between individuals (Jewett & Williston, 1971). The ABR is not significantly affected by sleep, wakefulness, psychic tension, awareness of stimuli or focus of attention: thus the ABR is characterised by a high degree of integrity, replicability and objectivity, within narrow limits of normality.

It is generaly agreed that peak I of the ABR derives from the activation of the acoustic nerve, whereas peaks II-V originate from the brain-stem, peak V being the most prominent and identifiable component of the response. Attempts have been made to establish a relationship between the components of the response and anatomical structures in the primary ascending auditory pathways (Stockhard & Rossiter, 1977), but most investigators believe that each peak reflects a composite activity of several neural structures (Jewett, 1970; Picton *et al*, 1974; Starr & Hamilton, 1976). In psychiatry, the very few ABR studies reported have dealt with autistic children (Sohmer & Student, 1978) or retarded adults (Squires *et al*, 1980). Schizophrenic patients have been studied by Pfefferbaum *et al* (1980) and Szelenberger (1983) but these studies yielded no significant findings in relation to the disease, possibly because the questions they asked and consequently the methods they used were different from ours.

The abnormal brain-stem responses reported here could not be ascribed to peripheral hearing impairment and were not related to sex or age. The fact that abnormal brain-stem responses were seen in five out of eight first-admission patients who had never received neuroleptics indicates that ABR pathology may be associated with the disease itself. However, it is well established that long-term neuroleptic medication can induce irreversible neuronal dysfunction within the basal ganglia manifesting itself as tardive dyskinesia (Gunne *et al*, 1984). Consequently, it is still possible that neuroleptics may influence the auditory pathways of the brain-stem. We have therefore initiated longitudinal studies of patients treated with neuroleptics and exhibiting a normal ABR prior to the treatment.

Our data do not support the hypothesis that the ABR pathologies were manifestations of a generalised cell loss in the central nervous system which would make itself evident in the form of enlarged lateral ventricles. The VBR *has* been found to be increased in a large proportion of schizophrenic patients (Johnstone *et al*, 1976; Weinberger *et al*, 1979) but in the present study VBRs did not differ between patients with and those without abnormal brain-stem responses. However, the sample was too small for definite conclusions. The same reservation applies when considering a possible genetic influence as a causative factor for the brain-stem response abnormalities. Three of the four patients with a family history of schizophrenia exhibited an abnormal ABR, a finding that deserves further attention in future studies.

A notable finding was the significant correlation between the presence of auditory hallucinations and abnormal auditory brain-stem responses. This observation indicates that auditory hallucinations may have an organic backround. One could speculate whether a dysfunction of the brain-stem might in some way generate impulses (like in denervation supersensitivity) which would be experienced as voices by the patient.

The different abnormalities noted in the ABR patterns of our schizophrenic patients do not permit firm conclusions to be drawn with regard to the anatomical localisation of the brain-stem pathology. In a postmortem study by Fisman (1975), encephalitic lesions with glial knots and perivascular infiltration were found in the brain-stem area in six out of eight schizophrenic patients but only one out of 24 controls. A significant role of the brain-stem function in schizophrenia has been suggested by studies on local cerebral metabolism using regional cerebral blood-flow recording (Ingvar & Franzen, 1974; Franzen & Ingvar, 1975; Mathew *et al*, 1981). It has been hypothesised that schizophrenics suffer from an abnormal distribution of activity within the brain due to an inability of the brain-stem and limbic mechanisms to activate the frontal lobes (Ingvar 1976; Weinberger & Wyatt, 1982). Such a deficiency might be caused by a sub-cortical dopaminergic dysfunction (Buchsbaum *et al*, 1982).

The present study is the first step by our group towards a more comprehensive mapping of the auditory brain-stem evoked response in psychiatric disorders, especially schizophrenia. Questions yet to be considered are whether abnormal ABR varies systematically with the clinical picture; and whether ABR is influenced by medication with neuroleptics or with the opiate antagonist naloxone, which has been demonstrated to reduce auditory hallucinations in schizophrenics (Gunne *et al*, 1977; Picar *et al*, 1982).

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Asymmetry of Lateralised Hemispheric Functions in Schizophrenia
Influence of Clinical and Epidemiological Characteristics on Quality Extinction Test Performance

SILVIO SCARONE, CARLO LORENZO CAZZULLO and ORSOLA GAMBINI

The Quality Extinction Test was used to detect lateralised abnormalities of hemispheric functions in schizophrenic patients. Course of illness significantly affected the distribution of tactile extinctions, chronic patients showing more left-side extinctions than sub-chronic ones. Age significantly affected the number of left extinctions, and sex the number of right extinctions. The importance of clinical and epidemiological characteristics of the patients in determining the quality and degree of hemispheric dysfunction in schizophrenia was confirmed.

In recent series of papers (Scarone *et al*, 1981, 1982, 1983) we reported the initial findings on various aspects of tactile 'losses' in schizophrenic patients. The Quality Extinction Test (QET) (Schwartz *et al*, 1979) provided the means for assessing their tactile sensitivity, by evaluating a blindfolded subject's verbal answers to simultaneous brushing of the palm of each hand with two different common materials. In general, compared with normal controls, schizophrenic patients have been found to have a significantly higher incidence of left extinguishing responses to the QET and a greater number of left and right extinctions; in the case of the latter, the left extinctions were significantly more frequent than the right ones (Scarone *et al*, 1981, 1982). Given the organisation of the neurofunctional pathways of tactile sensitivity (Critchley, 1953), these results suggest that both the parietal and anterior frontal regions of the brain might play a role in the pathophysiology of losses in tactile sensitivity.

A possible influence of the age of the schizophrenic patients on the proportion of left extinctions was also found, in contrast to the controls; there was no relationship between QET performances and the clinical paranoid/non-paranoid dichotomy (Scarone *et al*, 1983). Although the findings are not entirely consistent, recent reports lend support to the existence of a relationship between some of the clinical and epidemiological characteristics of schizophrenia and hemispheric malfunctioning (Gruzelier, 1984; Andreasen *et al*, 1982; Luchins *et al*, 1981). Consequently, it seems reasonable to assume that characteristics of schizophrenia itself might have a determining role, relative to the nature and degree of hemispheric malfunctioning in this disorder. The objective of the present study was to further examine the QET performances of a sample of schizophrenic patients controlled for sex, age, diagnosis, and course of the disease.

Method

The subjects were 136 patients with a diagnosis of schizophrenia; Table I shows their epidemiological and clinical characteristics. The diagnosis of schizophrenia, subtype classification, and chronicity characteristics were independently determined by two senior psychiatrists using DSM–III criteria. All the patients were right-handed, as assessed by means of a standardised questionnaire (Raczkowski *et al*, 1974). Patients aged over 55 were excluded from the study, since both extended hospital stay and long-term treatment with psychotropic drugs have been reported to exercise a significant influence on hemispheric functioning (Luchins *et al*, 1981; Serafetinides, 1973). The majority (79) of the subjects were aged 18–35; the remaining 57 were aged 36–55. The 112 chronic patients tended to be older than the 24 sub-chronic patients, their mean ages being 35.8 years and 21.2 years respectively.

TABLE I
Clinical and epidemiological characteristics of schizophrenic patients, categorised according to diagnosis

Sex	*Disorganised*		*Paranoid*		*Undifferentiated*		*Total*	
	Number	*Mean age*	*Number*	*Mean age*	*Number*	*Mean age*	*Number*	*Mean age*
Males	36	27.8	19	42.7	19	32.3	74	32.7
Females	22	30.0	16	44.9	24	32.0	62	34.8

TABLE II

Distributions of tactile extinctions according to age, sex, diagnosis, and chronicity

	Extinctions		
	Left	*Right*	*None*
Age			
Under 35	29	9	41
Over 35	29	5	23
Sex			
Male	34	9	31
Female	25	2	35
Diagnosis			
Disorganised			
Males	18	6	12
Females	9	1	12
Paranoid			
Males	5	1	13
Females	7	—	9
Undifferentiated			
Males	11	2	6
Females	9	1	14
Chronicity (total sample)[1]			
Sub-chronic	5	4	15
Chronic	56	7	49
Chronicity (age-matched)[2]			
Sub-chronic	5	4	15
Chronic	15	2	7

1. $\chi^2 = 7.88$, d.f. $= 2$, $P < 0.025$
2. $\chi^2 = 8.58$, d.f. $= 2$, $P < 0.025$

None of the patients had a history of brain damage, and all had negative findings on physical and neurological examinations before administration of the QET. All subjects had been receiving medication (phenothiazines or butyrophenones) for at least six months before the study and up to the time of QET testing. The QET was administered in accordance with the standard procedures (Schwartz *et al*, 1977). The total QET score was derived by subtracting the number of negative responses relating to the more accurate hand from that relating to the other, and expressing this difference as a percentage. The absolute number of left and right extinctions was also computed.

The distribution of left and right extinctions was tested by 2 × 2 and 2 × 3 contingency tables. A linear stepwise multiple regression analysis was performed, with the number of left and right extinctions as the dependent variables and age, sex, diagnosis, and chronicity as the independent ones.

Results

Table II shows the distributions of tactile extinctions according to age, sex, diagnostic categories, and chronicity. Chi-squared tests showed no significant relationships to the first three of these. The course of schizophrenia, however, does affect the extinction distribution: chronic patients show significantly more left extinguishing characteristics ($P < 0.025$). To check for the possible influence of age on this relationship, 24 chronic patients were age-matched with a sub-group of 24 sub-chronic patients. The results are also given in Table II; once again there is significant relationship between chronicity and tactile extinction type ($P < 0.025$).

When multiple regression analysis was performed, taking the number of left and right extinctions as dependent variables and age, sex diagnosis, and chronicity as independent variables, age was found to be the only independent variable affecting the number of left extinctions (R-square $= 0.470$, F-test $= 6.651$, $P < 0.025$). The insertion of the other variables into the regression equation did not significantly increase the proportion of variance accounted for. The multiple regression analysis relating to the number of right extinctions showed that sex was the only independent variable which increased the proportion of variance accounted for (R-square $= 0.703$, F-test $= 10.209$, $P < 0.001$).

Discussion

Degree of chronicity appears to be the clinical characteristic most markedly related to the distribution of tactile extinctions in the group studied, with the more chronic patients having far less left tactile sensitivity. Since the same differences appeared when age-matched subjects were compared, this finding seems to be unrelated to patients' age.

The relationship between chronicity and left tactile abnormal sensitivity might be interpreted in a variety of ways, but from the anatomico-functional viewpoint it would seem to reflect a specific impairment in hemispheric functioning – one inherent in the ongoing course of schizophrenia. Furthermore, this hypothesis is consistent with findings from other studies that have demonstrated a significant direct relationship between chronicity and degree of cerebral malfunctioning (Johnston *et al*, 1976; Gruzelier, 1984). Within this anatomico-functional context, there is also the possibility that other factors closely related to duration of illness, such as the length or number of admissions or exposure to pharmacological treatment, might affect tactile sensitivity. All these factors have been shown to have an influence on hemispheric functioning in schizophrenia (Luchins *et al*, 1981; Serafetinides, 1973).

The results reported here did not replicate a previous finding of a significant direct relationship between age and the incidence of left extinctions (Scarone *et al*, 1982). This might be explained, however by the fact that patients over 55 were not excluded from the previous study group; that group included a number aged over 60, all of whom had a marked tendency to be left extinguishers. The finding that there is no relationship between subtype of schizophrenia and the distribution of extinctions is consistent with results from an earlier and smaller sample (Scarone *et al*, 1983).

It is possible to conclude that QET responses in schizophrenia are independent of the clinical aspects of the illness and are related instead to some type of neurofunctional impairment in hemispheric functioning that is inherent in the disease itself. Since other neuropsychological and psychophysiological measures of lateralised malfunctioning show the same characteristics (Gur, 1978; Gruzelier & Hammond, 1980). It can be suggested that some hemispheric abnormalities reflect a certain degree of nuclear pathology in schizophrenic patients.

In schizophrenia, the quantitative aspects of tactile extinctions appear to be quite different from their qualitative ones; in fact, the present results show that patients' age significantly explains a part of the variance in the number of extinctions. Data from the neurological literature suggest that the left-sided pathways of tactile sensitivity are more vulnerable than the right-sided ones (Schwartz *et al*, 1979). The possibility thus exists that some age-dependent neurofunctional modifications in hemispheric assessment play a critical role in determining such vulnerability.

The finding that patients' sex explains a part of the variance in the regression equation for the number of right extinctions does not lead to any simple interpretation, and needs to be evaluated in the light of additional data. According to the neurofunctional interpretation of the tactile sensitivity pathways, however, a high rate of right-side extinctions almost exclusively reflects malfunctioning of the left hemisphere. A recent clinical study demonstrated that neurological lesions of the left hemisphere differentially affect males' and females' lateral functioning (Inglis *et al*, 1982), thus suggesting that sex has a marked effect on hemispheric functional organisation. It is therefore possible that lateral functional disorders, as well as anatomical lesions, might be sex-dependent where such organisation is concerned.

The *R*-square values obtained explain a small part of the variance in both of the regression analyses undertaken. Other variables that were not introduced into the equation, such as duration and number of hospital admissions or duration of psychopharmacological treatment, might be important in determining QET responses in schizophrenia. Only continued study, however, will allow greater understanding of tactile sensitivity and its precise relationship to schizophrenia.

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Correlates of Suicide Attempts and Ideation in Schizophrenia

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A group of 118 patients with a hospital diagnosis of schizophrenia was reviewed. The incidence of suicide attempts was significantly correlated with more frequent past psychiatric admissions, more frequent past diagnoses of schizophrenia, and poor work function. The apparent relationships could be spurious or based on a reversed causal sequence; for example, suicide attempts are likely to be followed by psychiatric admissions.

Although it has been estimated that about 10% of patients with schizophrenia commit suicide (Miles, 1977), little is known about the clinical characteristics of those schizophrenic patients who are most at risk of such an outcome (Wilkinson, 1982). The sparse data available suggest that important features associated with suicidal tendencies may include: feelings of depression or a known history of depression and unemployment (Roy, 1982), episodes of excitement with acute panic or protracted anguish (Planansky & Johnston, 1973), an extensive history of past psychiatric admissions (Roy *et al*, 1984), and in some cases an increased ventricle: brain ratio (Levy *et al*, 1984). Research involving a larger spectrum of possible predictors is needed both in respect of schizophrenic symptoms, such as anhedonic features or persecutory delusions, and of demographic, social, and anamnestic variables.

The present study examined the relationship of both suicidal behaviour and ideation to 143 variables, including anamnestic and sociodemographic variables, symptoms relevant for clinical assessment or research in schizophrenia, and the patients' diagnoses according to 12 systems for diagnosing schizophrenia, as outlined by Landmark (1982).

Subjects

One hundred and eighteen patients (46 men, 72 women) of the London Psychiatric Hospital in London, Ontario, participated; they were drawn from 120 patients examined in a series of studies (Cernovsky *et al*, 1985; Landmark *et al*, 1986). The mean age was 38.4 years (s.d. = 9.8). All had been diagnosed as schizophrenic at least once in hospital settings in the past, and all were classified as schizophrenic by at least one of the 12 systems for diagnosing schizophrenia described by Landmark (1982). At the time of assessment, all patients were stabilised on major tranquillisers for at least 12 months and were currently being treated with fluphenazine decanoate.

Method

The assessments were carried out on an individual basis by means of a diagnostic checklist and ratings described by Landmark (1982). The ratings provided the basis for the diagnosis according to 12 leading systems for diagnosing schizophrenia, while still another diagnosis was added by using Bleuler's (1920, 1950) system, with a cut-off score of three instead of four of his four criteria. The checklist also included quantitative information on a variety of sociodemographic and anamnestic variables: those included in the present study have been described in more detail by Cernovsky *et al* (1985). Ratings for all variables were based on interviews with patients and relatives as well as on data from hospital files.

Suicidal tendencies were retrospectively scored as follows: no suicidal thoughts or attempts (1); suicidal thoughts – rarely (2); suicidal thoughts – frequently (3); attention-seeking attempt – once (4); attention-seeking attempt – twice or more (5); serious attempt – once (6); and serious attempt – twice or more (7). The questions employed in conjunction with this scale are given by Landmark (1982). An alternative grading system was also created for suicidal attempts: no known history of suicidal thoughts or serious attempts (1); serious attempt – once (2); serious attempt – twice or more (3). The data for the latter grading system were examined for a subsample of patients (20 men, 39 women; mean age = 40.7, s.d. = 10.1 years) after patients with known suicidal ideation or attention-seeking attempts, but free of serious attempts, were excluded. This variable was defined to study the associations of serious suicide attempts independently of the confounding element of possible attention-seeking bahaviour.

The magnitude of the relationships of our two key variables – suicidal tendencies and suicide attempts – to diagnostic, anamnestic, and sociodemographic variables was evaluated by means of Kendall's tau. The analyses were exploratory: only a few expectations were held, and more than 200 relationships were examined. The criterion of significance was set at the 1 in 100 level (two-tailed).

Results

Serious suicide attempts were known in 27 patients (23%), and 14 (12%) had made repeated attempts. Attention-seeking suicidal behaviour was observed in 25 patients (21%), less than half of whom (11 patients) had made repeated attempts. Thirty-four of the remaining 66 patients (29% of the total sample) had a known history of suicidal ideation: the thoughts with suicidal content were rated as frequent in

TABLE I

*Correlates of suicidal tendencies and attempts, using Kendall's tau**

Correlate	*Suicidal tendencies (n = 118):τ*	*Suicide attempt (n = 59):τ*
Number of admissions for psychiatric problems	+0.27**	+0.42**
Number of times diagnosed as schizophrenic in the past	+0.23**	+0.35
Over-reaction to anxiety[1]	−0.23	
Present abuse of marijuana[2]	+0.23	
Mood alterations and fluctuations	+0.22	
Poor work function[3]		+0.35
Number of different addresses	+0.22	+0.34

*Only coefficients significant to $P < 0.01$ (2-tailed) are listed; $^{**}P < 0.001$. Information was not available for all patients on some of the variables, hence apparent inconsistencies in significance levels.

1. Coded: yes = 1, no = 2. 2. Coded: none = 0, 1–3 times/month = 1, 1–2 times/week = 2, more than twice/week = 3. 3. Coded on a scale of 1 to 4; 1 = good, 4 = poor (Landmark, 1982).

22 of these (19% of the total sample). Only 32 patients (27%) were free of a known history of suicidal ideation or attempts.

Only a few of the relationships evaluated were significant (Table I). The number of past hospital admissions was found to be the most salient marker for both suicide attempts ($\tau = 0.42$) and for suicidal tendencies ($\tau = 0.27$). In general, the results suggest that chronic patients may be more likely to show frequent suicide attempts or tendencies: they were more frequently admitted to hospital and diagnosed as schizophrenic, and had longer histories of schizophrenic symptoms. The work functioning of those who attempted suicide was poorer. Suicidal tendencies were associated with over-reaction to anxiety, mood fluctuations, and with a history of marijuana use. The first of these suggests impulsive maladaptive ways of dealing with anxiety-provoking situations. Marijuana use has similarities to suicidal tendencies, in that both can be forms of avoiding more direct coping with reality.

Suicide attempts or tendencies were unrelated to the incidence of other symptoms and to other sociodemographic, anamnestic, or diagnostic variables, including the diagnoses according to all the systems for diagnosing schizophrenia. No relationships were found to age, gender, or educational level.

Discussion

The findings of more frequent past psychiatric admissions, frequent changes of address, and over-reaction to anxiety confirm the reports of other investigators (Planansky & Johnston, 1973; Roy, 1982; Roy *et al*, 1984). However, in contrast to findings by Roy (1982), no significant relationship of suicide attempts or tendencies to the incidence of depressive symptoms (item 138 in Landmark's (1982) diagnostic checklist) was found in our analyses. Similarly, in contrast to a study by Brier & Astrachan (1984), no relationships were found to age, gender, or marital status, and in contrast to a study by Drake *et al* (1984), the educational level did not emerge as a significant risk marker. Further research is needed on this point.

Suicidal behaviour in schizophrenic patients is a major problem: 44% in our sample and 55% in the sample studied by Roy *et al* (1984) had a history of suicide attempts. This is probably not limited to our samples or to North American settings; for example, Hessø (1979) reported similar findings in Norway. There is some evidence that suicide rates in psychiatric patients, especially in functional psychoses, may have been rising in the last two decades (Hessø, 1979; Retterstøl, 1978). It is therefore important to identify predictors of suicidal risk. However, our search for such predictors is obstructed by an unexpected methodological problem: our most salient potential predictor was chronicity, in terms of repeated psychiatric admissions and repeated diagnoses of schizophrenia. Although this is consistent with past research, the variable is not necessarily a valid predictor of suicidal risk. The relationship could be spurious, or could be interpreted in a manner which reverses the causal sequence. Suicidal ideation and attempts may be more likely to be known for patients with more sustained contact with psychiatric institutions, simply because more information about the patient is likely to accumulate. The time span involved may be a confounding factor both in our findings and in those reported by Roy *et al* (1984).

If chronicity is an important factor, the correlation coefficients for the relationships of suicidal tendencies (or suicide attempts) to the duration of the longest stay in hospital and to the total number of months in hospital should be positive and significant. In our study, the coefficients involving these variables were close to zero and not significant (Pearson's $r = 0.02$ and 0.14 respectively for suicidal ideas, and $r = 0.01$ and 0.05 for serious suicide attempts).

Another manner of demonstrating the confounding variable is to examine the coefficient found between suicidal tendencies and chronicity of symptoms. A partial correlation coefficient computed for the relationship of the two variables, with control for the number of psychiatric admissions, was not significant at the level set ($r = 0.02$, $P = 0.038$, 2-tailed). The same was found with a coefficient controlled for the number of times the patient was diagnosed as schizophrenic ($r = 0.21$, $P = 0.027$, 2-tailed). Because of the nature of the study, we have required a more stringent level of significance than would otherwise be necessary; a replication study need only find a probability of 0.05, 1-tailed, in order to support the hypothesis .that chronicity has a relationship with suicide, notwithstanding the artefact which length of illness may produce. In fact, since there were several other findings which were all significant at a level of 1 in 1000 which related to suicidal tendencies or to suicide attempts and which arise from chronic effects, it seems likely that there is a true relationship between chronicity and suicidal tendencies or suicide attempts, notwithstanding some of our negative evidence.

It should also be noted that our key variable, the serious suicide attempt, is likely to be followed by re-admission, and thus these attempts are antecedents rather than consequences of the number of admissions. Hence, the causal sequence is reversed. To defend the opposite viewpoint, however, it may further be pointed out that a history of past suicidal attempts (or of ideation) is a good predictor of suicidal risk, at least in other populations (McCourt & Solomon, 1974).

Difficulties of interpretation associated with our study and with other past studies could be avoided if, for all patients in a sample, the time span during which the attempts are recorded were to be held constant. For example, chronic and less chronic patients could be compared, in a retrospective or prospective design, in respect of the incidence of suicide attempts within a specific time span such as two years. It is possible that chronicity, or the number of past hospital admissions and other related variables such as previous history of suicide attempts, would then be shown to be risk markers.

The main contribution of this study is thus the confirmation of the association of frequent admissions to psychiatric hospitals with the incidence of suicide attempts or tendencies in schizophrenia, and in demonstrating that this finding could be spurious or based on a reversed causal sequence, i.e. that suicidal attempts are followed by admission.

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Suicide and Social Response in Fiji: A Historical Survey[1]

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A historical review of suicide in Fiji shows that rates for people of Indian descent have always been higher than those of other groups, yet until now this phenomenon has attracted little official or public attention. Recent changes in the incidence and methods of suicide have led to the introduction of some preventive measures, but the emphasis has been on eliminating some of the methods rather than the reasons for suicide. The continuing high incidence of suicide by hanging involving some sectors of society appears to have been overlooked. Possible causes of this apparent lack of interest are examined, such as the low status historically of Indian immigrants and concepts held about them by other groups in Fiji, and the 'invisibility' of groups of people such as rural women and the aged.

In a study of suicide in Macuata, Fiji during the period 1979–82, the circumstances of suicide were discussed (Haynes, 1984). The present study examines the social and official response to this phenomenon over a 100-year period and attempts to define the factors which have shaped that response.

Historical background

Indians were first brought to Fiji under the Indenture system, which was introduced in 1879 and ended in 1920: this followed the pattern already established in the Caribbean, South Africa, and Mauritius to provide cheap labour for the sugar industry. The abuses of the Indenture system have been well documented both by contemporary writers (Andrews & Pearson, 1916) and by modern historians (Tinker, 1974; Gillion, 1962; Ali, 1979). In most respects the conditions of Indenture were little better than those of slavery, except that the contract (or 'indenture') was for a limited period of five or ten years. In principle at least there was an end in sight, even if in practice many did not survive the system.

High suicide rates were acknowledged as early as 1871, when Sir Arthur Gordon (successively the Governor of Trinidad, Mauritius, and Fiji) commented in a letter to Lord Kimberley on "the extraordinary frequency of suicide amongst Indian immigrants of this colony"; he was referring here to Mauritius, but the comment was equally true of other territories. This high rate he attributed to a low ratio of female to male immigrants, leading to rivalry and jealousy and to "nostalgia and the intense desire to return to India" (Tinker, 1974).

Suicides were frequently recorded at the first stage of recruitment, when Indians were collected in large depots at the ports of Calcutta and Madras and the realisation of what recruitment meant dawned on them – especially for the many who had been recruited fraudulently. As Andrews & Pearson (1916) recorded: "Many throw themselves into the Hughli to escape the emigrant ships". Interviews with those who had experienced the Indenture system showed that many had no idea that the contract involved travel overseas, and some were merely escaping minor problems in their villages or were seeking work in India itself (Ali, 1979; Naidu, 1980).

Sea voyages from India to various destinations in the Caribbean and Pacific might last 3–6 months, and the physical hardships associated with these voyages have been compared with the middle passage of slavery. Some emigrants threw themselves overboard during the voyage, overcome by despair and in many cases by the fear engendered by crossing the ocean, which meant defilement and loss of caste for Hindus. Commenting on these suicides, a medical officer in charge of one voyage in 1873 declared: "This amusement is getting rather too common" (Tinker, 1974). He was later dismissed, but his comment reflected, to some extent, contemporary attitudes towards emigrants. Disease was rife on these voyages and mortality rates high, but measures taken to control disease were motivated more by economic considerations than by thought for the well-being of the emigrants.

After arrival at their destinations, many more Indian emigrants committed suicide. "Of all aspects of sickness and mortality on the plantation, the most sombre was that of suicide. . . In the depression of the

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[1] Based on a lecture presented at the spring Conference of the British Medical Anthropology Society, March 1985.

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first year or two of indenture many sought to end their existence" (Tinker, 1974). Suicide figures for Fiji were particularly high compared with India itself and with other colonies where the Indenture system existed. Andrews & Pearson (1916) give suicide rates for Madras of 4.6 per 100 000 and for the United Provinces of 5.4 per 100 000; these were the main areas of recruitment. For Natal the figure was 64 per 100 000, and for Fiji they gave figures of 78 and 83.1 per 100 000 for the years 1903 and 1910 respectively. By 1915 rates in Fiji had reached a peak, and Andrews (1915) wrote in a letter to Lord Hardinge that "one in every 900 indentured coolies committed suicide – one in every 900!"

Drawing on statistics from the Fiji Royal Gazette, Naidu (1980) showed that the incidence of suicide was high in 1902–03 and again from 1911–15. In 1912 there were 18 suicides recorded among 15 369 indentured workers, in 1913 there were 20 among 15 961, and in 1915 there were 18 among 14 326, all giving rates of around 120 per 100 000. Some of these periods coincided with an influx of workers from South India, recruited in Madras, who often felt isolated in a society dominated by workers from North India. The Immigration Department of Fiji commented: "For what appeared to be inadequate reasons, Madrasis appeared to be prone to suicide and depression when placed among Northerners" (Gillion, 1962).

The Indenture system was characterised by violence of all kinds, and Fiji's reputation was seen as worse than most. Ali (1979) and Naidu (1980) interviewed over 40 elderly Indians who had been recruited, and graphic accounts were given of violence between employers and workers, and between workers themselves; of murder, manslaughter and suicide. Many mentioned the disproportion of the sexes (the male:female ratio was 3:1 in Fiji) as a major cause of violence, leading to constant rivalry and jealousy. But the general conditions which generated despair and nostalgia, the loss of intimate shipboard friends, ill treatment by those in authority, overwork, illness, sheer weakness, and exhaustion all contributed to suicidal tendencies.

It is likely that suicide and other forms of violence were under-recorded, workers fearing reprisals if crimes were reported and also having little faith in the current system of justice. From the planter's perspective it was in his interest to "hush up serious crime on his estates... The temptations to hide facts which might tell against the system must have been great indeed" (Andrews & Pearson, 1916). Planters maintained that workers were content with the lowest conditions, which were in practice often attributed to the Indians' alleged inferiority rather than to plantation mismanagement. "The moral degeneration of the 'lines' was seen by the planters and overseers as a reflection of the character of the recruits and the class they came from in India" (Naidu, 1980).

The stereotyping of Indians by contemporaries was almost invariably negative. Planters and administrators could best justify the system by denigrating the Indian character, and such stereotyping lasted long after Indenture ended. Native Fijians were also hostile; fears that Indians would eventually outnumber them were expressed by the Great Council of Chiefs in 1888: "The Indians in course of time will bring about a change in the manners and customs of our land... and will stamp us out... Presently we shall be blotted out by the number of these people who keep coming. Let them go back to their own country when they have finished their work" (Gillion, 1962). Today Indians outnumber native Fijians and are the largest ethnic group in Fiji. As such they may still be seen as a political and economic threat to Fijians, a factor not always conducive to harmony.

Figures are lacking for the period 1920–1960, but the consistency of later findings suggests that Indian suicide rates have continually been high, even though the reasons may have changed. Certainly the legend appears to have evolved that Indians are prone to suicide; this may well have affected the perceptions of the public and of officials such as coroners.

It should be noted here that Indian society in Fiji is far from homogeneous. 'Indian' usually means the Hindi-speaking descendants of emigrants from North-eastern India; occasionally it includes Tamils and Telegu from Southern India, Muslims, and Gujeratis (Jayawardena, 1980). Gujeratis, Sikhs and Punjabis all came to Fiji after the Indenture period, and their descendants today dominate the commercial life in major towns. Records kept by police and medical departments do not normally distinguish between these groups which are all classified as Indian. In this context ethnicity alone may be much less important than differences in culture and customs associated with these groups.

Suicide today

Studies dating from the early 1960s are consistent in showing that Indians are over-represented among suicides in Fiji, accounting for approximately 90% of all victims where they comprise just over 50% of the total population. The studies cover the periods 1962–68 (Ree, 1971), 1971–1972 (Karim & Price, 1973), and 1979–82 (Haynes, 1984). Those by Ree and Haynes were based on police records in

Macuata; that by Karim & Price was based on medical records for all of Fiji.

In all three periods suicide was found to be associated with Indian descent, Hindu faith, rural residence, youth (especially of females), joint family units (i.e. the extended family living in one homestead), an absence of diagnosed mental illness, and interpersonal conflict within the family. In all three periods hanging was the commonest method of suicide, accounting for 95% and 92% of all cases in the two earlier periods and for 74% in 1979–82; in all periods a larger percentage of women than of men died by hanging. The greatest changes in the past twenty years have been in the total number of recorded suicides (an increase of 80% since 1972, compared with a population increase of 19%), and in a change of method.

The studies by Ree (1971) and Haynes (1984) were conducted in the rural province of Macuata, and were based on police records of unnatural deaths which had received verdicts of suicide in the Magistrates' Court. These studies were comparable with respect to the total number of suicides, the distribution by age, sex, and ethnic origin, and also to the circumstances of suicide. In 1962–68 only 4 out of 73 suicides were by non-Indians, and in 1979–82 only 3 out of 69 (in Macuata province 75% of the population is of Indian origin).

In both periods, 82% and 83% respectively of female suicides were under the age of 30, and almost all of these died by hanging. Samples were generally too small for age-specific rates to have any significance, but in both periods there was a larger than expected number of Indian women aged 15–30.

Haynes (1984) gave the following rates per 100 000 for Indians in Macuata: males aged 10–29 years, 36.1; females aged 10–29 years, 74.8; males aged over 30 years, 70.5; females aged over 30 years, 28.2. This shows that young women and older men were particularly at risk of suicide. This is in contrast to rates for Fiji as a whole, where the rate for all Indian women in 1981–82 was only 33.1 per 100 000 and for all Indian men 45.2 per 100 000 (Table I).

Conflict within the family particularly between parents and children, and between young married women and their in-laws, appeared to be a major precipitating factor among female suicides. Childlessness was noted in 14 out of 28 married women in Ree's study, and was noted as a factor by Haynes where in-laws or spouses had taunted young women for failing to produce sons.

Family conflicts also featured in male suicides, although to a lesser degree, and some suicides appear to have been precipitated by shame, debt, or fear of disgrace or failure. Among older men (aged 60–69 years), whose high rate boosted the figure for all men over 30, suicide was usually preceeded by physical or mental illness (Haynes, 1984). This is a group for whom three old peoples' homes were established in Fiji, as so many became destitute in old age when they were no longer economically useful. Even those with families could be rejected in old age, and a survey of old peoples' homes in 1974 showed this group to be particularly vulnerable (Haynes, unpublished).

TABLE I

Suicides in Fiji, 1971–72 and 1981–82 (annual rates per 100 000)[1]

Period	*Sex*	*Indians*	*Fijians*
1971–72[2]	Males	24.9	2.1
	Females	23.1	0.7
1981–82[3]	Males	45.2	9.2
	Females	33.1	9.3

1. Rates are based on the population figures given for adults (aged 15 and over) in the 1966 (for 1971–72) and 1976 (for 1981–82) censuses. Estimates suggest that populations in 1971 and 1981 are each approximately 10% greater than these figures; the rates given are therefore slightly high.
2. Karim & Price (1973).
3. Haynes (1984).

The most recent change has been in the method of suicide. Karim & Price (1973) reported that in 1971–72, 92% of all suicides were by hanging; the remaining 8% used firearms, cutting instruments or drugs. By 1979–82 only 77% of female and 69% of male suicides in Macuata were by hanging, and paraquat was beginning to be recorded as a suicide agent. This is marketed in Fiji under the name Gramoxone, and is in common use on cane farms where it is recognised as an effective herbicide, safe when used correctly and quickly becoming inert on contact with the soil. Ingestion of 10 ml (i.e. one mouthful) can prove fatal, and deaths may be painful and lingering.

Records show that between 1981 and 1985, 177 people were admitted to hospital with paraquat poisoning, only 70 of whom survived. Approximately two-thirds of the 107 who died were suicides, and most of these were Indians aged 15–30 years (Vuetivavalagi, 1985). However, police records showed that the majority of suicides were still by hanging: in 1983 there were 60 by this method, and 23 by paraquat poisoning (Royal Fiji Police, 1984).

In examining methods of suicide it is important to remember that some of those who previously attempted suicide by poisoning probably survived. Paraquat was introduced into Fiji in 1972 (under the Pesticides Act 1971), but appears not to have been abused for purposes of suicide until much later. Attempts made with less toxic chemicals were less likely to be fatal, whatever the intention, and some of these who in the past were rescued or resuscitated may in fact have intended to die.

Intention in cases of poisoning can at best only be surmised, whereas hanging is a method rarely expected to fail. Deaths from poisoning were more likely in Fiji to be followed by an inquest than those resulting from hanging. Lack of evidence has frequently led to verdicts of accidental death, even where suicide has been suspected (Fiji, 1978).

It is also possible that Coroners' verdicts might be affected by the 'legend' of Indian suicides, and that a verdict of suicide is more likely to be given to an Indian than to a Fijian where circumstances of death are similar.

Social response

The official and public response to this new phenomenon has been remarkable in contrast to the apparent acceptance shown towards Indian suicides over the past 100 years.

Attempted suicide is still an arrestable offence, and official attitudes are somewhat punitive. A police officer may arrest and send for trial any person whom he suspects of attempting or preparing to attempt suicide; the normal sentence is one of probation (Fiji Criminal Procedure Code, 1978). This legislation appears to be applied relatively infrequently; the author found that in the period 1979–82 only 15 of 65 people admitted to one hospital following a suicide attempt were actually placed on probation.

Health planners seem until now to have overlooked the question of suicide, and mental health generally received scant attention in development plans. In the Eighth Development Plan (1981–85) priority was given to physical and environmental health, sanitation, nutrition, child health, and family planning; mental health received a passing reference, and suicide none at all (Central Planning Office, 1980).

Although suicide rates for Indians have always been high compared with other ethnic groups, the recent and sudden increase in deaths due to paraquat abuse has created widespread alarm. Since 1983, when such deaths were first publicly acknowledged, a series of conferences and seminars have been devoted to this problem. Representatives of the Ministries of Health and Agriculture, the police, the Consumer Council, and of many other departments have examined all aspects of paraquat use and abuse. Controls over the importation and distribution of paraquat, the feasibility and costs of adding emetics and stenching agents, the deterrent effects of such additives, and the treatment of symptoms have all been discussed. The need for stringent controls on the sale of agricultural chemicals, using only licensed retail outlets, is universally accepted. A major problem arises when paraquat is bought in bulk and transferred by farmers into unlabelled bottles, lacking instructions as to use, precautions or antidotes. Such careless storage methods have been responsible for a number of deaths, when victims have drunk impulsively from the nearest bottle – sometimes when intoxicated.

By July 1985 paraquat deaths had become the main subject of debate in Fiji's Senate. Some senators called for a complete ban on the importation of paraquat, and others for tighter restrictions on its sale and distribution. There was also discussion on the role of the media, one senator suggesting that the publicising of suicide only encouraged others to take their own lives, and another that such reporting would have a deterrent effect (Vuetivavalagi, 1985). For the first time it was acknowledged that more people died by hanging than by poisoning, and it was humorously suggested that perhaps there should be similar restrictions on the importation of rope!

Frequent allegations have been made that pesticides banned in the USA and Europe were being 'dumped' in Fiji and other parts of the Pacific. Such allegations were firmly denied by the Registrar of Pesticides in April 1984, when he reiterated that all pesticides entering Fiji were registered and that if used correctly they constituted no danger to man. He acknowledged that "the one serious problem is intentional suicide, because you can't stop that" (Keith-Reid, 1984).

The role of the media in publicising the often horrifying consequences of ingesting paraquat cannot be ignored. From early 1983, when the first editorial warned against the dangers of paraquat, there have been numerous articles in both English language and vernacular newspapers, and in professional and regional journals. Dramatic headlines and front page coverage have ensured that the public was made aware of the problem: examples are 'Poison deaths alarm doctors' (Lakhan, 1983), '107 die from paraquat' (Vuetivavalagi, 1985), and 'Chemical war in Fiji' (Keith-Reid, 1984). Almost all suicides are reported in the press; names of victims and circumstances are given, and some articles have given detailed accounts of paraquat deaths (Fiji Times, 1984). While the intention underlying such extensive coverage may have been to educate the public, its effects appear to have been far from deterrent, especially with respect to choice of suicide agent.

Quite apart from the influence of the media, the effects of a suicide on any small community must be considerable. In 1962–68 and 1979–82 only one of 142 reported suicides took place within the urban area of Labasa; all the others occurred in farming settlements of less than 1000 people or on relatively isolated cane farms. In one such settlement there were seven suicides in four years, and there were many more in a group of settlements in a cane farming valley (Haynes, 1984). Any suicide in these areas is likely to become common knowledge and perhaps affect the actions of others under stress.

The most striking feature of social and public response has been the emphasis, to the exclusion of all else, on the topic of paraquat: its toxicity, availability and control. While attention to this problem is essential, it is apparent that other suicides pass almost unnoticed: anecdotal accounts of suicides by hanging continue to be featured in the press, but these cases appear to merit little comment.

Discussion

A number of factors may have influenced official and social responses to suicide, and possibly prevented appropriate action from being taken.

It has been seen that Indians were historically accorded low status, and that stereotyping of this group by others may be a legacy of the Indenture period. Suicide was likely to be seen as proof of instability – of inferiority, rather than as a legitimate response to appalling conditions. The causes of suicide were neglected along with other physical causes of mortality. Today the causes of suicide may have changed, but some of society's responses have not.

Fiji is a plural society, in which there has been little assimilation except at a superficial level. The association between suicide and the Indian population has become almost a legend, a possible consequence of which is the neglect by the rest of society of what is seen as a peculiarly Indian problem. There has been much comment, especially by the media, but little action.

Not only have high suicide rates been overlooked by society at large, but within Indian society the lack of homogeneity prevents one group from identifying with the problems of another. For example, the problems of the Hindu farmer may have little meaning for the Gujerati businessman or for Muslims in the city. In the same way, men (who are dominant in this society) are unlikely, for various reasons, to champion the causes of women. Although Indians today comprise the largest ethnic group, they are still a political minority and until recently have been under-represented among government officials, the professional classes and those involved in health planning. Such factors may contribute to the failure of society to address itself to this problem. Where high suicide rates appear to affect a majority or ruling group, or where society is homogeneous, rising suicide rates represent a threat to the whole society and official response may be swift and all-embracing, as has happened in Western Samoa.

Within Indian society, especially in rural areas, young women are sometimes accorded low status, as are elderly men who have outlived their usefulness economically, and there may be few representatives of these groups who can voice their problems. Recent studies have shown high suicide rates (mostly involving hanging) for these groups, yet these deaths appear to have been overlooked by those involved in the latest preventive campaigns: these are the 'invisible' suicides.

The shame and guilt traditionally associated with suicide may also create something of a taboo, which prevents comprehensive study of this subject. Families involved often wish to deny or forget a suicide, especially if it has been inferred that they were in any way responsible. Among Muslims and Christians, religious prohibitions concerning suicide may also lead to denial or to a diminished response.

In recent years the spate of paraquat deaths, whether suicidal or accidental, has focused attention on treatment and preventive methods, and society has demanded an immediate solution. Efforts have been concentrated on controlling the supply of toxic substances or even of banning them altogether, and this type of research has overtaken investigations into the causes of suicide.

Official response has also been fuelled by the international scandal over pesticides. Paraquat abuse has been reported from as far afield as Trinidad, Thailand, Western Samoa, and Malaysia where, apart from suicides, there have been many accidental deaths (Reiss *et al*, 1983). The anti-pesticides lobby has been active in Fiji as well as in other parts of the world, and its allegations have required a response – if only in defence.

It is understandable that the initial public response is to seek a solution reasonably certain of success. If it is true that people in the past attempted suicide by poisoning but survived and the availability of a new and more potent toxin has resulted in more fatalities, then perhaps the logical step is to ban that substance. Such action may be effective in preventing both accidental and suicidal deaths from paraquat, but it fails to acknowledge the majority who die by hanging. Ultimately the question that should be asked is not "How?" but "Why?"

Further studies are needed, preferably conducted by local scholars familiar with the language and culture, into all groups at risk, into the psychodynamics of family life, and into different behavioural responses to stress. Comparisons need to be made between rural and urban families and between different religious and ethnic groups. Suicide is likely to remain a major problem until the reasons underlying it are systematically studied.

A possible preventive programme might be modelled on experience obtained in Western Samoa. Until the early 1970s the incidence of suicide was around six cases/year. Between 1972 and 1982 the incidence rose steadily to a peak of 49 cases in 1981 – mostly suicides by young men who had ingested paraquat. The 1981 rate for men aged 15–24 years was 94 per 100 000 (Keith-Reid, 1983; Oliver, 1983). A major campaign was mounted to examine the reasons for suicide, and a multi-disciplinary group representing the police, the judiciary, health and education departments, churches, and villagers initiated a public awareness campaign. Public meetings were held and careful use made of the media to educate the public. Knowledge of local custom meant that media coverage was tightly controlled, as it was felt that overdramatic accounts might actually attract some youths to a test of endurance like that once provided by full-body tattooing (now forbidden by law). Such a campaign, which gives recognition to local custom, might be successful elsewhere; it has certainly been effective in Western Samoa, where only five paraquat suicides were recorded in 1984.

Little will be achieved until the communities most affected acknowledge the problem and are motivated for change. Women's organisations and Indian communities in general need to be involved in planning and to make sure they have a voice. To start with, urban and educated people who have access to such groups may be needed to represent the more isolated and weaker members of society. While these groups can help, the ultimate responsibility lies with central government to ensure that sufficient resources are allocated to the understanding and resolution of this problem.

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Psychological Correlates of Unemployment Among Male Parasuicides in Edinburgh

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Variations in clinical features associated with unemployment among a sample of male parasuicides were examined. Employed and unemployed persons did not differ in mean scores on the Suicidal Intent Scale, but the unemployed were rated significantly worse on measures of depression (Beck Depression Inventory) and hopelessness (Hopelessness Scale). A covariance analysis showed that hopelessness, rather than depression, is the important discriminator. Different patterns of relationships between the three clinical measures were observed in the two groups. Hopelessness may be a key social-psychological variable for inclusion in any model of the pathways which link unemployment with parasuicide.

Numerous investigations carried out over the past 15 years have revealed more depression, anxiety, negative self-esteem, negative affect, psychological distress, and potential psychiatric illness among unemployed individuals, particularly men, than among those in paid employment (Warr, 1983; 1984). The cross-sectional design of the majority of these studies presents problems of causal interpretation, but there have been a number of longitudinal investigations (e.g. Banks & Jackson, 1982; Cobb & Kasl, 1977) which demonstrate the aetiological impact of unemployment on psychological well-being. Evidence of the role of joblessness as a contributory cause of severe morbidity is less convincing, but also points in the same direction. Roy (1981) found significantly more unemployment among depressed male patients than among control patients. In their community survey in London, Bebbington *et al* (1981) identified 6% of employed males as psychiatric cases, compared with 14% of unemployed males. On the basis of psychiatric interviews with 72 unemployed young Australians, Finlay-Jones & Eckhardt (1981) concluded that in 43% of those with a severe psychiatric disorder the onset followed unemployment in the absence of any other recent severe stress. In a further 23% of cases the onset of disorder also followed unemployment, but these individuals had also experienced at least one other severely stressful life event in the three months prior to onset.

Fruensgaard *et al* (1983) estimated that for 41% of their sample of 70 unemployed patients admitted to a psychiatric department in Odense, Denmark, unemployment had played an important part in their current psychiatric illness, although for only 11% was it the sole causal factor.

Strong evidence of a significant association between completed suicide and unemployment rates, both cross-sectionally and over time, is also available (Platt, 1984). One of the few studies of trends in unemployment and parasuicide (Platt & Kreitman, 1984, 1985) revealed that the risk of parasuicide among unemployed men in Edinburgh was 11 times that of men in work in the same city. Those most at risk were the long-term unemployed. Platt & Kreitman concluded that unemployment (especially prolonged) is an important aetiological factor in parasuicidal behaviour.

The present study examined variation in clinical features associated with unemployment among a sample of economically active male parasuicides (i.e. excluding students and retired persons). It is known that depression and hopelessness are significantly associated with parasuicide (Weissman, 1974; Wetzel, 1976) and with suicidal intent (Minkoff *et al*, 1973; Beck *et al*, 1975). The major question which this study seeks to answer is whether unemployed parasuicides will report more depression and hopelessness, and a higher level of suicidal intent, than employed parasuicides; i.e. will the finding of deficits in psychological well-being among the unemployed in the general population be repeated in a sample of parasuicides? A further objective is to examine the inter-relationship of hopelessness, depression, and suicidal intent within subsamples of parasuicides differentiated by their employment status. Dyer & Kreitman (1984) found that hopelessness was the essential mediating variable, correlating significantly with intent irrespective of level of depression. Their findings replicate those reported in a number of American studies (e.g. Minkoff *et al*, 1973; Beck *et al*, 1975; Wetzel *et al*, 1980). We were interested to ascertain whether these same inter-relationships were also evident in each employment status group separately.

Method

The method and subjects of this study, which was carried out at the Regional Poisoning Treatment Centre at the Edinburgh Royal Infirmary in 1979, have already been described (Dyer & Kreitman, 1984). Briefly, 120 parasuicides were interviewed, 30 in each of four age-sex groups. All cases were of parasuicidal self-poisoning, defined as a non-fatal act in which an individual deliberately ingests a substance in excess of any prescribed or generally recognised therapeutic dosage (Kreitman, 1977). The extent of suicidal intent, if any, was not considered when the 'diagnosis' was made. For the purposes of the present analysis only the 50 economically active males from the original sample were considered. Twenty-five were unemployed and 25 employed at the time of admission.

Subjects were given a structured interview in which data were collected on demographic, clinical, and treatment variables. This paper focuses on results obtained using three instruments which operationalise the key concepts of hopelessness, depression, and suicidal intent. Hopelessness was described by Beck in terms of a system of cognitive schemata that share the common element of negative expectations regarding the world, the self, and the future. The Hopelessness Scale (HS) (Beck *et al*, 1974*a*) is a self-rating scale consisting of 20 statements of thoughts or feelings about the future which the subject rates true or false. For every statement (of which 9 were keyed false and 11 keyed true) each response was assigned a score of 0 or 1, and the total hopelessness score (maximum = 20) was the sum of scores on individual items. Degree of depression was assessed by the Beck Depression Inventory (BDI) (Beck, 1970), a self-report instrument which measures depression as a syndrome rather than as a mood or diagnosis. The total score (maximum = 63) reflects both the number and severity of 21 possible symptoms or items (each rated on a scale of 0–3). Both the HS and the BDI were modified by rewriting items in the past tense and adding the word 'recently' in order to help subjects recapture their feelings prior to the parasuicidal act. Suicidal intent was conceptualised in terms of the relative weight of the patient's wish to live and wish to die, and the degree to which these suicidal wishes were transformed into a concrete act orientated towards death. The Suicidal Intent Scale (SIS) (Beck *et al*, 1974*b*) is a 15-item questionnaire, each item scoring 0–2 (therefore with a possible maximum of 30). The first part of the SIS ('Circumstances', consisting of 8 items) is concerned with the circumstances of the act; the second part ('Self-report', consisting of 7 items) assesses the subject's thoughts and feelings at the time of the act.

All data analyses reported in this paper were obtained using the Statistical Package for the Social Sciences (SPSS) (Nie *et al*, 1975). The statistical significance of differences between employed and unemployed groups was assessed by means of the χ^2 test using Yates' correction, where appropriate (categorical data), or the t-test (continuous data). Covariance analyses were performed in order to assess the main effect of employment status on hopelessness, controlling for the effect of depression, and on depression after partialling out the effect of hopelessness. The association between the major variables was represented throughout by the Pearson product-moment correlation coefficient. Partial correlation coefficients were also calculated in order to describe the relationship between two variables (e.g. suicidal intent and hopelessness) while adjusting for the effects of a third (e.g. depression). All P values are two-tailed, and the significance level (α) was set at 0.05 throughout.

Results

Compared with the rest of the economically active male parasuicides admitted to the RPTC during the same year, the study subsample ($n=50$) did not differ significantly in mean age, proportion reporting a previous hospitalised parasuicide, marital status, or social class breakdown.

Within the study sample, a comparison of the employed and unemployed groups on a number of selected demographic and clinical variables revealed two significant findings. More of the employed ($n=9$) than unemployed ($n=2$) were diagnosed depressed (χ^2 (depressed versus rest) = 4.25, d.f. = 1, $P<0.05$), even though the numbers diagnosed psychiatrically ill (10 employed, 6 unemployed) did not differ significantly. In respect of personality diagnosis, significantly more of the unemployed ($n=14$) than employed ($n=4$) were rated abnormal (χ^2 (normal versus rest) = 5.58, d.f. = 1, $P<0.02$). The groups did not differ in respect of marital status (the majority were unmarried), social class (the majority were in manual occupations), mean age (36 years among the unemployed, 33 years among the employed), current psychiatric status (the majority were receiving no treatment), or psychiatric disposal (half the sample were receiving in-patient, out-patient or general practitioner after-care). The tendency for more of the unemployed ($n=14$) than employed ($n=8$) to report a previous hospitalised parasuicide did not reach statistical significance.

The mean score for all 50 men was 4.0 (s.d. = 3.0) on the Circumstances part of the SIS and 6.2 (s.d. = 3.7) on the Self-Report part. The overall mean of 10.2 (s.d. = 5.6) was close to the typical value obtained from similar samples of parasuicides. The mean BDI score for the subsample was 25.4 (s.d. = 10.8), corresponding to a clinical judgement of moderate severity in two earlier studies (Beck *et al*, 1961; Metcalfe & Goldman, 1965). The mean HS score was 11.7 (s.d. = 5.1), higher than that reported in five previous studies (Pokorny *et al*, 1975; Minkoff *et al*, 1973; Beck *et al*, 1975; Goldney, 1981; Wetzel *et al*, 1980).

Comparisons between employed and unemployed revealed no differences in relation to part or total SIS scores. On the other hand, the unemployed scored significantly higher (i.e. worse) on both the BDI and the HS (Table I). The association between employment status on the one hand and depression and hopelessness on the other was explored further in two analyses of covariance (Table I). After controlling for HS score, employment status was no longer significant ($P>0.04$) in explaining variation in the

TABLE I

Comparison between unemployed and employed groups on the BDI and the HS

	Unemployed ($n=25$)	*Employed* ($n=25$)	*Significance*
BDI			
Score (unadjusted)			
Mean	29.5	21.4	$t=2.84$,
s.d.	9.0	11.1	d.f.$=48$, $P=0.007$
Mean score			
adjusted for	26.6	24.3	$F=0.6$,
covariate (HS)			d.f.$=1$, $P>0.4$
HS			
Score (unadjusted)			
Mean	14.2	9.1	$t=4.06$,
s.d.	4.1	4.7	d.f.$=48$, $P=0.000$
Mean score			
adjusted for	13.3	10.0	$F=7.75$,
covariate (BDI)			d.f.$=1$, $P=0.008$

TABLE II

Correlations and partial correlations of HS and BDI scores with full SIS scores[1]

Scale	*All*		*Unemployed*		*Employed*	
	r	*P*	*r*	*P*	*r*	*P*
Hopelessness scale[2]	0.41	0.003	0.22	NS	0.61	0.001
Beck Depression Inventory[2]	0.34	0.017	0.44	0.029	0.25	NS
Hopelessness[3] (depression controlled)	0.28	0.056	0.00	NS	0.59	0.003
Depression[3] (hopelessness controlled)	0.13	NS	0.39	0.060	−0.10	NS

1. Pearson product-moment correlation coefficient, two-tailed test.
2. d.f.$=48$ (where $n=50$) or 23 (where $n=25$).
3. d.f.$=47$ (where $n=50$) or 22 (where $n=25$).

BDI score. However, the main effect for employment status remained highly significant ($P=0.008$) in accounting for variation in the HS score after partialling out the effect of the BDI score. Thus, the original finding of a significantly greater level of hopelessness in the unemployed group is confirmed, even after taking account of the higher BDI score among the unemployed; whereas the finding of a significantly greater degree of depression among the unemployed has been shown to be misleading, in that it reflects the higher level of hopelessness in this group; hopelessness, rather than depression, seems to be the important discriminator.

Scores on the BDI and the HS were highly correlated over the whole sample ($r=0.60$, $P<0.001$), and among employed and unemployed separately ($r=0.51$, $P=0.01$, in each group). The correlations between these scores and the SIS score, for the whole sample and for each group, are shown in Table II. Level of suicidal intent is significantly associated with both hopelessness and depression in the whole sample. However, somewhat different patterns of relationships are observed among the unemployed and employed groups: in the unemployed group depression is significantly correlated with suicidal intent, whereas hopelessness is not; among the employed, hopelessness is significantly associated with suicidal intent, but depression is not. However, formal statistical tests of the differences between correlations shown in each column of Table II (unemployed versus employed) shows that the only significant finding relates to the correlation between suicidal intent and hopelessness, controlling for depression ($r=0$ among the unemployed, $r=0.59$ among the employed; $z=2.2$, $P<0.05$).

Discussion

Before discussing the major findings of this study, attention should be drawn to a number of factors which may limit their generalisation. Firstly, results are reported for men only; clinical correlates of unemployment among female parasuicides may or may not be similar. Secondly, the rating of unemployment is limited inasmuch as no information was recorded on the duration of the unemployment spell, but evidence from Edinburgh (Platt & Kreitman, 1985) and Oxford (Hawton & Rose, 1986) in 1982 and from elsewhere in the 1960s (Smith & Davidson, 1971; Morgan *et al*, 1975) suggests that it is probably reasonable to assume that the majority of unemployed parasuicides in the present study had gone at least six months without a job. The final shortcoming of the study is the small number of subjects in the sample. However, it has been shown that the sample is broadly representative of all economically active male parasuicide patients admitted to the Centre during the year of study. The findings are thus probably generalisable to this larger population, which is itself fairly representative of all medically-treated parasuicides (Kennedy & Kreitman, 1973).

The first set of data which require comment are the comparisons on the key clinical variables of suicidal intent, hopelessness, and depression. The failure to differentiate between groups on the SIS is unlikely to have been caused by any social desirability effect, since employed and unemployed were rated similarly on the objective Circumstances part as well as the more subjective Self-Report section. Had the sample recorded high levels of suicidal intent, then any employment status effect might have been masked or attenuated by a more powerful underlying psychopathology. However, the majority of both groups were not given a psychiatric diagnosis and SIS scores tended to be moderate.

The results relating to depression and hopelessness were in the expected direction, with higher scores (i.e. worse outcome) among the unemployed. Covariance analysis supports the robustness of the finding relating to HS score, but casts doubt on the independent status of the BDI result. The HS score is higher among the unemployed *despite* the fact that the BDI score is also higher, whereas the BDI score is higher in the unemployed group *because* hopelessness is more pronounced. The inconsistency between the BDI and clinical diagnosis results – whereas the unemployed were rated more depressed overall, there were more patients in the employed group given a diagnosis of depression – may be more appearent than real. The BDI is not intended to produce a clinical diagnosis, but rather to measure the behavioural manifestations of depression. There may be more depressive symptomatology overall among the unemployed, while a greater proportion of the employed are psychiatrically ill (i.e. clinically depressed). Nevertheless, we are mindful of Buglass's comments on the finding of a relationship between diagnosed depression and social class in a sample of parasuicides: the higher the social class, the more frequent the diagnosis of depression (Buglass, 1976). She argued that while this may reflect something about the patients' mental state, it may equally be that the clinical assessment patients receive is influenced by their social status. She referred to a study of medical in-patients by Schwab *et al* (1967), in which doctors appeared to show a marked bias towards a diagnosis of depression in upper-class patients which was at variance with test results using the BDI. We cannot dismiss the possibility that the patient's employment status may also influence doctors' clinical judgement.

The matrix of relationships between depression, hopelessness and suicidal intent was somewhat dissimilar between the two groups (Table II). Overall, the results mirrored those of the main study from which this sample was taken and of previous (mostly American) studies (Dyer & Kreitman, 1984). Among employed parasuicides the strength of the association between suicidal intent and hopelessness was particularly marked, while the correlation between suicidal intent and depression was not statistically significant. Among the unemployed, however, there was a significant association between suicidal intent and depression (which remained of borderline significance even after controlling for hopelessness) and no relationship at all between suicidal intent and hopelessness. This finding has only been reported in one previous study (Pokorny *et al*, 1975). Taken at face value, it casts doubt on the unequivocal assertion of Beck *et al* (1975) that "hopelessness accounts for the relationship between depression and suicidal intent... (It) is a stronger indication of suicidal intent than is depression itself". However, there is a risk of over-interpreting the importance of these differences. The number of subjects was small, and only one correlation (between suicidal intent and hopelessness, controlling for depression) was significantly greater in one group than in the other. Although further discussion would best be postponed until the findings have been replicated in a larger sample, extremely high levels of hopelessness may increase the risk of self-harmful behaviour without any discernible impact on the degree of suicidal intent.

The correlational analysis may raise questions about the relationship between the major clinical variables in the study. However, it does not undermine the outstanding finding of a significant relationship between hopelessness and unemployment. Hopelessness may be a key social-psychological variable for inclusion in any model of the pathways which lead from unemployment to parasuicide (or suicidal behaviour generally). Fifty years ago, clinicians drew attention to the impact of unemployment on attitudes to the future (e.g. Rundquist & Sletto, 1936; Israeli, 1935; for a more extensive review, see Eisenberg & Lazarsfeld, 1938). Jahoda *et al* (1933) differentiated the unemployed of Marienthal into four groups according to their prevailing attitudes. Apart from the 'unbroken', the remaining groups, while differing in some respects, shared at least one common feature – that of hopelessness.

Surprisingly, little attention has been paid to the prevalence or extent of hopelessness among the unemployed in the numerous more recent publications which utilise standardised psychometric tests to measure psychological well-being. Some tangential data are provided by Feather (1982), who showed that unemployed males tend to be more apathetic and lacking in interest than those in employment. Payne *et al* (1984) examined health, well-being, perceptions, and beliefs among middle-class and working-class white married men aged 25–39 who had been unemployed for 6–11 months. A high proportion reported anxiety about what would happen to them in the future, e.g. whether they would become permanently unemployable, lose their self-respect, become really depressed, lose the ability to make decisions, or have insoluble money problems. The working-class sample were significantly more fatalistic, exhibiting a higher degree of belief in external control. Warr (1984) proposes that insecurity about the future is one of the major features of the unemployed person's role which may account for the reduction in psychological well-being which he or she suffers. We believe that more attention should be paid to the assessment of hopelessness in employed and unemployed samples, and to an elucidation of its significance in the genesis of parasuicidal behaviour, particularly among the unemployed.

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Psychiatric Morbidity in Primary Health Care

A Two-stage Screening Procedure in Developing Countries: Choice of Instruments and Cost-effectiveness

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The use in two developing countries of a two-stage psychiatric screening procedure with an emphasis on minor morbidity is described. This was the first use of the Self-Reporting Questionnaire and Clinical Interview Schedule in conjunction. Health auxiliaries with limited training administered the first-stage screening instrument. In comparison with other psychiatric screening techniques used in developing countries, the procedure described may prove to be more cost-effective in primary care and community surveys, and more appropriate for assessing the wide range of minor psychiatric morbidity commonly encountered in such settings.

A series of psychiatric field surveys have been reported from the developing world since the 1960s, but such surveys, especially if carried out by teams of psychiatrists, use resources (in money, time and trained manpower), which developing countries can ill afford. Regrettably, the majority of these studies have utilised screening questionnaires that have not been adequately validated, and unstandardised clinical interviews for psychiatric assessment. An exception is that by Kapur (1975), in which the development and application of a multi-stage standardised screening questionnaire was reported in some detail.

This paper described the use of a two-stage screening procedure suitable for application in primary health care facilities and community surveys in developing countries. Two major issues will be discussed: the choice of instruments, and the cost-effectiveness of the procedure. This is illustrated with reference to two studies which were carried out in the cities of Calcutta in India and Sao Paulo in Brazil (Mari & Williams, 1986a).

Method

Study areas

Two-stage psychiatric screening was conducted in three primary care clinics in each city. The majority of patients were skilled, semi-skilled, or unskilled manual workers and hawkers, living mostly in slums or crowded housing with primitive sanitary conditions. More than half of the population in both areas were either illiterate or had not completed elementary schooling.

Research instruments

Self-reporting questionnaire (SRQ) (Harding *et al*, 1980). This consists of 25 items, and is divided into two sections: the first 20 items are designed to detect non-psychotic disorders, and the remainder to screen for psychotic disorders and epilepsy. A version consisting of the first 20 items was used for both the studies reported here; it usually takes 5–15 minutes to administer. For the Calcutta study, translation of the questionnaire from English into Bengali was carried out by the research psychiatrist (BS) and back-translated by a school teacher unacquainted with the original English version. The back-translation was subsequently compared with the original by BS and an English psychiatrist, to obtain semantic equivalence of the items. Minor discrepancies which had occurred necessitated slight modifications. A Portugese translation prepared for a WHO study (Harding *et al*, 1983a) was used in Sao Paulo.

Clinical Interview Schedule (CIS) (Goldberg *et al*, 1970). This is a standardised, semi-structured inventory designed for use in community surveys and general practice (Shepherd & Clare, 1981). Its feasibility in general practice and primary care settings has been tested in settings as varied as the UK (Goldberg & Blackwell, 1970), Brazil (Mari & Williams, 1984), Jamaica (Harding, 1976), Kenya (Dhadphale *et al*, 1983), Mexico (Medina-Mora *et al*, 1983) and Taiwan (Cheng, 1985). Its reliability has also been tested in a number of countries (Goldberg *et al*, 1970; Campillo-Serrano *et al*, 1981, Cheng *et al*, 1982, Mari *et al*, 1986). All operational terms in the CIS are carefully defined, and detailed criteria for rating provided in a manual. It is intended for administration by a psychiatrist standardised in its use. The CIS was translated into Bengali and Portugese in the manner described above. The interview takes 10–15 minutes for a healthy individual and 30–50 minutes for patients with psychiatric symptoms.

Collection of data

The Calcutta study was designed as a pilot project to validate the SRQ and establish its best cut-off point. The Sao Paulo study was part of a larger research project, for which a feasibility study had previously been conducted (Mari & Williams, 1984).

The Calcutta study was conducted during July and August, 1985, while the Sao Paulo one took six months (September 1982 to February 1983). Consecutive adult patients (aged 15 years and above in Calcutta, 16 years and above in Sao Paulo) attending the primary care clinics were screened with the SRQ (In Sao Paulo the General Health Questionnaire, GHQ-12, (Goldberg, 1978) was simultaneously administered). A random sample of 'probable cases' and approximately the same number of 'probable non-cases' were then interviewed using the CIS. The initial cut-off point for a 'probable case' for this purpose was decided on the basis of GHQ-12 scores in Sao Paulo (Mari & Williams, 1986a); in Calcutta it was taken as the median of the SRQ cut-off scores (7–8) determined by Harding *et al* (1980) and by Mari & Williams (1986a). The psychiatric interview was conducted 'blind', i.e. without knowledge of the patient's SRQ status. Patients who were too ill to answer the questionnaires (two in Calcutta; eight in Sao Paulo), and those who had already attended once during the exercise were excluded from the study. The psychiatric interview was refused by three respondents in Sao Paulo, but by none in Calcutta.

Basic sociodemographic data (age, sex, marital status, education, occupation and family income) were collected for each patient screened. The screening procedure was carried out independently of the usual consultation process, but any incovenience to the patients, e.g. having to wait too long for the psychiatric interview, was minimised. In general, patients selected for the psychiatric interview seemed happy to have two doctors for consultation instead of the usual one (the physician at the primary care clinic). The enthusiastic cooperation of both the clinic staff and the patients contributed considerably towards the successful completion of the projects.

Personnel and training

In Calcutta, three health workers, who each had 8–12 years of schooling followed by training in primary health care, carried out the first-stage screening. They received a 30-minute orientation session, during which it was ascertained that they understood the SRQ questions and the procedure required of them. In Sao Paulo, screening with the SRQ was conducted by a qualified and experienced social worker, who had had four years of university training; she received one week's training in administration of the SRQ.

Results

In Calcutta, a total of 202 patients were screened with the SRQ, of whom 114 (56%) were interviewed by the research psychiatrist. In Sao Paulo, 875 patients were screened and 260 (30%) were interviewed. There were no significant differences in either area in sociodemographic variables between the total sample and the subsample selected for interview. Thus, the subsample may be regarded as representative of the adult patients attending the primary care clinics in the respective cities.

Appendix 1 provides a summary definition of the statistics used to determine the validity of the SRQ as a screening instrument. Sensitivity (i.e. the proportion of cases correctly identified by the screening test) is said to indicate the 'utility' (the extent of benefit) of a test, whereas the proportion of false negatives and false positives, which together represent the overall misclassification rate (at a given prevalence), are representative of the 'cost' of a test. The 'cost' constitutes the cost of referral of the false positives (for the individual and for the physician) (Skuse & Williams, 1984). The missed cases or false negatives also represent a 'cost' for the subjects concerned, and may be estimated in terms of work lost due to lack of treatment.

The positive and negative predictive values are more useful in determining the 'utility' of a questionnaire for screening purposes. They measure the extent to which the test succeeds in *predicting* psychiatric cases or non-cases

TABLE I
Cross-tabulation of observed and scaled-up SRQ and CIS results

SRQ finding	*CIS finding*		*Total sample*	*Expected CIS finding for total sample*[1]	
	Positive	*Negative*		*Positive*	*Negative*
Calcutta					
Positive	38	12	105	80 (*A*)	25 (*B*)
Negative	14	50	97	21 (*C*)	76 (*D*)
Sao Paulo					
Positive	114	26	455	371 (*A*)	84 (*B*)
Negative	22	98	420	77 (*C*)	343 (*D*)

1. A–D are defined in Appendix 1.

TABLE II
Validation indices for the SRQ obtained in Calcutta and Sao Paulo (percentages)

Index	*Calcutta*	*Sao Paulo*
Sensitivity	79	83
Specificity	75	80
Positive predictive value	76	82
Negative predictive value	78	82
Misclassification rate	23	18

among the primary care clinic patients or community. The predictive values vary with the prevalence of psychiatric morbidity; the higher the prevalence the greater the positive predictive value (Meehl & Rosen, 1955; Galen & Gambino, 1975).

Table I gives the results found in Calcutta and Sao Paulo respectively, and Table II gives the validation indices derived on the principles described in Appendix 1. The optimum cut-off point of the SRQ in Calcutta was 11–12, while in Sao Paulo it was 7–8. The cut-off point was determined by calculating the sensitivities and specificities for a range of values of the case/non-case threshold; the chosen threshold was that which permitted the best trade-off between sensitivity and specificity, which are inversely related.

All indices of validity were somewhat higher in the Sao Paulo study, although the differences were not statistically significant.

Discussion

The studies described here are the first to use the SRQ in conjunction with the CIS. The objective of this paper is to illustrate a two-stage screening procedure which may be cost-effective, and which could be applied in large-scale primary care or community surveys with the emphasis on minor psychiatric morbidity in developing countries, where resources are always scarce.

These two investigations have concentrated on two aspects. Firstly, the feasibility of using health auxilliaries (e.g. social workers and health workers trained in primary health care) with limited training to screen effectively for psychiatric disorders was studied. Although health auxilliaries were used for first-stage psychiatric screening in the World Health Organisation (WHO) collaborative study (Harding *et al*, 1983a,b), their work has not yet been replicated. The Calcutta study also served as an initial assessment of the extent to which the SRQ could be successfully used as a screening tool by auxilliaries with limited education and minimal training in the administration of the questionnaire. Secondly, the success of the procedure in screening for the wide range of psychiatric disturbances which are commonly seen in primary care or in the community was examined. Adequate validation of the first-stage screening instrument and choice of the appropriate instrument for second-stage screening are crucial for both these purposes.

Choice and validation of the SRQ

The choice of the SRQ was influenced mainly by the fact that this instrument was developed by the WHO for use in primary care settings in developing countries (including India and Brazil), where it has previously been validated (Harding *et al*, 1983b). It was also determined on an earlier occasion (Sen, unpublished) in Calcutta that health auxilliaries with limited education found the SRQ, with its yes/no responses, less complicated to administer than the GHQ (Goldberg & Blackwell, 1970), with which they experienced problems in conveying to the respondents the concepts of "no more than usual" and "more than usual". This problem was experienced in Sao Paulo to the extent that respondents did not understand some of the questions on the GHQ-30 (Mari & Williams, 1984). This difficulty did not, however, recur with the GHQ-12 on a subsequent occasion (Mari & Williams, 1985), although it was observed that administration of the GHQ-12 took more time than the SRQ. In addition, the social worker administering the two questionnaires reported that the SRQ questions were more acceptable to the respondents than the GHQ-12.

Harding *et al* (1983a), in their WHO study in seven developing countries, stated that there is considerable variation in the optimal cut-off point (varying between 5–6 and 10–11 in different areas) which "results from substantial variation in response rates". The most likely explanation for this difference in the two studies described here (11–12 in Calcutta and 7–8 in Sao Paulo) may lie in the varying socio-cultural characteristics of the populations studied; e.g. in Sao Paulo, a lower cut-off point (5–6) in men gave the best results (Mari & Williams, 1985). There is also evidence that individuals with little or no education are more likely to be false positive on the SRQ (and the GHQ) than those who had more than eight years of schooling (Mari & Williams, 1986b). The fact that 47% of the respondents in Calcutta, but only 24% of the respondents in Sao Paulo, had less than one year of schooling tends to favour the use of a higher cut-off point for the SRQ in Calcutta.

There is some evidence that increasing psychiatric experience of the rater is associated with a tendency to rate less abnormality (Copeland *et al*, 1975); Wing *et al* (1977) also reached similar conclusions. This might mean that the less the interviewer's exposure to psychiatric disturbances, the more is his likelihood of rating a greater number of symptoms. There is some suggestion that similar factors operated in the Calcutta study, when compared with that in Sao Paulo. In Calcutta, the three health workers who administered the SRQ had had little mental health experience and only a brief orientation session in conducting the SRQ screening. In Sao Paulo, on the other hand, the SRQ was administered by a qualified social worker with some training in mental health and, in addition, a week's training in the administration of the SRQ. Although the SRQ simply requires yes/no answers, an element of judgement is involved in determining the presence or absence of a symptom with answers like: "Yes – once in a while – it doesn't upset me greatly" or "Yes, some time back".

The two studies discussed here support the recommendation (Harding *et al*, 1983a) that the SRQ should be adequately validated – for determination of the cut-off point – before being employed for large-scale primary care or community surveys.

Appropriateness of the CIS for second-stage screening

Dohrenwend & Dohrenwend (1982), in their global review of epidemiological literature in psychiatry, commented on "the central unsolved problem of case identification" among the studies published up to the 1970s. In the majority of these studies, unstandardised psychiatric interviews by psychiatrists remained the ultimate criterion to determine 'caseness', with the result that the reported prevalence rates varied so widely that it rendered any comparison between them virtually meaningless. This applies especially to the non-psychotic disorders, which constitute the overwhelming majority of psychiatric disturbances in primary care or in community samples.

Many studies, both in India and elsewhere, have applied the operational definition of a case adopted by the WHO Expert Committee on Mental Health (1960). But, as Wig (1982) points out, this definition concentrates solely on the severe types of mental illness, excluding 'minor' psychiatric disorders.

It is now generally agreed that the two-stage screening procedure, which utilises a validated screening technique followed by a standardised psychiatric interview, is an improvement on earlier studies in estimating the prevalence of psychiatric disorders in primary care or in the general population. The WHO Collaborative Study on Strategies for Extending Mental Health Care (Harding *et al*, 1983a) utilised the SRQ for first-stage screening and the Present State Examination (PSE) (Wing *et al*, 1974) for subsequent psychiatric interview.

However, the PSE was developed in a hospital setting and rates symptoms that must be "clinically fairly severe"; it only identifies those psychiatric disorders "familiar to psychiatrists in hospital practice" (Wing *et al.* 1981). There is also a suggestion that the PSE, when used as a standardised instrument for second-stage screening with the GHQ or SRQ for the first stage, produces low validation indices (Cleary *et al*, 1982; Harding *et al*, 1983b; Byrne, 1984).

Although the focus of psychiatric epidemiology has shifted from hospital studies to primary care and community surveys, case definition criteria have lagged behind, remaining rooted in concepts derived from hospitalised patients. As Williams *et al* (1980) point out, "it is as if, in a study of the community prevalence of diabetes, diabetes were defined by the presence of diabetic retinopathy".

In the two studies illustrated here, the CIS was considered to be the most appropriate instrument for psychiatric assessment for two reasons. It was developed so that it "should be acceptable to individuals who may not see themselves as psychiatrically disturbed", and "the content of the interview should be appropriate to the types of psychiatric disturbance encountered in the community" (Goldberg *et al*, 1970).

Cost-effectiveness of the procedure

For the purposes of this paper, cost-effectiveness is assumed to be concerned with the 'least cost' solution to a given objective, which in this instance is the estimation of psychiatric morbidity in primary health care and the community in developing countries. As Kapur (1975) has shown, to have the first-stage psychiatric screening conducted by health auxilliaries not only saves on the research psychiatrist's time, but utilises health personnel available in most developing countries at comparatively low cost, and thus contributes greatly to the cost-effectiveness of the project. This applies to the two studies described here. Moreover, in the Calcutta study the health auxilliaries were able to conduct the first-stage screening as part of their routine duties.

Assuming that two or more screening procedures are equally cost-effective at a given level of prevalence, a further reduction in the proportion of false positives and false negatives obtained by one or other of the procedures should result in an increase in the cost-effectiveness of that procedure. In both Calcutta and

Sao Paulo, around one-fifth of the screened individuals were estimated to be misclassified, which does not compare unfavourably with the proportions misclassified with the GHQ-30 (Vieweg & Hedlund, 1983) or the GHQ-12 (Mari & Williams, 1985). It is likely that a period of training of health auxilliaries might lead to a lowering of the misclassification rate, and thus make the test more cost-effective. For a technical discussion of misclassification as an indicator of cost-effectiveness, the reader is referred to Skuse & Williams (1984).

The procedure discussed here will form the basis of community surveys now being planned to be conducted in both Calcutta and Sao Paulo. Replication of this procedure in other developing countries may lead not only to the simplification of the screening questionnaire (e.g. by reducing the number of questions), but also to more information about minor psychiatric morbidity, which has been inadequately investigated in developing countries. It is hoped that this will lead to early intervention in their management, possibly by health auxilliaries trained in mental health care (Wig *et al*, 1981), and thus prove to be of value in preventing patients with primary psychological problems being subjected to unnecessary and often expensive medical investigation (Mitchell, 1985). There seems to be little doubt, however, that such research endeavours will, at the very least, serve to focus attention on the extent of minor psychiatric morbidity in the health centres – where, despite causing considerable distress, they go largely unrecognised.

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Appendix 1

A summary definition of the statistical norms used in the analysis of SRQ as a screening instrument is given here. The quantities *A* to *D* are defined in Table III.

TABLE III
Quantities involved in definition of statistical norms

SRQ finding	*Psychiatric assessment (using CIS)* *Positive*	*Negative*
Positive	*A*	*B* (false positive)
Negative	*C* (false negative)	*D*

$$\textit{Sensitivity}\ (\%) = \frac{\text{Number of cases correctly identified by SRQ}}{\text{Actual number of cases}}$$

$$= \frac{A}{A+C} \times 100$$

$$\textit{Specificity}\ (\%) = \frac{\text{Number of non-cases correctly identified by SRQ}}{\text{Actual number of non-cases}}$$

$$= \frac{D}{B+D} \times 100$$

Percentage of false positives = 100 – specificity

Percentage of false negatives = 100 – sensitivity

$$\textit{Misclassification rate} = \frac{\text{False positives} + \text{False negatives}}{\text{Grand total}}$$

$$= \frac{B+C}{A+B+C+D}$$

Positive predictive value (PPV) (probability of a probable case being an actual case)

$$= \frac{\text{Number of cases correctly identified by SRQ}}{\text{Number of probable cases}} = \frac{A}{A+B}$$

Negative predictive value (NPV)(probability of a probable non-case being an actual non-case)

$$= \frac{\text{Number of non-cases correctly identified by SRQ}}{\text{Number of probable non-cases}} = \frac{D}{C+D}$$

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Family-Genetic Studies and Identification of Valid Diagnostic Categories in Adult and Child Psychiatry

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Family-genetic studies of child and adult psychiatric disorders have become increasingly fashionable over the past decade. The development of structured diagnostic interview schedules, the emergence of uniform diagnostic criteria such as DSM–III, and the use of refined design and analytic techniques from the field of chronic disease epidemiology have made substantial contributions to the methodology of such studies. Advances in molecular genetics, particularly our emerging capacity to perform chromosomal linkage studies throughout the human genome, have renewed hope that the constitutional underpinnings of some psychiatric disorders can be identified and that the pathophysiology of these disorders can be elucidated. Family-genetic techniques in child and adult psychiatry are discussed with a particular focus on their potential value in validating diagnostic categories spanning developmental epochs.

A variety of 'family-genetic' strategies have been developed over the past century (Kidd & Matthysee, 1978). Classically, four types of evidence are suggestive of genetic or familial factors being aetiologically important in behavioural disorders: (a) an increased frequency of the disorder observed in at-risk family members; (b) an increased frequency of the disorder in the adopted-away biological offspring of affected individuals; (c) a higher concordance for the disorder among monozygotic than among dizygotic twins; and (d) evidence for a specific genetic factor transmitted in an area adjacent to a known chromosomal locus. Although this paper will focus primarily on the first and last of these, many of the comments regarding assessment methodologies apply equally to twin and adoption studies.

Recent methodological advances

Each of these family-genetic strategies requires the ability to assess reliably the psychiatric status of large numbers of individuals, many of whom have never been in treatment. Over the past decade, family-genetic studies in the USA have increasingly been influenced by putative advances in psychiatric nosology and by corresponding changes in assessment procedures. Historically, the search for reliable diagnostic criteria and the development of standardised methods to elicit the needed information can be traced to work at Washington University in St Louis. Feighner *et al* (1972) published criteria for 15 major psychiatric disorders and developed a structured interview instrument, the Renard Diagnostic Interview, to collect systematically information relevant to the application of these criteria (Robins *et al*, 1977). These criteria were subsequently modified and elaborated, and specific 'Research Diagnostic Criteria' (RDC) were proposed (Spitzer *et al*, 1978*a*; Endicott & Spitzer, 1979). The RDC focused on past and present episodes of illness and provided inclusion and exclusion criteria by which clinically homogeneous groups of patients may be diagnosed and studied. The Schedule for Affective Disorders and Schizophrenia (SADS), a structured interview, was developed at the same time to ensure that the information on signs and symptoms were collected systematically (Spitzer & Endicott, 1978). A 'lifetime' version of the SADS was also developed to record information concerning past as well as present episodes of illness for use in family studies. Use of the RDC and the SADS greatly improved inter-rater agreement (Endicott & Spitzer, 1979). DSM–III (American Psychiatric Association, 1980) is a direct descendant of these earlier diagnostic schemata.

Similar developments in the standardisation of diagnoses have taken place in the United Kingdom – particularly the work of Wing *et al* (1967, 1974) on the Present State Examination (PSE), which incorporates standard methods of defining, eliciting, and recording information about 140 symptoms and signs in an interview format. The PSE is reliable, and modified versions have been widely used in large-scale international studies (Wing *et al*, 1977). Only recently, however, have attempts been made to modify the PSE to include information on 'lifetime' symptoms (Torgersen, 1983).

The movement to define reliable categories has also led in the USA to a proliferation of diagnostic categories in both the adult and childhood areas of DSM–III. Whether this is a real advance or merely legerdemain is debatable (Rutter & Shaffer, 1980; Tyrer, 1985); our ability to construct reliable diagnostic categories may have outstripped our ability to validate such categories. The hierarchical organisation of diagnoses in DSM–III has also proved to be problematic. Although the exclusion of some diagnoses, such as anxiety disorders, during episodes of depression or schizophrenia is parsimonious, it may be misleading when the validity of the hierarchical scheme is unproven (Leckman *et al*, 1983*a*). The revision of DSM–III currently under way largely does away with these exclusionary criteria.

Validation of diagnostic categories and distinctions

How can we 'validate' a diagnostic category? In the absence of specific neuropathological evidence, how can we establish that any two individuals are suffering or have suffered from the same disorder? This fundamental question has yet to be answered for most, if not all, psychiatric disorders. The identification of clinically homogenous groups of patients is insufficient (Rutter & Shaffer, 1980). DSM–III offers many examples of such *a priori* groupings. While they may be acceptable first approximations, they incautiously assume that cross-sectional clinical homogeneity accurately reflects an underlying aetiological homogeneity. Indeed, some medical disorders with similar pathophysiological and clinical expressions, have been found to be aetiologically heterogenous, e.g. anemias, coagulation disorders, and congenital deafness. Historically, most psychiatric researchers have emphasised the use and importance of natural history data regarding the onset, course, and outcome to establish valid diagnostic categories. Differential response to treatment and the presence of a 'biological-marker' have more recently been proposed as alternate means to validate categories of psychiatric disease. Although a thorough review of these strategies is beyond the scope of this paper, it is clear that none of them is wholly adequate. Each is vulnerable to the presence of factors which are unrelated to the underlying disease process but which may dramatically influence its onset, course, or treatment outcome.

The use of family history and family study data to help define nosological categories more precisely is well-known (Robins & Guze, 1970; Spitzer *et al*, 1978*b*). For example, more than a score of family studies of schizophrenic probands have found that the risk of schizophrenia and related conditions significantly exceeds the risk observed in the general population, thus confirming Kraepelin's (1904) original view that familial factors are aetiologically important in this disorder. Although the familial nature of schizophrenia has recently been challenged, the most rigorous family studies of schizophrenia using DSM–III criteria and structured interviews continue to provide strong support for the view that schizophrenia is a familial disorder (Tsuang *et al*, 1980; Kendler *et al*, 1985). The Iowa studies have also helped to clarify the possible connections between schizophrenia and a variety of other disorders. While the relationship between schizophrenia, psychotic major depression, and schizoaffective disorders remain obscure, the evidence is persuasive that alcoholism, unipolar depression, non-psychotic depression, major depression, and some anxiety disorders are aetiologically unrelated to schizophrenia (Tsuang *et al*, 1980; Kendler *et al*, 1985).

In providing an independent source of data, family-genetic studies also allow investigators to assess directly the importance of various clinical features of a putative disorder. By stratifying groups of at-risk relatives on the basis of the presence or absence of a particular symptom or pattern of symptoms in the index cases, it has been possible to assess the predictive value of such symptoms or symptom constellations by their effect on the rates of illness and the types of illness among the relatives. Recent examples of such work have focused on the importance of primary social role impairment (Gershon *et al*, 1986), age of onset (Weissman *et al*, 1984*d*), sex of proband (Merikangas *et al*, 1985*a*), and 'endogenous' symptom patterns in discriminating familial from non-familial forms of major affective disorder (Leckman *et al*, 1984*a*,*b*; Andreasen *et al*, 1986).

Family study data have provided valuable information by tentatively identifying alternate manifestations of the same underlying diathesis. The relationship of obsessive-compulsive disorder to Gilles de la Tourette's syndrome (TS) is one such example, where high rates of obsessive-compulsive disorder were found among the first-degree relatives of probands with TS (Pauls *et al*, 1986*b*; Pauls & Leckman, 1986). Better known is the apparent association of schizophrenia with a 'spectrum' of related disorders (Kety *et al*, 1968; Kendler & Gruenberg, 1984).

Restratification techniques have also proved useful in studying the relationship of co-morbid conditions, such as panic disorder and alcoholism, with major affective disorder (Leckman *et al*, 1983*b*; Merikangas *et al*, 1985*b*). In the case of panic disorder, we have investigated the relationship between symptoms of depression and recurrent panic attacks as part of a large scale family-genetic study of major affective disorders (Weissman *et al*, 1984*a*,*b*). As noted above, the prevailing diagnostic conventions in the United States assert that prominent anxiety symptoms (including recurrent panic attacks) that occur solely during depressive episodes do not have any nosological significance. Our family study data, on the contrary, have indicated that the association of depression and recurrent panic attacks in index cases was associated with increased rates of depression and anxiety in the adult at-risk relatives, compared with the adult at-risk relatives of depressed probands without prominent anxiety symptoms (Leckman *et al*, 1983*a*,*b*). This increased risk was observed regardless of whether or not the recurrent panic attacks occurred concurrently with the depressive episodes. Similar findings were also observed in the offspring under the age of 18 years in these families (Weissman *et al*, 1984*c*).

These data call into question the validity of the prevailing diagnostic convention and suggest that additional studies are needed to clarify the aetiological relationship between recurrent panic attacks and depression; they have also led to the hypothesis that some forms of depression and some forms of 'panic disorder' partially or wholly share a common underlying diathesis. If this can be established and the critical clinical features identified, more precise studies in other areas of clinical investigation will follow. Specific differences in natural history, treatment response, and biological measures may be expected. Even our capacity to apply sophisticated mathematical models and to perform state-of-the-art genetic linkage studies in high-density families using newer recombinant DNA techniques will be enhanced through our ability to identify aetiologically more homogenous patient groups.

Another example of assessing the relationship of a co-morbid condition involves an effort to sort out the nosological significance of frequent symptoms of hyperactivity, attentional impairment, and impulsivity in children with TS. In a family study of TS we found that the at-risk relatives of probands with TS and attentional impairment had an increased rate of attentional impairment, but no increase in the rate of TS or chronic multiple tics compared with the at-risk relatives of probands with TS only (Pauls *et al*, 1986*a*). These data, together with the observation that the attentional impairment and the tics or TS did not co-segregate within these families, have led us to conclude that the TS diathesis is largely independent of the aetiological factors which underlie the attentional problems observed in a sizeable proportion of TS patients (over 50% in most clinic samples). The increased frequency of attentional problems among TS patients, which has been observed in clinic patients, may be due to a referral bias (Berkson's bias). Children with two disorders may be more likely to come to the attention of health care professionals, particularly if both adversely affect school performance and behaviour.

Limitations

The use of family study data to establish valid diagnostic criteria is largely limited to those disorders in which transmission within families is known to occur. As such, efforts to refine and validate criteria logically follow studies in which familial or genetic factors have been demonstrated to be aetiologically important. Even with this constraint, the list of disorders for which such a determination has been made is substantial and includes schizophrenic disorders, affective psychoses, paranoid states, some forms of alcoholism, panic disorder, agoraphobia, and anti-social personality disorder in adulthood; in children it includes infantile autism, attention deficit disorder, tic disorders, stuttering, and some learning disorders. In other instances, suitable studies have not yet been performed on which to judge the aetiological importance of familial or genetic factors. Conversely, hypotheses invoking environmental factors transmitted within families cannot be rejected for most psychiatric disorders. However, the potential importance of genetic factors in creating unique environments should not be discounted (Scarr & McCarthy, 1983).

On a more practical level, the need to establish 'lifetime' diagnostic estimates is associated with important methodological problems involved with the recall of symptoms for remote episodes of illness. These problems are compounded when the individual is dead or otherwise unavailable for direct interview. In such cases the diagnostic estimates must be made on the basis of indirect reports from other relatives or from medical records. The under-reporting of psychiatric disorders associated with indirect 'family history' methods has been well documented (Andreasen *et al*, 1977). Not surprisingly, individuals living in the closest proximity, such as spouses, are generally the best informants (Thompson *et al*, 1982). Nor is it surprising that severe disorders can be more consistently identified by family members (Orvaschel *et al*, 1982). These methodological difficulties culminate in making final 'best estimate' diagnoses on the basis of all available information. Inevitably, the quantity and quality of data available for this procedure are often quite variable. However, good inter-rater agreement using the best estimate method was found for major depression, depressive personality, alcoholism, drug abuse, panic disorder, phobia, and antisocial personality among interviewed individuals (Leckman *et al*, 1982).

The diagnostic procedures become even more problematic in dealing with childhood disorders. Although structured and semi-structured instruments have been developed for school-aged children (Herjanic *et al*, 1975; Puig-Antich & Chambers, 1978), the test-retest reliability of these instruments is marginal for many diagnoses (Chambers *et al*, 1985). Similarly, the level of parent-child agreement is often low. A related problem has to do with attempts to make childhood diagnoses, such as separation anxiety, retrospectively, in adult subjects who no longer display the symptoms. Many of these difficulties are substantive and may not yield to refinements in assessment methodology. Examination of these issues raises fundamental questions concerning the developmental continuities or discontinuities of psychopathological states. They also highlight the limitations in perception and recognition over time by individual children, parents, and investigators of mental disorders as opposed to states that are perceived as being within the range of 'normal' experience.

Another major limitation involves the potential effects of assortative mating – the tendency for couples with similar phenotypic traits to select one another more frequently than would be expected by chance alone. Assortative mating for psychiatric disorders in general, as well as for specific psychiatric disorders, has been consistently reported (Merikangas, 1982). This non-random mating can influence the distribution of disorders in a population, by increasing the population variance and the proportion of homozygous individuals, and so mislead the unwary investigator. The Danish Adoption Study and the American-Danish High Risk Project provide examples of the potentially confounding effects of this phenomenon in studies of schizophrenia (Rosenthal, 1975; Parnas, 1985).

Improvements in the statistical methods for handling these large data sets from family-genetic studies have included: the development of methods for quantifying lifetime prevalence which handle the age correction of the data without ancillary information on population age-specific incidence rates and are independent of mortality (Thompson & Weissman, 1981); the successful application of log-linear models to deal with potentially confounding variables which may be related to outcome (Kidd *et al*, 1981; Weissman *et al*, 1982); and the use of survival time models with non-proportional hazard functions (Weissman *et al*, 1984*d*; Wickramaratne *et al*, 1986).

Prospects for the future: genetic linkage studies

Genetic linkage has long been recognised as a way of demonstrating that a specific genetic factor is aetiologically important in the transmission of a particular disorder. If linkage can be demonstrated it may be possible, given the current revolution in human genetics, to isolate a specific genetic factor and gain insight into the basic constitutional events that may predispose an individual to the expression of a particular psychiatric disorder (Cloninger *et al*, 1983; Kidd, 1982, 1985). Equally exciting is the possibility that by ascertaining which individuals are at risk it will be possible through the use of appropriate longitudinal research designs to identify those risk and protective environmental factors which mediate the expression of the disorder. Important and rational strategies for treatment and possible prevention would also be anticipated.

Until a few years ago this method had limited applicability to human disorders, because of the small number of polymorphic genetic markers that were available. Over the past few years the field of human molecular genetics has changed dramatically. Using recombinant DNA techniques, investigators are identifying a growing number of highly polymorphic markers. As of mid-1985 over 800 new polymorphisms had been identified (Willard *et al*, 1985), and that number has significantly increased during the last two years. Since new polymorphisms are being discovered at an ever-increasing rate, it should be possible to construct a genetic linkage map of the entire human genome in the next few years. This development may have major consequences in the field of psychiatry. For example, the recent reports of Gerhard *et al* (1984) and Egeland *et al* (in press) detailing the possibility of linkage of manic-depressive disorder in large Amish pedigrees to markers on chromosome 11, if confirmed, may clarify the relationship of unipolar to bipolar affective disorders in at least some cases, as well as herald other dramatic advances in our understanding of this disorder.

Although the number of psychiatric disorders which may be amenable to this technique (i.e. for which a single major locus accounts for a sizeable portion of the total phenotypic variance) is unknown and may well be small, the potential power of these recombinant DNA techniques argues against any premature judgement that complex behavioural traits with non-Mendelian patterns of inheritance are necessarily polygenic in origin.

Finally, genetic linkage strategies may provide an elegant means of 'validating' some diagnostic categories in psychiatry. The outcome of such a validation might be the delineation of 'families' of related disorders that have the same underlying vulnerability but differing phenotypic expression. Tracing the range of phenotypic expression for specific disorders from childhood on will be a particularly important undertaking for investigators interested in the possible continuities between adult and child psychopathology. Pathognomonic symptoms may be identified that may have relatively little clinical significance but which are highly associated with the underlying vulnerability. It will also permit the identification of phenotypically similar but aetiologically distinct disorders which can confound empirical psychiatric research.

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The Newcastle Adolescent Behaviour Screening Questionnaire

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As part of a larger study, a brief rating-scale was developed which focusses on the mid-adolescent phase of development. Completed by teachers, the questionnaire has an inter-rater reliability of 0.78, with a test–retest correlation of 0.82. When the performances of various screening instruments were compared it became clear that no single questionnaire was obviously more efficient than the others at detecting potential disturbance in an urban adolescent population. Indeed, different questionnaires seemed to highlight particular facets of functioning. The Newcastle Adolescent Questionnaire proved to be a reliable and valid screening measure.

Several teacher questionnaires measure facets of the behaviour of children and adolescents. Some of these questionnaires are standardised for specific stages of development, e.g. the Devereux Scale (Spivack *et al*, 1967), while others are used across the whole school age-range with only minor attempts to standardise for age (Rutter, 1967; Conners, 1969). The Rutter B2 Scale consists of 26 items and gives rise to two sub-scales, neurotic and antisocial behaviour, while the Conners Scale consists of 39 items and generates four sub-scales: inattentive–passive; passivity; anxiety; and hyperactivity. The Behaviour Problem Checklist (Quay & Peterson, 1979) consists of 55 items and also generates four factors. Although this instrument has been well validated, especially in the younger age-group, the factors show a bias toward detecting conduct and personality problems, so that the checklist may miss a portion of the distressed adolescents in the community (Place & Kolvin, in preparation). One of the few scales which is specific for adolescence is the Adolescent Life Assessment Checklist (Gleser *et al*, 1977), but this 40-item scale has only been validated for selected samples (Boyle & Jones, 1985) and so its value as a community screening instrument is unclear.

As part of a larger study, it was decided to develop a questionnaire which would be very brief and would focus on the mid-adolescent phase of development. Using clinical experience and published reports, we considered a large number of behavioural features for inclusion. Seven were eventually chosen because they often give rise to concern in educational settings: they include a sense of confusion, excessive day-dreaming, lability of mood and emotion, lack of confidence, and being easily upset in the face of educational failure (see Figure 1). In addition it was decided to include three more global ratings. The first reflects more widespread neurotic behaviour, the second antisocial behaviour. As a final item we decided to include educational difficulties which were serious enough to warrant special attention. We suspected that a positive rating on this item would prove a useful indicator not only of general educational difficulties but also of personal and interpersonal problems.

The aim of the current paper is to explore the use of this brief questionnaire in an adolescent sample drawn from the community. Our primary hypothesis was that adolescents detected as being potentially disturbed using a 'multiple-criterion screening' technique (described below in the 'Method' section) would prove to have significantly high scores on this brief questionnaire. This would in effect provide a form of validation of the questionnaire. A second aim was to study its sensitivity and specificity compared with a psychiatric interview. Finally, it was planned to compare the scores obtained on this instrument with the scores obtained on longer screening questionnaires by the same population.

Method

The 4th and 5th-year pupils of four large comprehensive schools agreed to participate in the study: 1446 adolescents formed the population for this phase of the study, and they were screened using a 'multiple-criterion screen' method. This technique uses the number of extreme scores that an individual obtains on a variety of instruments as a measure of potential disturbance. The detailed methodology may be found elsewhere (Place *et al*, 1985), but in brief the screen consisted of:

(a) Rutter B2 Scale (Rutter, 1967, 1973)
(b) a sociometric assessment technique (MacMillan *et al*, 1978)

Newcastle Adolescent Behaviour Questionnaire

Name of Child .... School..........

Date of Birth .... Form ....

Below are a series of questions describing behaviour often seen in adolescents. If the description applies to this youth please mark the third column. If it does not apply please mark in the first column. If it applies to a lesser degree please put a cross in the box in column two.
Please consider only recent behaviour, i.e. in the last 6 months.

	No	Somewhat	Yes	For Official Use Only
1. Excessive day dreaming	☐	☐	☐	☐
2. Often looks puzzled or confused	☐	☐	☐	☐
3. Over-emotional when checked or frustrated	☐	☐	☐	☐
4. Lacks confidence in educational situations	☐	☐	☐	☐
5. Tends to vary in mood	☐	☐	☐	☐
6. Easily upset when cannot master educational tasks	☐	☐	☐	☐
7. Reserved and uncommunicative	☐	☐	☐	☐

It would be most helpful if you could make an overall assessment as to whether this youth:

	No	Somewhat	Yes	For Official Use Only
is nervous, i.e. emotional, tense, worried and anxious, to the extent of impeding him in his school work or relationships	☐	☐	☐	☐
shows problem behaviour, i.e. difficult and disruptive behaviour, disobedient, etc.	☐	☐	☐	☐
has educational difficulties which merit special attention	☐	☐	☐	☐

(c) Junior Eysenck Personality Inventory (Eysenck, 1965)
(d) General Health Questionnaire (Goldberg, 1972)
(e) Rutter Malaise Inventory (Rutter *et al*, 1970).

(a) Rutter B2 Scale This is a teacher-report instrument which has been used in many settings, both in this country (Ryle & MacDonald, 1977; Rutter, 1973; Wolkind, 1974; MacMillan *et al*, 1980) and abroad (Minde, 1975; Zimmermann-Tansella *et al*, 1978). The instrument was designed for the 6–13 age-group (Rutter, 1967), for whom it has an inter-rater reliability of 0.72 and test–retest reliability of 0.89. Little validation work has been carried out on the instrument in the adolescent age-group.

(b) Sociometry This technique uses the responses of peers to gauge an individual's social standing. It allows identification of those youths who are likely to be avoided by classmates (rejection) and those who would not be chosen as friends (isolation). It has been shown to be a rapid way of gathering useful information from younger children (MacMillan *et al*, 1978) with a test–retest reliability of 0.87 for rejection and 0.72 for isolation (tests carried out 4–5 weeks apart), but its effectiveness with adolescents seems less clear (Place & Kolvin, in preparation).

(c) Self-Report The Junior Eysenck Personality Inventory (Eysenck, 1965) was used as the main item of self-report, although previous work with 11-year-olds has shown that it tends to select a rather unique population compared with other measures (MacMillan *et al*, 1980). The neuroticism scale is reported to have a split-half reliability of 0.84 and a test–retest reliability of 0.77 (Eysenck, 1965).

In addition, 605 youths completed the 30-item General Health Questionnaire (Goldberg, 1972) and 1237 completed the Rutter Malaise Inventory, which has a test–retest reliability of 0.91 (Rutter *et al*, 1970). In an effort to reduce the workload on teachers and also to minimise the disruption of classes for the pupils, all were tested, but not everyone with all the tests. This also ensured that the exercise remained within the resources of the research team.

The next step was to compare the results obtained from the questionnaire with the data obtained from a psychiatric interview. For these purposes a sub-sample of 82 youths

TABLE I

*Correlation of items in the Newcastle Adolescent Behaviour Questionnaire. All correlations are significant at the 0.001 level, except for * ($P < 0.05$)*

	A	B	C	D	E	F	G	H	I	J	K
A. Educational difficulties	1.0										
B. Disruptive and disobedient	0.16	1.0									
C. Tense, worried and anxious	0.43	0.14	1.0								
D. Reserved and uncommunicative	0.25	0.06*	0.39	1.0							
E. Easily upset	0.29	0.37	0.42	0.2	1.0						
F. Variable mood	0.17	0.58	0.27	0.11	0.53	1.0					
G. Lacks confidence	0.44	0.18	0.44	0.43	0.47	0.32	1.0				
H. Overemotional	0.22	0.58	0.28	0.08	0.58	0.68	0.29	1.0			
I. Puzzled	0.42	0.13	0.38	0.38	0.38	0.26	0.66	0.25	1.0		
J. Day dreams	0.36	0.19	0.39	0.43	0.29	0.28	0.52	0.25	0.61	1.0	
K. Total Score	0.56	0.51	0.63	0.54	0.69	0.65	0.76	0.64	0.72	0.69	1.0

was selected on a random basis from the youths who had an extreme score on at least one of the four screening instruments. The percentage levels used for cut-off were varied to ensure the inclusion of at least 20 individuals on each major criterion, but because of overlap the number selected on each was approximately 30. In addition, 50 adolescents were selected at random from the youths who had no extreme scores on any of the measures. All of these 132 youths were given a structured psychiatric interview, the interviewer being unaware of the questionnaire results. Psychiatric disturbance was rated as 'absent', 'dubious', 'moderate' or 'marked' and these ratings were then further combined to produce two groups—'no significant disturbance' (absent/dubious) and 'significant disturbance' (moderate/marked).

For comparison, 30 adolescents who were resident in a medium-stay psychiatric unit were assessed with the instruments mentioned above, with the exception of the sociometric technique. Additional data were obtained from the teachers of this in-patient group to allow a study of the Newcastle Questionnaire's inter-rater and test–retest reliabilities to be undertaken: two of the teachers were asked to rate the youths' behaviour at the time of the survey and again one month later.

Results

It is important to establish the reliability and validity of any new behavioural measure, and this may be done in various ways.

Reliability

(a) Inter-rater reliability

It is necessary to confirm that different teachers will give similar ratings for similar behaviour. Greater agreement occurs when the instructions on the use of the instrument are carefully presented in a standard form, and it was for this reason that we chose to follow the presentation used by Rutter in his teacher questionnaire (Rutter, 1967). To test reliability we used the 30 adolescent patients in a medium-stay psychiatric unit: community surveys yield small numbers of potentially disturbed youths and so the correlations found may be influenced by a lack of spread in the data. The questionnaire's inter-rater reliability was found to be 0.78 ($n = 30$), which is satisfactory for this kind of brief instrument.

(b) Test–Retest reliability

This was established using the same 30 in-patients as for inter-rater reliability: they were reassessed after one month. Such exercises, which are essential to test the reliability of an instrument, are often bedevilled by the variation of behaviour which occurs over time. Nevertheless, the correlation value obtained (0.82) is an acceptable one for this type of screening instrument.

(c) Split-half reliability

After pairing items, one member of the pair was assigned randomly to either the first or second half of the questionnaire. A Pearson correlation of 0.84 was obtained between these halves, and applying the Spearman Brown Prophecy formula the split-half reliability was calculated at 0.91. This is a highly satisfactory level of reliability for a scale of 10 items, particularly as one of the items is not intended to measure behaviour directly.

To confirm this finding, the split-half reliability exercise was repeated without the more global assessments being included. This yielded a Pearson correlation coefficient of 0.85, which is almost identical to the value obtained for the full scale.

In addition, the means and standard deviations of the split-halves were calculated, and proved to be almost identical, indicating that they are equivalent measures of behaviour.

TABLE II

Validation: mean scores on the Newcastle Adolescent Behaviour Questionnaire for three different groups selected using other previously validated screening instruments

	A *Controls (screen –)*	B *Screen +*	C *Psychiatric in-patients*
n	1144	302	30
Mean	1.99	3.98	6.87
s.d.	2.73	3.94	3.35
Significance (*t*-test)	A *vs* B $t = 10.2$ $P < 0.0001$	B *vs* C $t = 3.88$ $P < 0.001$	
		A *vs* C $t = 7.91$ $P < 0.0001$	

Validity

(a) Content validity

Content validity is usually taken to mean that the items reflect the type of behaviour that the instrument is intended to measure. We believe the items chosen for this instrument have face validity. However, their relationship to each other and to the total score was explored. As Table I shows, although some of the correlations are reasonably high and some are low, all the correlations are positive. The average inter-correlation of the items was 0.34, and this suggests that the items are measuring similar aspects of behaviour. Cronbach has developed a formula for assessing the internal consistency of a questionnaire (Bynner, 1969), which provides a coefficient based on the average correlation between items and the number of items used. For an instrument to have valid internal consistency the Cronbach coefficient should be more than 0.5. The present scale gives a coefficient of 0.69, which is satisfactory.

(b) Concurrent validity

This compares the scores obtained on the scale with an external criterion, either information obtained from a clinical interview or scores on other instruments which are known to be valid. We explored our instrument's ability to distinguish between three groups of youths:

A. Controls Youths who did not emerge as high scorers on a number of previously validated instruments of child disturbance, i.e. youths who were 'screen negative'.

B. Screen positives Youths with a number of extreme scores on previously validated instruments designed to detect disturbed behaviour.

C. Psychiatric cases Adolescents whose difficulties were so severe as to not permit them to survive in the community and so warranted admission to a psychiatric unit on an in-patient basis. These youths could be expected to be more disturbed than the screen-positive adolescents who were surviving in the community.

Table II demonstrates a highly significant step-by-step increase in mean score on the questionnaire as we move from group A (the least disturbed) to group C (the most disturbed).

Table III gives teachers' ratings on the individual questions for each of these three groups, and confirms that all the questions distinguished between the controls and the potentially disturbed subjects. Such a finding adds further to the conclusion that the Newcastle Adolescent Behaviour Questionnaire is a valid instrument for an urban adolescent population.

(c) Sensitivity, specificity and efficiency

Cut-off scores of 3, 4, 5 and 6 on the Newcastle Questionnaire were explored in relation to the findings on the psychiatric assessment, and it was established that a score of 4 or more proved to be optimal for identifying potential cases in this particular population. Such an exploration tests the validity of the questionnaire at each particular cut-off score: it checks the proportion of 'correct' cases the instrument detects against the proportion it misses (*sensitivity*); the proportion of true negatives it correctly identifies (*specificity*); and the proportion of correct selection in the sample as a whole (*efficiency*).

The method of selecting the interview sample did not permit direct inferences about the total study population. The number of disturbed adolescents in the total sample was estimated from the proportion of disturbed youths found at each score in the interview sample. This estimate was then used to explore the performance of the questionnaire when the optimum cut-off was used (Table IV). Our method of calculating sensitivity, specificity and efficiency is detailed elsewhere (Place *et al*, 1985). The results reveal that the instrument is capable of selecting potential cases of adolescent disturbance with a moderate degree of accuracy, but is far less efficient than the Behaviour Screening Instrument developed by Richman & Graham (1971) for pre-schoolers, particularly in terms of specificity, and less efficient than scales developed for adult populations (Goldberg, 1972).

Comparison with other measures

In order to establish the comparative efficiency of this measure compared with other available scales which might be considered appropriate for screening adolescents, all the instruments were compared using their estimated performance in the study population when the optimum cut-off was used: the sensitivity, specificity and misclassification rate were established for each instrument. This is a rather crude analysis, but it showed that no instrument was clearly superior to the others. The sociometry scales showed a low sensitivity (isolation 30%, rejection 19%); highest were the Malaise Inventory (61%) and General Health Question-

TABLE III
Validation: percentage of high scores on each question of the Newcastle Adolescent Behaviour for each of three different groups (selected with previously validated screening instruments)

	A *Screen*	*B* *Screen+*	*C* *Psychiatric in-patients*	*Significance*		
	(n=1144)	*(n=302)*	*(n=30)*	*$P<0.05$*	*$P<0.01$*	*$P<0.001$*
Day dreams	2.9	11.3	8.9			AvB
Puzzled and confused	4.7	12.6	15.6	AvC		AvB
Over-emotional	0.9	10.6	11.1			AvC AvB
Lacks confidence	4.9	20.2	17.8	AvC		AvB
Variable mood	2.3	19.5	11.1	AvC		AvB
Easily upset	1.2	9.6	13.3			AvC AvB
Reserved and uncommunicative	4.5	12.3	15.6		AvC	AvB
Tense, worried and anxious	0.9	9.3	8.9			AvC AvB
Disruptive and disobedient	0.9	15.6	6.7			AvC AvB
Educational difficulties	0.09	7.0	6.7			AvC AvB

TABLE IV
Efficiency of the Newcastle Adolescent Behaviour Questionnaire in identifying cases in an adolescent population, compared with the estimated prevalence of disturbance. For these calculations the optimum cut-off score was used (4 or above)

	n	*Sensitivity*	*Specificity*	*Misclassification*	*κ*
Total population	1446	51%	82%	26%	0.32
Boys	745	57%	83%	23%	0.38
Girls	701	46%	80%	30%	0.26

naire (57%). The Newcastle scale fell in-between, with 51%. Although the Rutter B2 Scale was not very sensitive (32%), it had a lower misclassification rate (22%) than the Newcastle Scale (26%). The performance of the Rutter B2 Scale in this population shows that some of the items were not as sensitive at this age-level as they had been in the younger age-group.

The Newcastle Questionnaire scores were correlated with the scores obtained on the other measures, and for comparison the exercise was repeated for the Rutter B2 Scale (Table V). The significant correlations are with the peer-report measures and they are low; but they are of the same order as those obtained by the Rutter B2 Scale. The inter-correlation of the Newcastle Adolescent Questionnaire and the Rutter B2 Scale gives a reasonably high coefficient (0.69) which suggests that the two scales have much in common. There is a moderate but statistically significant correlation between the Newcastle Questionnaire and the multiple-criterion screen score. It is lower than the correlation between the multiple-criterion screen score and the B2 Scale, but the B2 Scale contributes to the screening procedure and so a high correlation is to be expected.

Comparing the Newcastle and Rutter B2 Scales (both teacher-report instruments) with questionnaires which explore other facets of functioning, we can clearly see that correlation between some of the scales is poor. It has been pointed out elsewhere (MacMillan *et al*, 1980) that the Junior Eysenck PI neuroticism scale has little in common with other screening measures for 11-year-old subjects, and it is perhaps not surprising to find a similar result in adolescence. However, all the self-report inventories correlate well with each other, and they show equal validity when compared with a psychiatrist's assessment (Place & Kolvin, in preparation). If we assume that these inventories are tapping internalised distress, there seems little doubt that the potential cases that they select are likely to be distinct from those who have shown behaviour problems at school. This distinction between internalised distress and externalised problems of conduct is not new (Rutter *et al*, 1970), but cannot be tapped in adolescence unless specific questionnaires are used. If this is not done, any survey tends to reflect objective behaviour problems and show minimal internal climate problems. With such little overlap between scales which measure such functions, the importance of using both sources of information is reaffirmed.

The Newcastle scale appears to be more sensitive for boys

TABLE V
Correlations of Newcastle Adolescent Behaviour Questionnaire and Rutter B2 Scale with other measures of adolescent behaviour

	Newcastle	*Rutter B2*
Multiple-criterion screen	0.33*	0.55*
Sociometry[1]		
Isolation	0.19*	0.21*
Rejection	0.19*	0.20*
Rutter Malaise Inventory[2]	0.03	0.04
General Health Questionnaire[2]	0.02	0.03
Junior Eysenck Personality Inventory[2]		
Neuroticism	0.03	0.03
Extroversion	0.01	0.02
Rutter B2 Scale[3]	0.69*	—

1. Peer report
2. Self-report
3. Teacher-assessed

*$P < 0.0001$ ($n = 1446$)

than for girls, a finding which is supported by the higher kappa score (Table IV). When considered with the above findings this suggests the hypothesis that behaviour scales may be more accurate at detecting disturbed boys while self-report measures may be more accurate at detecting disturbed girls. Such a theory is discussed in more detail elsewhere (Place & Kolvin, in preparation).

Discussion

New rating scales seem to appear frequently, and disappear almost as quickly. Snaith (1981) has urged that existing measures be refined and improved rather than perpetuating the development of more and more new instruments. This is a most appropriate aspiration because the response to a rating scale must be considered in the light of various factors.

'Overall Agreement Set' (Cronbach, 1942) identifies respondents' desire to give the answer that is expected or at least which appears less critical of themselves. This may be a particular problem in school-based research which uses the teachers as a source of information, when the results may be used to assess a school's ability to educate and control (Rutter *et al*, 1979; Rutter 1980). Despite this limitation many surveys have depended upon school-based research (e.g. Rutter *et al*, 1970; Offer, 1969; Coleman, 1974; Newman 1979) because, as Lewine *et al* (1978) have pointed out, "teachers are capable of making sophisticated and valuable observations regarding the psychological adjustment of their students". In addition, this type of research is popular with epidemiologists because the number of 'missing cases' is lower than with other methods of identifying a population: this advantage may be reduced in an adolescent population, however, because absentee rates tend to be high in this age-group (Fogelman *et al*, 1980).

In screening populations it is usual to seek a brief measure which is reliable and valid and of low cost (Boyle & Jones, 1985). Such a measure is already available for pre-school children (BSQ: Richman & Graham, 1971). The Rutter B2 Scale is a well-established instrument for use with school children but is not specific for adolescents. The Newcastle Adolescent Behaviour Questionnaire has been developed in an attempt to fill this gap, but its advantages over the Rutter B2 Scale, other than brevity, have yet to be established. However, its very brevity makes it suitable for investigating large populations.

One area of concern is the high misclassification rate which is obtained when this instrument's scores are compared with the findings at psychiatric interview. However, the other instruments tested show a similar range of misclassification values. Although some authors report minimal misclassification rates (Goldberg, 1972), others have obtained rates similar to those reported here when studying urban community populations (Tarnopolsky *et al*, 1979).

The lack of correlation between the self-report measures and the teacher-assessed scales could reflect a lack of validity in the new scale or could indicate that the self-rating scales are measuring different components of behaviour. Since the lack of correlation is similar for the well-validated Rutter B2 Scale, and with evidence of the validity of the self-report measures being available, it seems likely that the different types of instrument are detecting different components of behaviour.

Goldberg (1972) has pointed out that when screening a population research workers assume that psychiatric disturbance is evenly distributed throughout the population in varying degrees of severity. An individual's score on a questionnaire may then be seen as a quantitative estimate of that individual's degree of potential disturbance. These assumptions allow the researcher to decide upon a limit score, or cut-off, such that subjects with scores above it have a high probability of showing disturbance. However, like most screening instruments, such a questionnaire is not intended to make diagnostic distinctions.

Our results indicate that no single screening instrument is superior for detecting potential disturbance in adolescence. Indeed, different questionnaires seem to highlight particular facets of functioning, which are not necessarily detected by the others. Behavioural measures must sample both internal and external qualities if they are to fully explore disturbance in an adolescent population. The Newcastle Adolescent Questionnaire, although very brief, has proved to be almost as reliable and as valid as most of the other screening instruments available for this age-group.

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Affective Illness After Stroke

S. EBRAHIM, D. BARER and F. NOURI

Mood assessments were made after six months of 149 survivors taken from a register of all patients admitted to hospital with acute stroke. Using a General Health Questionnaire score of 12 or more as a criterion of important affective illness, its prevalence was 23%. There was no difference in risk of affective illness between left and right hemisphere strokes. Affective illness was strongly associated with functional ability, with limb weakness and with longer hospital stay in those with good functional recovery. Only 15% of those with high scores were receiving antidepressant drugs. The general practitioner is in the best position to detect psychiatric illness in stroke survivors; the use of mood rating scales such as the GHQ, in conjunction with clinical assessment, may improve detection.

Although depression is well recognised as a barrier to recovery after stroke (Adams & Hurwitz, 1963) and occurs commonly in selected groups (Robinson & Price, 1982; Robinson *et al*, 1984*a*), it is frequently missed in practice (Fiebel *et al*, 1979). Robinson & Price (1982) assessed mood at various stages after a stroke in selected groups of referred patients and in a small prospective study (Robinson *et al*, 1983). In the latter study the majority of the patients were relatively young, working class and black. No representative series reflecting the age and ethnic structure of the contemporary British population has been studied.

We chose to use the General Health Questionnaire (GHQ) to assess mood as it is easy to use, has been well validated (Goldberg & Hillier, 1979), has previously been used with stroke patients, and compares well with other mood assessments (Robinson & Price, 1982). We aimed to measure the prevalence of affective illness six months after an acute stroke in patients admitted to hospital, observing possible associated factors such as the site and severity of stroke, length of hospital stay, and degree of functional recovery. Finally, we assessed the opportunities for detection and treatment of post-stroke affective illness.

Method

All patients admitted to Nottingham hospitals with an acute stroke over an eight month period were followed up. Clinical information was collected on admission, and at one and six months afterwards, but mood was assessed by GHQ at six months only. Follow-up assessments were performed by a single observer. Neuroradiological investigations were not routinely available, so in most cases the diagnosis and location of the lesion were made clinically, using the World Health Organisation (1978) definition of stroke: 'Rapidly developing clinical signs of focal or global disturbance of cerebral function, lasting more than 24 hours, or leading to death, with no apparent cause other than of vascular origin'.

Severity of motor impairment was measured by summing scores for each affected limb (normal = 1, some weakness = 2, complete paralysis = 3). Patients' functional ability was assessed using the following activities of daily living (ADL): drinking, feeding, washing, transfers from bed to chair, walking, dressing, undressing, using the toilet, making a hot drink and bathing. Patients scored one point for each activity done independently (with or without aids) and zero if help or verbal prompting were required. A score of 10 indicated ability to carry out all the items, and a score of zero none of them. This has been shown to constitute a ranked ordinal scale, so that comparisons can be made even between patients with different kinds of disability (Ebrahim *et al*, 1985). The Mini Mental State examination (Folstein *et al*, 1975) was used to assess cognitive ability; patients with a score of less than 10 out of 30 were excluded from the study. To assess mood at six months a GHQ form was posted to each patient to fill in; this was collected by the interviewer during the home visit.

The GHQ consists of four component scales: somatic symptoms, anxiety and insomnia, social dysfunction and severe depression. Although a GHQ score of 5 or more (out of 28) is usually taken to indicate clinically important illness (Goldberg & Hillier, 1979), a score of 12 or greater is more appropriate for use with patients with physical illness (Bridges & Goldberg, 1984).

The non-parametric significance tests (Mann-Whitney U, Kruskal-Wallis ANOVA, and chi-squared) were used to examine differences between groups.

Results

During the eight month study period 463 cases of acute stroke were registered. Of these, 235 (51%) survived for six months, and all but 13 were followed up (eight had moved

TABLE I

Distribution of GHQ scores for different types of stroke patients

GHQ score	*Hemisphere stroke Left (n=73)*	*Hemisphere stroke Right (n=56)*	*Bilateral stroke (n=6)*	*Brainstem stroke (n=6)*	*Subarachnoid haemorrhage (n=8)*	*All groups (n=149)*
0	11	10	0	1	2	24
1–5	29	21	1	3	3	57
6–11	14	16	2	0	2	34
12+	19	9	3	2	1	34

TABLE II

Relationship between functional ability at one and six months after the stroke, motor impairment at six months, and GHQ score

Mean GHQ score at 6 months	*n*	*Mean ADL score One month*	*Mean ADL score Six months*	*Mean motor impairment score at six months*
0–5	81	7.9	9.4	4.59
6–11	34	5.7	8.1	5.15
12+	34	6.0	7.9	5.33
P^1		0.001	0.002	0.002

1. Kruskal-Wallis ANOVA test.

out of the area or could not be traced, and five refused follow-up). Assessment was not possible in some cases: 26 were dysphasic, 17 were too cognitively impaired, and 13 who were still in hospital could not be assessed in the same manner as those at home. Seventeen patients living at home did not receive a GHQ because of administrative errors. This latter group did not differ significantly in terms of age and sex distribution, functional ability or side of stroke from the rest. The final study sample was composed of 149 patients, all of whom were living at home, Their mean age was 69 years; 76 (51%) were men, 82 (55%) were married and 46 (31%) were living alone; 137 (92%) were living in their own homes, and the remainder were in sheltered accommodation. For the majority (84%) this was their first stroke.

Thirty four (23%) patients had GHQ scores of 12 or more, and all but two of them scored on the 'severe depression' component of the GHQ; two-thirds answered positively to three or more of the seven 'severe depression' questions. There were high levels of correlation (from +0.75 to +0.85) between the four GHQ component scales and the total score.

Laterality and affective illness

The distribution of GHQ scores for different types of stroke is shown in Table I. There was no significant difference in mean GHQ scores between left and right hemisphere strokes. Of those patients who were unable to walk at six months, three out of seven with left hemisphere strokes and four out of nine with right hemisphere strokes had scores of 12 or more. Of 38 patients who were dysphasic on admission but who improved enough to be assessed at six months, 9 (24%) had scores of 12 or more, compared with 25 (22%) of the 111 without speech problems.

Associations with disability and other factors

Strong relationships between GHQ score at six months and functional ability at both one and six months after onset of stroke and an association with motor impairment were found (Table II). Among seven patients whose disability increased between one and six months, three had high GHQ scores of 12 or more. Nineteen (25%) out of 76 patients whose ability improved had high scores, compared with 12 (18%) out of 66 patients whose ability did not change ($P<0.05$).

Functional ability at six months was strongly associated with length of hospital stay (Spearman $r=-0.55$, $P<0.001$), as was GHQ score at six months (Spearman $r=0.21$, $P<0.01$). Of 21 patients with a GHQ score of 12 or more, but with good functional recovery (ADL score of 9 or 10 at six months), 13 (62%) stayed in hospital longer than the median stay of 1/.4 days compared with 46 (47%) out of 98 patients with GHQ scores below 12 ($Z=2.31$, $P<0.05$). The GHQ score did not correlate with length of stay for patients with poorer functional recovery. Patients with sensory inattention also had higher scores ($Z=3.01$, $P<0.01$) but when disability was controlled for there was no association.

Age, sex, marital status, living alone and previous stroke showed no association with the GHQ score. Six out of 34 (18%) patients with scores of 12 or more were incontinent of urine, compared with 13 out of 115 (11%) with low scores. Significantly more patients with high GHQ scores had had impaired mobility (18%) and had been on antidepressants (6%) before their stroke.

Use of services

Patients with high GHQ scores saw the district nurse and social worker significantly less often than those with low scores, but the differences in frequency of family doctor visits (78, 52% overall) and out-patient attendance (48, 32% overall) were not significant. Only five (15%) patients with high GHQ scores were being treated with antidepressants at six months.

Discussion

The GHQ agrees well with other questionnaires used to assess depressed mood (correlation coefficients: $r=+0.86$, Zung; $r=+0.88$, Hamilton; $r=+0.94$, Present State Examination – Robinson & Price, 1982), although it may tend to overestimate the prevalence of affective illness as physical disability can contribute to the 'social dysfunction' component. Using a higher cut-off of 12 or more, as recommended by Bridges & Goldberg (1984), reduces this tendency, but increases the likelihood of missing cases. Twenty-three per cent of survivors had GHQ scores of 12 or more six months after their stroke, indicating a high probability of affective illness. It is not possible to compute estimates of the true prevalence of affective illness, as outlined by Bridges & Goldberg (1984), because measures of the accuracy of the GHQ for this type of patient are not available. If GHQ accuracy is low (ie. sensitivity and specificity about 80%) then the likely true prevalence of affective illness will be very low – about 5%. However, if the sensitivity and specificity of the GHQ are similar to values obtained from community studies (ie. sensitivity and specificity about 86%), the estimated prevalence of affective illness is 44%.

It is likely that most of the patients with high scores were suffering from depression, as so many of them answered positively to questions about severe depression. Furthermore, Bridges & Goldberg (1984) showed that the majority of in-patients suffering from neurological illness with scores of 12 or more did have depression or mixed depression and anxiety states, when compared with standardised psychiatric assessments.

Robinson & Price (1982) found that 23% of their patients had GHQ scores of 6 or more out of 28, but some were seen up to 10 years after their stroke, by which time symptoms might well have resolved. In another study Robinson *et al* (1984*a*), using different diagnostic methods, classified 30 of 50 patients seen six months after stroke as depressed. However, less than two thirds of the original cohort were assessed. Just over a third of those seen were labelled as suffering from major depression by DSM—III criteria (American Psychiatric Association, 1980) and this is comparable with the 23% of our group with GHQ scores of 12 or more.

Laterality and affective illness

Losses and life events are known to be important causes of depression in the elderly, and post-stroke depression may well be a reaction to loss of physical health and function (Murphy, 1982). However, Folstein *et al* (1977) found that stroke was more likely to be associated with depression than orthopaedic disability of similar severity. Robinson & Coyle (1980) have put forward an alternative hypothesis based on the catecholamine depletion that can follow diffuse or focal brain injury. They and others (Dimond *et al*, 1976; Giainotti, 1972) have shown that right hemisphere damage is more frequently associated with these biochemical changes in the rat and suggest that this may be associated with clinical depression seen in humans after stroke.

We found a strong relationship between the degree of physical disability and the likelihood of a high GHQ score, but no significant difference between left and right hemisphere lesions after controlling for degree of disability. A recent study by Robinson *et al* (1984*b*) showed an increase in depression scores with proximity to the left frontal pole and an inverse relationship for right hemisphere lesions. The strength of these associations depended on very small numbers of patients with very high and very low depression scores; thus they could well have been spurious. In contrast, Folstein *et al* (1977) demonstrated an excess of depression among patients with right rather than left hemisphere lesions. Lishman

(1968) studied 670 patients with traumatic penetrating lesions caused by bullets or shell fragments during World War II. He showed that among 113 patients with affective disorders, depressive symptoms and poor concentration were commoner with right hemisphere damage, particularly in the frontal and parietal regions.

Depression may often be missed in patients with left hemisphere strokes because of dysphasia; certainly fewer of these patients receive antidepressant treatment (Lim & Ebrahim, 1983). Exclusion of dysphasic patients from study at six months may have caused a biased assessment of the relationship of side of lesion with mood disturbance, but it is of interest that those patients initially suffering with dysphasia were no more likely to have a high GHQ score than those without speech disorder. It is probably unwise to generalise about mood disturbance from dysphasics who recovered their speech to those who did not, but out study is no more biased in this respect than others and had sufficient power to detect an important difference in laterality and mood disturbance.

Associations with disability

The link between stroke severity and likelihood of affective illness has not been a consistent finding in other studies. Robinson *et al* (1982, 1983) found no relationship with functional ability among 'chronic' stroke patients and no association with volume of brain damage (1984*b*). The numbers in our study were much larger, and the increased frequency of high GHQ scores in those whose physical disability increased between one and six months, and the association with degree of motor impairment both suggest that stroke severity and mood disturbance are strongly related. It is unlikely that the GHQ scores were merely mirroring physical disability, since patients with intermediate (6–11) scores and those with high (12 or more) scores had very similar levels of physical disability (see Table II).

Although our study was organised on a prospective basis, the GHQ was only used at the six month follow-up. Thus we are not able directly to assess the effect of affective illness on other outcomes. Nevertheless, as mood disturbance is likely to have been present soon after onset of a stroke (Robinson *et al*, 1984*a*), the positive association between GHQ score and time spent in hospital is likely to be causal. Depression is a recognised cause of failure of rehabilitation (Adams & Hurwitz, 1963) and we have shown that high GHQ scores contribute to longer hospital stays for patients with relatively minor functional disability. Patients with major disability stay in hospital much longer and mood disturbance does not appear to influence this.

Use of services

General practitioners have the greatest opportunity for detection of affective illness after stroke, as between 70% and 90% of patients aged over 65 are seen in any year by their general practitioner (Royal College of General Practitioners, 1974). Over half of the patients in our series had been seen in the month prior to interview. Nevertheless, only five (15%) patients with high GHQ scores were taking antidepressant drugs, despite evidence of efficacy in certain categories of patients (Lipsey *et al*, 1984; Rosen and Martorano, 1965). Increased awareness of post-stroke affective illness by doctors and therapists will help in providing support for both the patient and the family.

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rescribing Information ▼ **Presentation:** Round, yellow, enteric-coated tablets each
mg fluvoxamine maleate, imprinted with 'Duphar 291'. Pack of 60 tablets, basic NHS price
s: Treatment of those affective disorders characterised by persistent low mood, impaired
activities and psychosomatic complaints. **Dosage and Administration:** The tablets should
without chewing and with water. Adults: The effective daily dose usually varies between
00mg and should be adjusted to the individual response of the patient up to a maximum of
minimum recommended starting dose is 100mg given in the evening. A total daily dosage in
mg should be given in divided doses. Elderly: The same dosage regimen may be utilised in the
he general population. Children: There are insufficient data to recommend the use of Faverin in
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nent may be seen. Use in pregnancy and lactation: There has been little human use but there
adverse animal evidence. Avoid in pregnancy and in nursing mothers if a better known and safer
available, although safety in pregnancy is likely. Interactions with other drugs: In interaction
studies increased plasma levels of propranolol and warfarin were seen during concurrent administra
Faverin. Care should be taken when Faverin is prescribed to patients already receiving propranol
warfarin as the dose of these drugs may need to be lowered. No interactions were seen with digoxin
atenolol. **Side-effects:** The commonest symptom observed with Faverin administration is nausea
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during clinical studies were somnolence, constipation, agitation, anorexia and tremor. Other reported
are often associated with the illness and their incidence in controlled trials is comparable t
experienced with placebo. Faverin may cause a decrease in heart rate. During treatment, limited chan
repolarization were observed in the ECG, but no causal relationship could be demonstrated. The incide
postural hypotension was similar to that seen in patients treated with placebo. Treatment of overdosa
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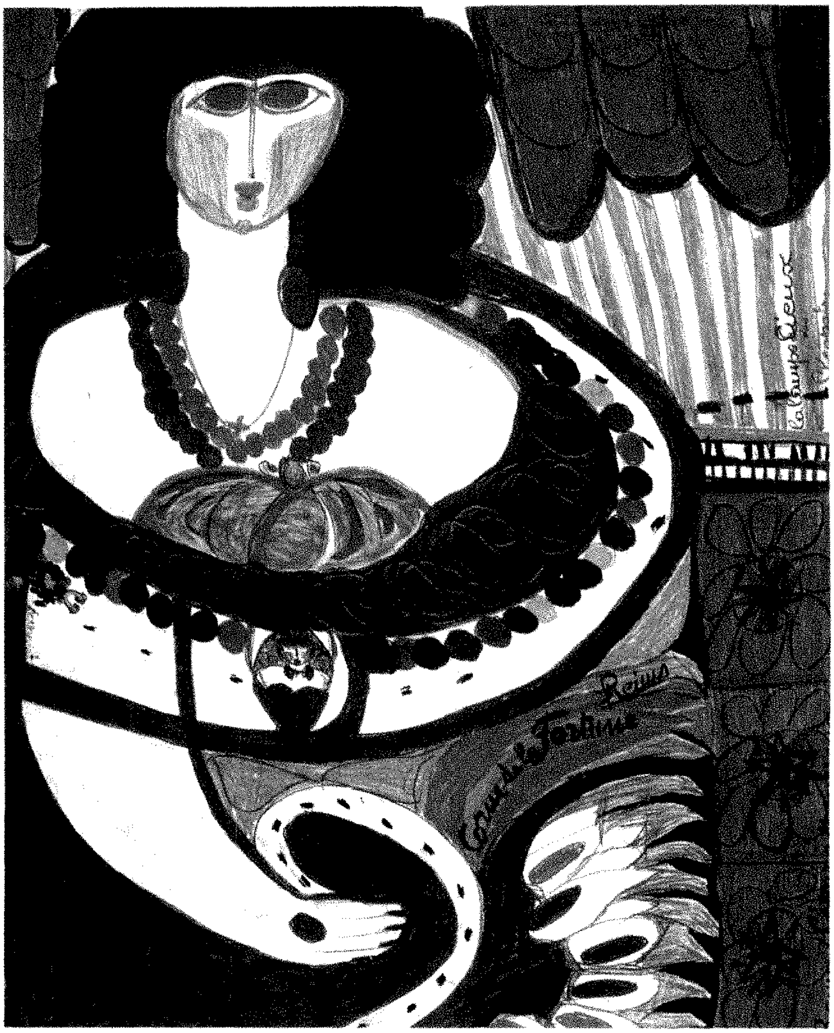

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References: 1. Pakes G. E. In 'Therapeutics Today' Ed D. A. W. Johnson. ADIS Press. (1982), **2**.
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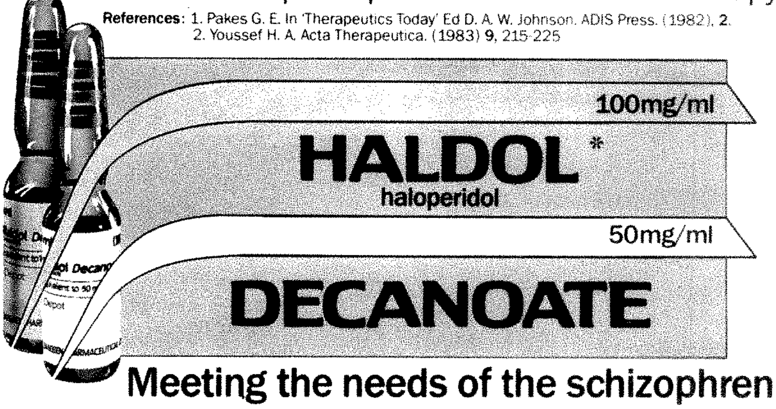

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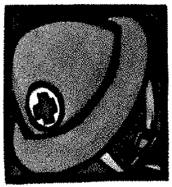

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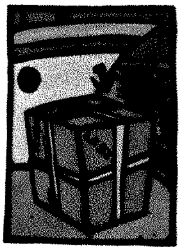

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British Journal of Psychiatry (1987), **151**, 57–62

The Outcome of Neurotic Disorders After Out-patient and Day Hospital Care

P. TYRER, M. REMINGTON and J. ALEXANDER

New psychiatric out-patients with depressive, phobic, and anxiety neurosis were randomly allocated to out-patient care or to one of two types of day hospital treatment, one specialising in psychotherapy and the other offering all forms of day care. Of 106 patients who entered the study, 78 had assessments of psychiatric symptomatology and social adjustment both before treatment and after 4, 8 and 24 months. There was no significant difference in outcome between depressive, phobic, and anxiety neurosis, and no overall difference in response to treatment between the three types of care. Suicidal symptoms were significantly less common in out-patients. In many respects, neurotic disorder can be regarded as a single syndrome.

Considerable argument still occurs over the most appropriate treatment for the great range of neurotic disorders seen in clinical practice. Although there is good evidence that behaviour therapy (Marks, 1981), cognitive therapy (Beck *et al*, 1979), and drug treatment with tricyclic antidepressants and monoamine oxidase inhibitors (Rowan *et al*, 1982; Sheehan *et al*, 1980) are all effective treatments, there is no consensus on their relative value. Treatment is also governed by important practical considerations such as the availability of adequate time and personnel; for example, if psychotherapeutic procedures were preferred, they could not yet be used on a wide scale because of the cost involved in training extra personnel. However, the expansion of day hospital services over the past 30 years has offered an opportunity of treating patients with neurotic disorders more intensively than as out-patients, and also relatively cheaply. This has led to the growth of three classes of day patients: one consisting of relatively serious psychiatric illness that might otherwise need psychiatric in-patient care if the day hospital were not available (Hirsch *et al*, 1979; Dick *et al*, 1985); another with chronic disorders that come for support rather than an active treatment programme; and a third group of less seriously ill patients, frequently with neurotic disorders, who attend the day hospital for specific treatment, which is often psychotherapeutic (Gath *et al*, 1973).

We have presented the preliminary findings of a study of the efficacy of out-patient treatment for the three most common neurotic disorders – anxiety, phobic and depressive neurosis – compared with treatment in two day hospitals, one specialising in psychotherapeutic intervention and the other offering a range of treatments to patients with all types of psychiatric disorder (Tyrer & Remington, 1979). No important differences were found between the two types of care with respect to either symptomatic change or social adjustment, but patients in general were more satisfied with out-patient treatment than with day care. We report here further details of the outcome of these patients, with assessments made two years after initial treatment.

Method

The main part of the study was between March 1976 and November 1977. New patients referred to the out-patient clinics of four consultant psychiatrists and two academic consultants in Southampton were included if they were diagnosed as having anxiety, phobic, or depressive neurosis, using the terminology of ICD-8 (World Health Organization, 1967), had a disorder of intermediate severity so that attendance as a day patient or as an out-patient was equally feasible and acceptable to the patient, and were aged between 16 and 60. After initial assessment, the patients were allocated randomly to out-patient or day care. Those allocated to out-patient care continued to attend the clinics of the consultant team to whom they were originally referred. Of those patients allocated to day care, half were referred to a specialised day hospital that had a higher level of staffing (one professor, one lecturer, one senior house officer, one part-time psychologist, and three trained day nursing staff for a unit with six beds and up to ten day patients). This concentrated on psychotherapeutic management (including individual and group psychotherapy and psychodrama) and had a selection bias in favour of neurotic patients. The remaining patients were referred to a more typical day hospital which had a mixed therapeutic orientation and catered for a wider range of psychiatric disorder; the staffing of this comprised one consultant, one registrar, four trained nursing staff, and one part-time psychologist for a maximum of 40 patients.

Assessment of symptoms was made without knowledge of the patients' treatment and form of management. Ratings included a modified form of the Present State Examination (PSE) (Wing *et al*, 1974), in which cut-off instructions were ignored, all items referring to neurotic symptomatology scored, and additional items of anxious symptomatology (cardiovascular symptoms, respiratory symptoms, gastrointestinal symptoms and tremor) also included. The scoring of these items included both the conventional categorical rating of the PSE and scores on analogue scales, each of 100 mm. Social adjustment was assessed using the Social Functioning Schedule (Remington & Tyrer, 1979), in which function in 12 areas of activity is recorded on analogue scales. Both clinical status and social functioning were assessed before treatment and after four and eight months. A total of 106 patients were included in the study, of whom 89 completed treatment with these two assessments. No significant differences were found between the three forms of care for global symptomatology and social adjustment, but scores for individual symptoms and diagnostic groups were not analysed at that stage.

Because many of the patients were still handicapped by symptoms and were receiving care after eight months of treatment, and because there is evidence that the full effects of psychotherapy are not manifest for many months (Weissman *et al*, 1974), patients were followed up two years after their first assessment. All were contacted by letter, and assessments made at their home. If contact was not established, further enquiries were made to determine the patient's whereabouts and likely progress. Even if patients were not interviewed, further information was obtained for as many of them as possible, since the results of follow-up enquiries in neurotic disorder can be vitiated by lack of information on a significant proportion of patients, who often provide the most important information (Sims, 1973). Ratings of clinical state and social adjustment were made using the same instruments as in the earlier assessments, so that direct comparison could be made. Good inter-rater reliability had been established previously with the use of tape recorded interviews and consecutive assessments of patients, and the reliability of the analogue PSE ratings was shown to be at least as good as the conventional categorical scoring system (Remington *et al*, 1979).

Results

Patients

During the 21 months of recruitment, 264 patients were screened as possibly suitable for entry; 158 of these were excluded at initial interview (49 who could not attend or were considered not suitable for day hospital treatment, 20 whose disorders were considered too minor, 17 for whom the diagnosis was not that of depressive, phobic, or anxiety neurosis, 9 who were admitted to hospital immediately, 3 for other reasons, 26 who did not attend, and 34 for whom no reason was given by the doctor for their exclusion). Of the 106 patients included, 78 (74%) were seen at four months, eight months, and two years. The reasons for failure to complete assessments are given in Table I. No patients were in-patients at the time of follow-up, and all patients who refused interview had no current contact with the psychiatric services. There was a similar drop-out rate in each of the treatment and diagnostic groups. Of the 78 patients completing assessment, 51 had an ICD-8 diagnosis of depressive neurosis, 16 a diagnosis of phobic neurosis, and 11 a diagnosis of anxiety neurosis.

Differences between treatments

The clinical and social adjustment ratings showed roughly normal distributions, and so differences between groups over time were subjected to parametric analyses, primarily of covariance. During the two years of follow-up it was considered unethical to confine patients' treatment to either day hospital or out-patient settings, and clinical management was left to the discretion of the individual clinician concerned. In the first eight months of treatment a detailed record was kept of all treatment given; this has been

TABLE I
Reasons for drop-out during study

	General day hospital: n	*Specialised day hospital: n*	*Out-patient: n*
Entering study	23	25	58
Completing after 2 years	14	18	46
Reasons for dropping out			
Dead	2[1]	1[2]	—
Refused interview at some stage of study	2	3	4
Emigration	2	—	—
Moved from Southampton area	1	2	5
No contact made, whereabouts not known	2	1	3

1. Suicide.
2. Homicide (see text).

TABLE II

PSE symptoms showing significant differences in outcome between treatment groups

PSE symptom[1]	*Mean percentage improvement in symptom*[2]								
	4 months			8 months			24 months		
	DG	DS	OP	DG	DS	OP	DG	DS	OP
Worrying	69.7*	40.0	42.4	57.0	49.5	40.7	52.4	64.5	43.3
Anxious foreboding	70.5*	42.8	61.2	72.9	41.3	54.0	81.0	81.7	81.3
Panic attacks	38.5	79.6	43.9	30.4	78.5	57.9	53.5	77.1*	61.5
Hypochondriasis	−6.0	56.1*	−13.0	54.0	53.5	15.6	50.0	73.7	19.5
Tremor[3]	41.1	34.3	27.4	33.8	61.7	40.7	40.5	62.7*	55.6
Subjectively ineffective thinking	71.5*	−12.6	45.1	37.6	25.7	46.7	68.9	77.7	66.7
Poor concentration	67.0*	21.9	46.4	50.3	49.9	51.5	59.4	70.0	56.9
Loss of interest	61.0	36.4	43.5	85.2	57.8	50.9	55.5	74.3*	64.3
Hopelessness	57.7	40.2	37.8	88.5	90.0*	42.9	74.9	74.3	72.7
Suicidal plans or acts	67.6	62.2	42.3	90.7	73.8	74.8	75.6	63.4	91.9**

1. Symptoms not included showed no significant differences at any stage of the study.
2. DG = general day hospital ($n = 14$), DS = specialised day hospital ($n = 18$), OP = out-patient groups ($n = 46$).
3. Not included in full PSE.

*Improvement significantly better than for other groups, $P < 0.05$; **$P < 0.001$.

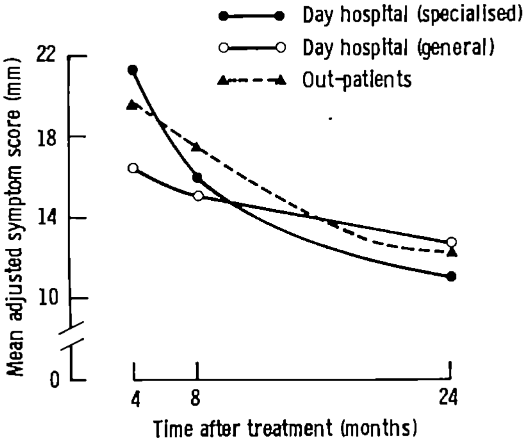

FIG. 1 Mean adjusted PSE scores of each treatment group

summarised by Tyrer & Remington (1979). Predictably, patients attending the out-patient clinic had a significantly shorter duration of treatment, a smaller number of attendances, and less group and occupational therapy. Between the eight-month and two-year follow-ups, only three patients originally allocated to the out-patient group had been referred to a day hospital facility, and in all three cases this was for less than two months. A much larger proportion (76%) of patients originally treated at a day hospital had received some form of out-patient care in this time. The data after two years therefore came from an out-patient group that had received out-patient care almost universally throughout this period and a day patient group who had received a mixture of day hospital and out-patient care. A small number in each group had also received in-patient care (six in the day hospital and four in the out-patient groups).

The mean adjusted symptom scores on the analogue forms of the PSE showed no significant differences between the three types of treatment. Although patients in the general day hospital showed more rapid early improvement, this was not maintained, and the patients at the specialised day hospital showed most of their improvement later in the course of treatment (Fig. 1). These differences, however, fell short of significance ($F = 1.2$). Similar findings were shown with the mean adjusted social function score, except that the amount of improvement shown over the course of the two years was less than that for symptoms (Fig. 2).

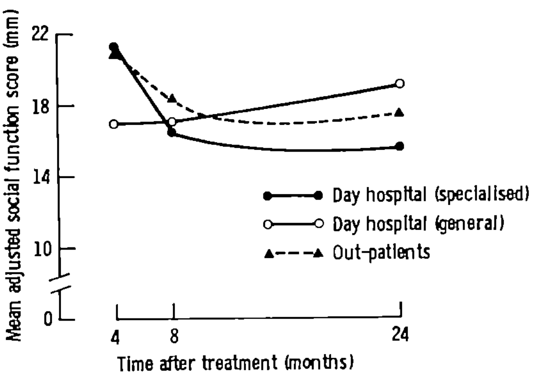

FIG. 2 Mean adjusted social function scores of each treatment group

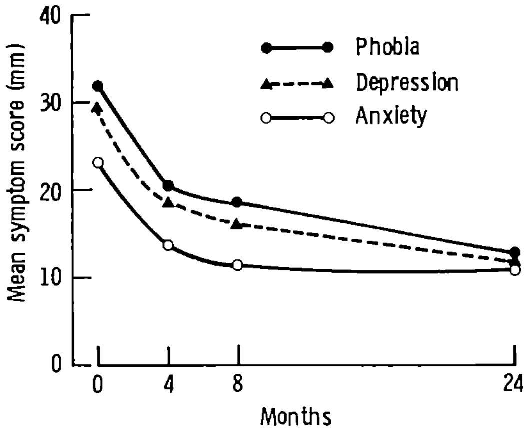

FIG. 3 Mean PSE scores of each diagnosis group

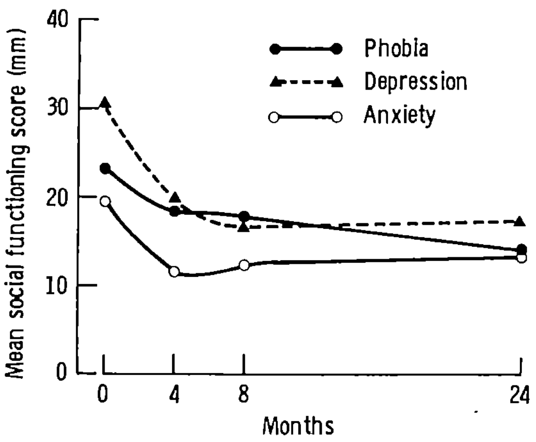

FIG. 4 Mean social function scores of each diagnosis group

The 30 symptoms constituting the mean symptom score were analysed separately; the results for these showing significant differences are given in Table II. After four months the symptoms of worrying, anxious foreboding, subjectively ineffective thinking, and poor concentration were significantly better in the general day hospital group, while one symptom, hypochondriasis, improved more in the specialised day hospital group. After eight months, ratings of hopelessness were significantly better in the specialised day hospital group, as were the symptoms of panic attacks, tremor, and loss of interest in the same group after two years. For all these items significant superiority was only shown at the 5% level; in view of the large number of comparisons, several of these could have occurred by chance. The only highly significant difference was for ratings of suicidal plans or acts, where the out-patient group had significantly lower scores than both day hospital groups at follow up ($P < 0.001$).

Differences between diagnostic groups

There was a similar improvement in the symptom scores of all three diagnostic groups (Fig. 3). Phobic patients had the most symptoms at the beginning of treatment, but this was not significantly different from the other two groups. By the time of follow-up, all three groups had similar mean symptom scores. a similar pattern was shown for ratings of mean social function (Fig. 4). At the beginning of treatment, phobic patients had significantly worse social functioning than anxious ones ($F=4.2$; d.f. 2, 75; $P < 0.02$), but at follow-up these differences became less and were not significant. After two years, the mean social functioning was similar in all three groups.

Discussion

The results show that there is no appreciable difference between the short or medium-term outcome of neurotic patients treated initially at out-patient clinics or at day hospitals. Thus, it is fair to conclude that a day hospital facility for the treatment of neurotic disorders alone is not a necessary part of a psychiatric service. However, as the selection criteria for the study ensured that the patients treated had an intermediate degree of handicap which allowed out-patient and day hospital care to be practical alternatives, it would be wrong to generalise too widely from the findings. Day hospitals are now used increasingly as an alternative to hospital admission, and the degree of handicap appropriate to this use was excluded from our study.

Such differences as were shown favoured each type of care, but at different times of follow-up. The general day hospital had its greatest therapeutic impact early on in treatment, and tended to show a better outcome than the other facilities after four months. As the general day hospital provided a full range of treatments, particularly those directed to symptomatic improvement, this result is not surprising. What is unexpected is the failure of patients to maintain their improvement over the two-year period of follow-up, to the extent that after two years this group showed greater social dysfunction than after four months. It is possible that the availability of all psychiatric treatments in a supportive setting might prevent investigation of the underlying causes of poor social functioning and also lead to a degree of dependence on the day hospital.

The specialised day hospital showed an opposite trend, with relatively poor improvement after four months and steadily greater improvement thereafter, so that after two years this group had the best social adjustment and symptomatic outcome (Figs 1 and 2). This is in keeping with evidence that the beneficial effects of dynamic psychotherapy often take many months to become established fully, and that a period of increased distress and symptomatology often precedes the improvement (Weissman *et al*, 1974).

The out-patients occupied an intermediate position in outcome, and showed only one important difference from the two day hospitals groups – on ratings for suicidal behaviour after two years ($P < 0.001$). It may also be relevant that two patients committed suicide in the day hospital groups and one was killed by his wife during the period of follow-up; she was subsequently cleared of murder and convicted of manslaughter because of his extreme provocation. This finding is also consistent with evidence of greater distress during the initial phase of psychotherapy, and suggests that patients thought to be at risk of suicide should be assessed carefully before any form of psychotherapy is considered.

Taking the relative costs of out-patient and day patient care into consideration, the findings indicate that out-patient treatment is a cheap and effective form of management, but could be improved further by developing a greater range of treatments, including psychotherapy. Day hospital care is also probably effective, but as it was combined with out-patient care for much of the follow-up period it is difficult to establish its main contribution to outcome. The future development of psychiatric day hospitals cannot be planned clearly because of equivocal findings concerning their value (Vaughan, 1985), but the results of this study counsel caution in developing specific facilities for treating neurotic disorder.

Economic considerations also are important in preferring different types of psychiatric service. Day hospital treatment is considerably cheaper than in-patient care (Dick *et al*, 1985), and when all the administrative costs of running a day hospital are taken into account, out-patient care is cheaper. One advantage of the day hospital is that less expensive medical time is spent with the patient. Analysis of the length of time spent in treatment by each patient after eight months, however, showed that out-patients averaged four hours of treatment, whereas the average duration of therapy of each day patient was 103 hours in the general day hospital and 73½ hours in the specialised day hospital (Tyrer & Remington, 1979). These figures suggest that although the out-patient contact may be more expensive per unit of time, it is considerably more economical over a course of treatment.

The outcome of the three diagnostic groups also showed no important differences in the two years of follow-up. Anxious patients had significantly better social functioning and relatively fewer symptoms than phobic and depressed patients before treatment (Figs 3 and 4), but this difference disappeared over time. This agrees with other evidence that anxious patients have relatively good social functioning compared with other diagnostic groups (Casey *et al*, 1985), and that phobic and depressed patients differ from anxious ones in having specific symptoms together with those of anxiety, rather than having qualitatively distinct disorders (Prusoff & Klerman, 1974). In the course of follow-up the symptoms and social function scores of each of the three groups converged, so that after two years the mean scores were virtually identical. Thus, the distinguishing features that helped to make the diagnosis before treatment did not remain consistent over time. Our findings confirm other evidence that the temporal reliability of neurotic disorders is low (Kendell, 1974), and that for most purposes neurotic patients can be treated as a relatively homogeneous group, those having higher PSE scores at initial assessment usually being classed as having phobic or depressive disorders, and those with lower scores being diagnosed as having anxiety neurosis.

The evidence of this study, together with the arbitrary nature of current classification of neurotic disorder (Tyrer, 1984), suggests that many of the diagnostic subdivisions of neurosis are of limited clinical relevance (Tyrer, 1986) and that many cases could be classified as a single disorder, the general neurotic syndrome, which runs a prolonged course with variation in the predominance of different neurotic symptoms over time (Tyrer, 1985). The difference in outcome between depressive and anxiety neurotic disorders shown by some workers is not as contradictory as it first appears; e.g. Schapira *et al* (1972) found a greater percentage improvement in depressed patients compared with those with anxiety states over a period of four years after first assessment. Our findings are similar, except that we make the important additional point that patients with predominant anxiety are at a relatively better phase in the course of the general neurotic syndrome, and so are less likely to improve than patients with many more symptoms, including depression. Thus each patient can have predominantly depressive, phobic, and anxiety symptoms at different times; the labelling of the neurosis through its dominant current symptomatology is not such an important distinguishing feature as conventional classification would have us believe.

More evidence is needed, using DSM-III (American Psychiatric Association, 1980) and other systems of classification more appropriate to research studies than ICD-8, before this conclusion can be accepted. Nevertheless, it suggests that other research workers should take into account temporal variations in neurotic symptomatology, as well as those dominating at the time of presentation, before attaching firm diagnostic labels.

Acknowledgements

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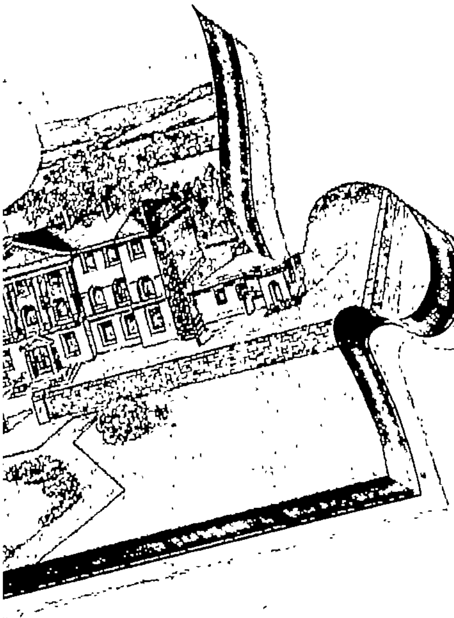

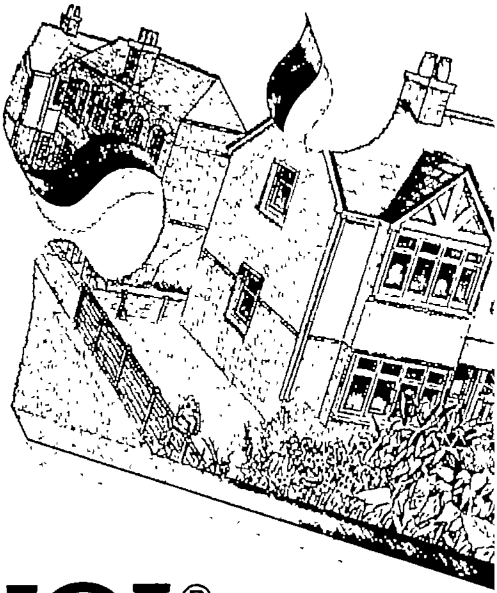

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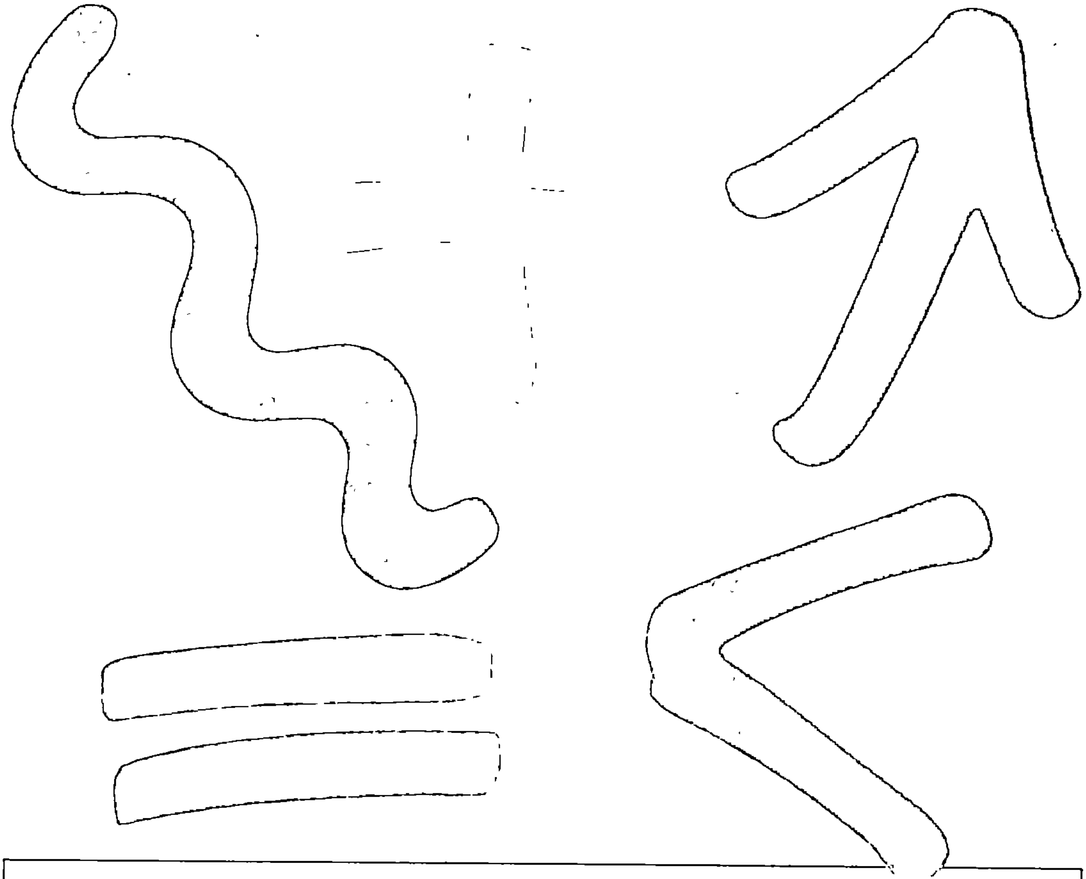

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Does Violence Have Cognitive Correlates?

G. ROBERTSON, P. J. TAYLOR and J. C. GUNN

The relationship between cognitive function and violence in 76 remanded prisoners, without formal psychiatric illness, was investigated. The violent group tended to be of slightly lower general ability than the non-violent group, but not abnormally so in relation to the general population; no relationship was found between specific patterns of cognitive functioning and violence. The violent group reported significantly higher levels of neurotic symptoms than the non-violent group, and were more socially deviant. To a small extent, general intelligence (reasoning ability), in interaction with many other factors, may be related to a propensity for violent behaviour, but no particular aspect of cortical functioning seems to be related to violence.

The search for features of brain structure and function which underlie behaviour is inevitable. While social and other environmental influences are undeniably important in formulating behaviour, neuronal activity is the final common pathway for perception, judgement, and action.

In the field of violence and cerebral pathology there has been an almost exclusive preoccupation with the link between cerebral dysrhythmias and aggression, perhaps principally because of the episodic nature of most violence; Kligman & Goldberg's critical review (1975) still stands as one of the most important contributions. Little can be inferred from most of the data, since terminology, whether covering behaviour or electroencephalogram (EEG) findings, is vague and over-inclusive. Double-blind studies, despite being easily conducted in this field, are almost non-existent, although Fenton *et al* (1974) reported one in a series of special hospital patients which showed no more than a slight trend for patients with posterior temporal slow wave anomalies to be more violent than those with normal EEGs. The organic contribution to violence is even more difficult to interpret from other neurophysiological studies, since most refer to 'psychopaths', the definition of which is often based on subjective criteria and leaves the level of violence unclear. Howard (1984) attempted to tighten the criteria, but even so, the nature and degree of violence within the population must generally be inferred from such data. Certainly the relationship between violent behaviour, personality type, and EEG abnormality is extremely complex.

Neuropsychological testing has been, perhaps, the greatest growth area. Spellacy (1978) reported that he could classify subjects as violent or non-violent with a 95% accuracy on a neuropsychological test battery; his violent sample was of significantly lower general intelligence than his non-violent group. Krynicki (1978) also used a neuropsychological test battery to compare repetitively assaultive adolescents with non-violent delinquents and an age-matched group with organic brain disorder. The violent and the organic groups were almost indistinguishable, with a tendency for left-sided disturbance to predominate in the violent group.

The remaining literature, in common with the neurophysiological material, focuses on 'psychopathy' – defined on various scales – with the recurring difficulty that raw data on violence are not reported. In relation to psychopaths, Flor-Henry (1973) has asserted that this condition is associated with left-hemisphere pathology, although his early data was from a small series of seven 'homicidal psychopaths', six of whom, on neuropsychological testing, showed disorder in the anterior limbic region, five of them bilaterally but with a stronger tendency to right-sided disorder. Yeudall *et al* (1981), from the same department, reported that 70% of aggressive psychopaths had abnormal neuropsychological profiles, indicating dysfunction localised to the frontal and temporal regions of the brain. Fedora & Fedora (1983) added that the psychopaths differed from controls only on neuropsychological variables considered to be left hemisphere, and on anterior/impulsivity markers. Gorenstein (1982) reported a relationship between frontal lobe deficits and psychopathy, but his findings have been questioned by Hare (1984), who stressed the need for more adequate definitions of what constitutes psychopathy and for caution in the interpretation of studies involving cognitive tests. In so far as localisation of function can be inferred by the studies described, evidence does favour the association of disruption of left-hemisphere function with violence.

The purpose of the study reported here was to determine, using a specially devised psychological test battery, whether any cognitive differences could be found between men who had shown a predisposition to violence and those who had not. Earlier work by Taylor *et al* (1982) had indicated that, in terms of handedness, non-violent men and men who had been violent on one occasion only could be distinguished from those with a more substantial record of violent behaviour. The criterion used for inclusion in the violent group in the present study was that the man had a long and significant record of violence. The relationship between violence and cognition was investigated in a mentally ill population and for men regarded as non-ill. This paper concerns the latter population; a report on the former is in preparation.

Method

Subjects

Between April and December 1982, 167 men, 76 of whom were not suffering from any formal mental illness, were interviewed and tested. All the non-ill men were tested and interviewed in Brixton prison, which was chosen because it holds men during the period before trial, thus providing a large turnover of potential subjects. The range of 'violence' represented in this population was enormous both in terms of severity and frequency.

Both violent and non-violent non-ill groups were selected, in some measure, by exclusion criteria. Men with a history of any of the following were excluded: schizophrenia, affective psychosis, epilepsy, brain injury, other known or suspected organic illness, or dependence on drugs or alcohol. Selection was carried out on the basis of age compatibility with the ill group, and men were selected for the violent, non-ill subgroup if the offence with which they were charged was of serious personal violence and if their records also indicated that they had been convicted of violent offences in the past.

Each man selected was called to the hospital wing and asked if he would take part in the study. A brief explanation of its purpose was provided, and the confidential and voluntary nature of the study stressed. Most men agreed to participate, but a number who had been chosen on the basis of their violent record alone refused; these tended to be those with extremely long criminal records of extensive violence.

Procedure

Having agreed to take part, each man was interviewed and assessed on a battery of cognitive tests developed for this project. Interviews took 20–40 minutes to complete, and covered criminal and psychiatric history, basic demographic material such as education, marital status, and employment, and the circumstances surrounding the man's arrest and detention (subjects were prepared to take on trust assurances of confidentiality, and spoke freely about the latter despite the fact that some were going to plead not guilty). Cognitive testing followed the interview, and took 1–1½ hours. Each man was then asked to complete a present state symptom checklist – the SCL-90 (Derogatis *et al,* 1973) – which allowed him to describe his subjective experience of psychopathology, while the interviewer completed the observer ratings of the Comprehensive Psychopathological Rating Scale (CPRS) (Asberg *et al,* 1978) for an objective description.

Tests used

Wechsler Adult Intelligence Scale

The Wechsler Adult Intelligence Scale (WAIS) (Wechsler, 1955) was used to measure verbal IQ (prorated from vocabulary, similarities, and digit span sub-scales) and performance IQ (prorated from picture completion, block design, and picture arrangement sub-scales).

Verbal fluency

A decrease in verbal fluency has been reported as being associated with frontal brain damage (Benton, 1968). In the present study, verbal fluency was measured on four subtests, all of which required the production of words for one minute each. The first part – 'free association' – allowed the production of any word (except plurals, tenses, etc); the second required the person to name as many animals as he could think of in the space of a minute; and the third and fourth parts required the production of words beginning with the letters S and F respectively. These results are reported as the total number of legitimate words recorded in each one-minute period.

Visual retention test

Brain-damaged patients with lesions of the right hemisphere have been reported as deficient in their recognition of unfamiliar faces (Warrington & James, 1967). This has been used as the basis of three sub-tests, grouped here as the 'visual retention test'; each was designed to reflect hemispheric function, other factors being held constant. In each sub-test 20 pictures were shown, individually, for approximately three seconds each. Immediately afterwards the same 20 pictures were presented, this time each one being set alongside a similar picture. The task was to identify the picture he had seen initially. The score was taken as the total number of wrong answers out of 20.

The first sub-test ('simple verbal') consisted of single items which were deemed to be easily remembered in a verbal fashion, e.g. a car or a packet of cigarettes. The second ('mixed function') consisted of items which were much more complicated or which required much greater discrimination for correct identification at second presentation than those in the first sub-test. The third subtest ('faces') consisted of 20 pictures of unfamiliar faces.

The theory underlining the design of the tests was that they should reflect a shift from non-verbal (right hemisphere) function through to purely verbal (left hemisphere) function.

Visual recognition test

This was adapted from a procedure used with a brain-damaged population (Faglioni *et al*, 1969). The adaptations reflect our interest in pattern of response. In each of the four sub-tests the subject was asked to identify a letter or figure at the top of a column and then match that letter or figure with one of a series of eight or five alternatives presented below it. The time taken to identify the correct figure was recorded, as was the number of errors made; the cumulative time taken for the items in each sub-test was calculated, and is used to represent the test result. Sub-tests were designed to reflect a shift in function from 'pure' verbal function (sub-test 1) through to 'pure' spatial organisation (sub-test 4). They have been titled: (1) 'alphabet'; (2) 'embedded letter'; (3) 'broken letter'; and (4) 'broken figure'.

In addition to these tests, reading age was assessed, using the Schonell Graded Word Reading Test (Schonell & Schonell, 1965), and handedness preference, using the Annett Handedness Preference Scale (Annett, 1970).

Results

The division into violent and non-violent subgroups was made on the basis of the scores obtained by men on two violence rating scales (Gunn & Robertson, 1976). The first of these five-point scales was concerned with the present offence, and the second with the man's record of violence; details are given in Appendix 1. Because of the manner of selection of the violent group, the previous and present violence scales were highly correlated in the non-ill population ($r=0.72$), and it was decided that the best distribution of the criterion variable would be obtained by totalling these scales. Those men with a total score of 0–3 were placed in the non-violent group, and those with scores of 4–8 in the violent category. Only one man had a score of 4, and the criterion cut-off point is considered adequate.

The raw scores of the cognitive test data were transformed to z standard scores, with a mean of 0 and a standard deviation of 1; this information is presented in the form of cognitive profiles in Fig. 1. The most striking feature of the two profiles is their similarity in pattern. The two groups were not found to produce statistically different distributions on any of the sub-tests used, although the violent group produced inferior scores on every sub-test bar one (the similarities sub-test of the WAIS). The two groups were similarly distributed with regard to age, the mean for the non-violent group being 34.4 (s.d. = 8.94 years) and that for the violent group 31.4 (s.d. = 8.84 years), and with regard to the assessment made of their reading ability ($t=0.24$, $P<0.81$).

Although not different on any single cognitive test in the battery, the two groups could be distinguished from one another fairly well in terms of overall functioning. Evidence of this was provided by a classification of cases into violent and non-violent groups on the basis of scores determined from a discriminant function analysis. Four men were excluded from the analysis because of missing data. The overall rate of correct identification was 75%. Of the 34 men in the non-violent group, 24 (71%) were correctly classified on the basis of their scores on the function derived from the analysis and 10 (29%) were misclassified. Of the 38 in the violent sample, 30 (79%) were correctly grouped and 8 (21%) incorrectly categorised on the basis of their discriminant function scores. The nature of the derived function, as assessed by the discriminant function coefficients of the sub-test, appears to be non-specific as regards hemispheric function and biased towards higher reasoning ability. Irrespective of sign, the sub-tests with the highest loadings were: similarities sub-test (WAIS), 0.72; production of words beginning with the letter S, 0.71; performance on the broken figure and embedded letter sub-tests in the visual recognition group, 0.58 and 0.51 respectively; and scores on the block design sub-test (WAIS), 0.48.

The two groups were also found to differ on a number of variables related to social functioning (Table I). Although similar proportions of men in both groups had married, the violent group contained significantly more men who were either separated or divorced. Long-term unemployment was also more frequent among the violent men, all of whom had had a previous criminal record (compared with 80% of the non-violent sample), and significantly more violent men had juvenile convictions on their records.

Table II shows the psychiatric variables on which the two groups differed. There were indications of greater instability in the violent sample; for example, a greater proportion of them had previously received psychiatric treatment, and more were receiving medication at the time of interview. Despite large within-group variability, the two groups were also distinguished with regard to the reporting of neurotic symptoms on the SCL-90 self-report schedule. In particular, the violent group tended to report a greater awareness of paranoid feelings, although men in the violent group may in fact have had real enemies in the criminal underworld who wished them harm. The violent group also proved to be 'deviant' in terms of their previous drinking and other drug-taking behaviour. The two groups did not differ when rated by the interviewer on the observed psychopathology section of the CPRS rating scale; however, such scales are relatively insensitive, particularly when used on subjects without formal psychiatric illness.

Discussion

In regard to the pattern of their cognitive profiles, the violent men in this study did not differ from non-violent men. A discriminant function analysis produced a good degree of separation between the groups, and this has been interpreted as reflecting relative inferiority among the violent group in terms of general ability. It may be argued on the basis of the discriminant analysis (e.g. the high loading of the similarities sub-tests and of one of the verbal fluency

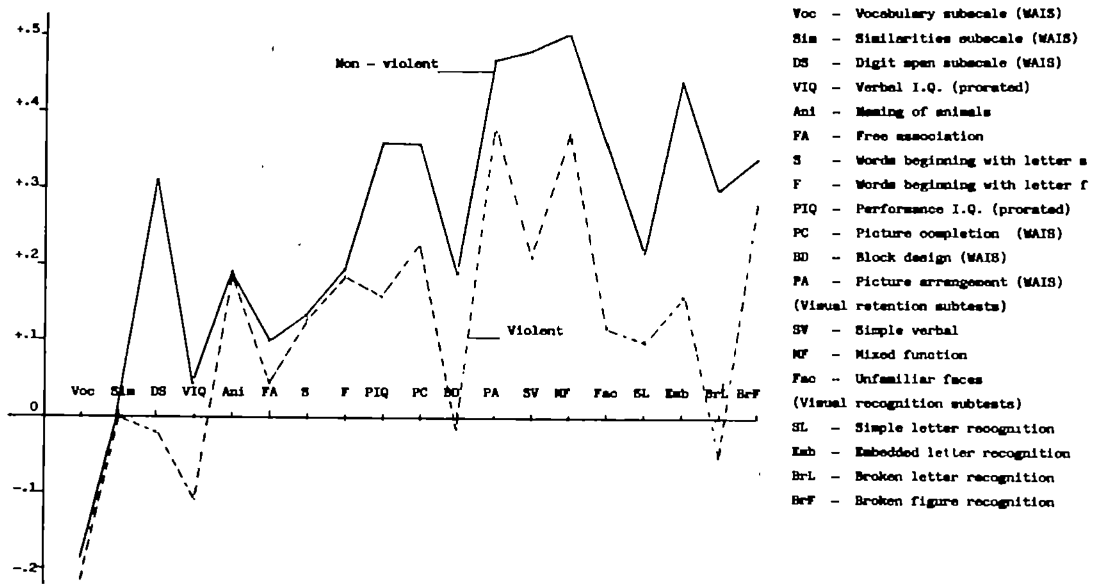

FIG. 1 Comparison between violent and non-violent groups (all scores have been subjected to a Z transformation, so that mean = 0 and s.d. = 1).

TABLE I

Comparison of violent and non-violent, non-ill groups on social and criminal variables

	Violent (n = 35)		*Non-violent (n = 41)*		*t*	*P*
	Mean	*s.d.*	*Mean*	*s.d.*		
Drink rating (historical)	1.68	1.15	0.79	0.85	3.92	0.001
Drugs rating (historical)	0.74	0.95	0.29	0.56	2.46	0.017
Age at first offence	15.03	3.35	22.85	11.20	4.21	0.001
Number of juvenile offences	3.28	4.64	0.88	1.74	2.90	0.006
Total court appearances	12.88	8.42	5.48	5.29	4.44	0.001
Previous prison sentences	4.71	4.84	1.44	2.20	3.69	0.001
	Yes	*No*	*Yes*	*No*	χ^2	*P*
Whether separated or divorced	13	22	6	35	3.97	0.05
Unemployed more than 6 months	20	15	12	29	6.02	0.02

sub-tests) that frontal lobe functioning is particularly impaired in the violent group. However, such an interpretation would be incorrect, as it was on the similarities sub-test that the violent group produced the higher mean score. Furthermore, a higher proportion of men in the violent group were receiving medication (Table I); those who were on medication were significantly inferior on the similarities sub-test to those who were not. The mean score for men not on medication was 11.03 (s.d. = 1.63), compared with a mean score of 9.16 (s.d. = 1.60) for those receiving drugs ($t = 2.55$, $P < 0.015$).

Rather than implicating a particular area of the brain, the present results are better accounted for in terms of a 'general cortical function' or 'general intelligence' model. The overall distribution of the prorated intelligence quotients for the violent group, though slightly inferior to that of the non-violent

TABLE II

Comparison of violent and non-violent, non-ill groups on psychiatric variables

	Violent ($n=35$) Mean	s.d.	Non-violent ($n=41$) Mean	s.d.	t	P
SCL-90 total score	85.48	62.76	56.75	53.96	2.12	0.038
SCL Paranoia scale	7.33	5.76	3.32	4.03	3.39	0.001
	Yes	*No*	*Yes*	*No*	χ^2	P
Any previous psychiatric contact	11	24	5	36	3.12	0.07
Receiving any medication	6	29	1	40	3.28	0.07

sample, was well within normal limits, 59% of the group producing full scale IQ scores of between 90 and 110.

How do these results compare with earlier studies? Yeudall *et al* (1981) reported that "70% of aggressive psychopaths were found to have abnormal (neuropsychological) profiles indicating dysfunction localised to the frontal and temporal regions of the brain". This 'localisation', however, may reflect the nature of the tests used; indeed, given that most 'higher order' cognitive activity probably takes place in the temporal and frontal lobes, to report the same is to do no more than to report general impairment of function. In general, tests which purport to measure functioning outwith these areas are described and used as measures of gross impairment, rather than in terms of normal distribution statistics. Furthermore, the method of classifying what constituted abnormality was different. We considered group scores, whereas in Yeudall *et al*'s series individual profiles were rated for evidence of abnormality. Unfortunately, neither the criteria used to define that term nor the nature of the control group of normal subjects used in that work were made explicit.

The study by Spellacy (1978) resembles the present work in that it compared groups of violent and non-violent prisoners; he concluded that his subjects "could be classified correctly as violent or non-violent with 95% accuracy by use of (his) neuropsychological battery alone". The principal difference between his violent and non-violent subjects was the significantly lower general intelligence of his violent sample. He reported verbal, performance, and full-scale IQ discrepancies on the WAIS in the region of 12–14 points; these differences were all statistically significant at the one in a thousand level, and are thus among the most discriminatory items in his test battery. However, there was much greater overlap in the IQ distributions of the two groups in the present study, and it must be assumed that the reason for this marked difference in test results is due to sampling differences. In Spellacy's series "the violent group was defined by an arrest record that contained at least two assault charges... and a demonstrated history of unpredictable aggressive behaviour". The assault charges could range from indecent assault to murder, and it is possible that this fairly broad definition biased the violent sample in the direction of minor assaultative violence and poor general intelligence – a bias not so apparent if the violent group is selected using more serious violence as a criterion.

This disagreement is an important one, in that earlier results clearly suggested that brain pathology, as measured by cognitive tests, is related to criminal aggression to a degree which merited further study. In contrast, the present work suggests that general ability may be related to a predisposition to violent behaviour, but to a more modest degree than that indicated by earlier studies, and that considerable overlap exists between violent and non-violent groups. No pattern emerged to implicate either hemisphere in relation to a history of violent behaviour.

Acknowledgements

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Appendix

iolence rating for present offence (abbreviated)

0. Completely non-violent.
1. Minimal violence (e.g. verbally aggressive, shouting or gesturing, even if this was not obviously directed at others).
2. Moderate violence (e.g. attack on a person resulting in no serious injury, fighting or brawling, or damage to property when this was the main intent).
3. Moderately severe violence (e.g. attack which resulted in serious injury but not detention in hospital for more than 24 hours, or damage to property which was extensive or which could have resulted in threat to life.
4. Severe violence (victim died or life and health seriously endangered).

Violence rating scale – overall record

0. Never been convicted of violence – never gets into fights.
1. Some evidence of violence (occasional fights but no convictions).
2. One or two convictions for minor assaults or damage to property.
3. Three or more convictions for violence, but none serious in the sense of '4' below.
4. One or more severely violent episode in which someone's life or health has been seriously endangered.

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Cerebral and Brain Stem Changes After ECT Revealed by Nuclear Magnetic Resonance Imaging

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Nuclear magnetic resonance images of the brain were obtained in fourteen patients with major depression during a course of ECT. The T_1 relaxation time rose immediately after the fit, reaching a maximum 4–6 h later. The T_1 values then returned to their original level; no long-term increase occurred over the course of treatment. These results are consistent with an extensive but temporary breakdown of the blood-brain barrier during ECT.

Nuclear magnetic resonance (NMR) is a long established technique in physics and chemistry, but relatively new in medicine. Nuclei in a constant magnetic field can be made to resonate by the application of an oscillating magnetic field in the radiofrequency range. As the resonance decays to zero the radiofrequency radiation emitted by the nuclei can be detected; this is the basis of the NMR signal. The time taken for the nuclei to return to their original equilibrium position is the spin lattice relaxation time, T_1.

In medical NMR imaging, the signal is usually obtained from hydrogen nuclei (protons) within the body. Since most biological systems consist of 70–90% water, it is these water protons that cause the major component of the NMR signal. Solid tissues have short T_1 values, while relatively fluid tissues have longer ones. The T_1 of grey matter is significantly longer than that of white matter because of their different fat:water ratios. T_1 times can be altered by changes in the ratio of 'free' to 'bound' water in a tissue (Mathur De Vre, 1984) or in its total water content (MacDonald *et al*, 1985). NMR is therefore a useful tool for investigating possible changes in the brain during a course of ECT, particularly as there is no ethical objection to scanning patients repeatedly. Information obtained in this way about changes in the brain induced by ECT may also shed light on the intracerebral changes occurring after spontaneous epiletic fits.

Method

Eleven patients (4 male, 7 female, mean age 49.6 years) had an NMR scan before and after the administration of the first treatment (scans 1 and 2) and again before and after a later treatment (scans 3 and 4) of a course of ECT. A further three patients (all female, mean age 40.3 years) had a scan before and after their last treatment and then six further scans during the 6 h following the treatment. One of these had a final scan after 25 h. Of the total group of fourteen patients, eleven had bilateral and three had unilateral electrode placings; all of them satisfied Research Diagnostic Criteria for a major depressive episode (Spitzer *et al*, 1978). Patients were excluded if there was a history of serious head injury, brain damage, alcohol abuse or previous radiological abnormality. None had received ECT in the previous 6 months. A standard anaesthetic procedure was used: methohexitone (60–100 mg) and suxamethonium (30–80 mg). Four patients also received diazepam (2.5–10 mg i.v.) before scan 2. The treatment was delivered using an Ectron CC–100 machine, except in one case where a Mecta machine was used. The post-ECT scan was performed as soon as possible after the patient had recovered from the effects of the anaesthetic.

An M&D Technology Ltd 0.08 T resistive magnetic resonance imaging system was used. For each scan, calculated T_1 images were obtained in three planes: a transverse section 10 mm above the maximum diameter of the lateral ventricles, a midline sagittal section of the brain stem, and a coronal section through the parietal and temporal lobes. All sections were 12 mm thick, and the pixel size was 2 mm^2. Measurement of mean T_1 of the entire area of the transverse section (the grey and white matter together but excluding the cerebrospinal fluid (CSF)) was made. The area of each hemisphere to be sampled was delineated separately using an irregular region of interest; a cursor was used to draw the boundary of the area to be measured directly onto the image of the section as it was displayed on the monitor. The mean T_1 within the region of interest was calculated and displayed. CSF has a high T_1, and is white on the coloured display. It is therefore possible to exclude sulcal CSF by ensuring that it is not included within the boundary of the area to be sampled. In addition, the computer was programmed to present the mean T_1 of the tissues in this area that were within the range 60–600 ms. This range includes grey and white matter, but not CSF, which has a much higher value. This latter method also ensures that in the small regions of interest sulcal CSF is not included in the samples of grey matter that were analysed. Individual small areas of T_1 in the pons, mid-brain (at the level of the

TABLE I

Change in T_1 of the transverse section

	T_1: ms							
	Scan 1		*Scan 2*		*Scan 3*		*Scan 4*	
	Mean	*s.d.*	*Mean*	*s.d.*	*Mean*	*s.d.*	*Mean*	*s.d.*
Unilateral ($n=3$)	350	7	354	7	347	11	348	10
Bilateral ($n=8$)	341	18	348	15	340	10	346	9
Total ($n=11$)	344*	16	350*	13	342**	10	346**	9

*$P<0.01$; **$P<0.05$; all other comparisons NS (Wilcoxon's paired rank test)

optic chiasma) and posterior corpus callosum (sagittal section); frontal and occipital lobes (transverse section) and parietal and temporal lobes (coronal section) were also measured. T_1 was measured at the mid-point of each area. In each case the sample measured had a volume of 1.41 cm^3, except the mid-brain and posterior corpus callosum (0.42 cm^3).

To control for the effects of the general anaesthetic, five volunteers (three male, two female, mean age 32.6 years) had a transverse section scan before and 30 minutes after an identical anaesthetic procedure.

Results

Patients had a mean of 5.4 treatments given at a mean frequency of 2.4 per week prior to scan 3. As those having unilateral ECT did not show any lateralisation of change in T_1, unilateral and bilateral treatments have been analysed together. There was no significant change in T_1 in any of the areas examined between the beginning and the end of the course of ECT, i.e. when scan 1 was compared with scan 3 and scan 2 with scan 4.

In the transverse section there were significant differences in the mean T_1 of the whole section before and after the individual treatments, i.e. between scans 1 and 2 and between scans 3 and 4 (Table I). The mean change between scans 1 and 2 was 6 ms ($P<0.01$). The rise after bilateral ECT was larger (7.3 ms) than after unilateral ECT (4.0 ms), although this was not significant, probably because of the small numbers in the study. There was a smaller mean increase in T_1 of 4 ms between scans 3 and 4 ($P<0.05$); bilateral ECT produced a larger increase (6.7 ms) than unilateral ECT (1.3 ms), although again this difference was not significant. Patients were scanned sooner after the last ECT (15.8 ± 4 min, range 13–28 mins) than after the first (30.9 ± 16 min, range 15–62 mins), probably because by then they were less depressed and also familiar with the technique ($t=2.69$, d.f. ≐ 19, $P<0.02$); this suggested that the maximum rise in T_1 may not have occurred within 15 min of treatment. This was shown to be so in the three patients scanned six times during the six hours following ECT (Fig. 1). In the patient scanned after 25 h the T_1 time had returned to below its starting value.

The same anaesthetic sequence in the five controls was associated with a non-significant decrease in mean T_1 from 340 ms (s.d. 9 ms) to 336 ms (s.d. 8 ms).

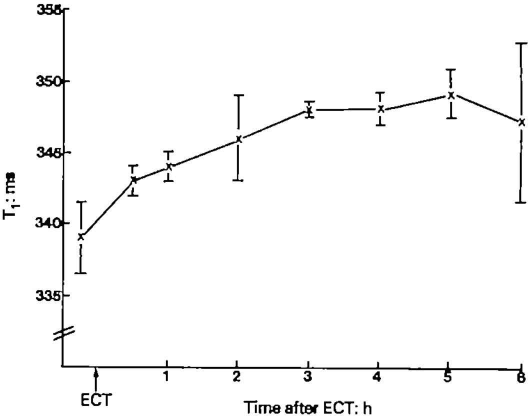

FIG. 1 Change in mean T_1 for the three patients scanned over the six hours following ECT.

Of the nineteen individual small regions in the grey and white matter, T_1 was found to increase in ten between scans 1 and 2; there was a decrease in seven, and no change in the other two. This increase was statistically significant in the frontal and occipital white matter ($P<0.05$) and in the left temporal grey matter ($P<0.01$). For the 8 patients having bilateral ECT only, there was a significantly decreased T_1 in the right occipital grey matter ($P<0.01$). Between scans 3 and 4 the T_1 in eleven regions increased; there was a decrease in six and no change in the other two.

Discussion

The mean T_1 of the transverse section showed a significant increase after treatment, both at the start and end of a course of ECT, reaching a maximum 4–6 h after the fit. This was shortlived, and no cumulative effects of ECT were noted. The increases reported in this study are likely to be underestimates of the changes produced by ECT itself, since anaesthesia alone was associated with a fall in T_1.

Measurement of the 19 small regions described above added little further information. Most of the T_1 values in the transverse and coronal sections (20 of 32) increased following treatment. Because there were 64 comparisons involved, it is likely that some of the significant differences arose by chance.

The brain stem showed a non-significant fall in T_1 in five of the six measurements. This may be due to the anaesthetic having a greater effect in this area, and is especially interesting since the brain stem reticular formation is involved in arousal.

Interpretation of changes in T_1 is difficult. There is no good evidence that ECT causes brain damage (Meldrum *et al*, 1974; Galloway *et al*, 1981), and recently necroscopy could reveal no pathology attributable to ECT in a patient who had received over 1250 treatments (Lippmann *et al*, 1985). Similarly, it is unlikely that these T_1 changes represent inflammation or brain damage, since they are reversed so quickly. It is more likely that they are the result of an increase in the total water content of the brain. It is known that ECT produces a temporary breakdown of the blood-brain barrier (BBB) and that this is probably due to the concomitant increase in blood pressure (Bolwig *et al*, 1977*a*) and cerebral blood flow (Bolwig *et al*, 1977*b*). It is also known that repeated convulsions at short intervals produce cerebral oedema (Laursen *et al*, 1985). It has been shown that macromolecules leak into cerebral tissue during the temporary breakdown of the BBB induced by ECT (Bolwig *et al*, 1977*a*). This would cause an increase in the relative osmotic pressure of the brain. After restoration of the BBB, movement of water from blood vessels into the tissues would therefore occur, and this would provide an explanation for the rise in T_1 over time. It is likely that similar changes occur after spontaneous epileptic fits, although the possibility that the observed changes in T_1 are induced by the electrical stimulus rather then by the subsequent convulsive activity in the brain cannot be excluded.

More work is needed to determine the duration and magnitude of the change in T_1 and to find out if it is related to clinical improvement or the side-effects of treatment.

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In-Patient Treatment for Drug Abuse

A. H. GHODSE, M. LONDON, T. H. BEWLEY and A. V. BHAT

During 1984 there were 253 admissions to the in-patient drug dependence treatment unit at Tooting Bec Hospital. Of the 198 patients responsible for these admissions, 60% were male and 43% were over the age of 30 years. There were 151 patients admitted for opiate detoxification, and 75% completed the withdrawal schedule. However, only 15 out of 25 patients admitted for benzodiazepine withdrawals were found to be physically dependent. Other reasons for admission included stabilisation of the dose of opiate (24%) and the treatment of physical complications of addiction.

In 1965 the report of the second Brain committee recommended, among other measures, that drug dependence clinics should be set up to deal with the rising incidence of opiate dependence in the United Kingdom (Interdepartmental Committee, 1965). It was initially envisaged that in-patient units would form an integral part of treatment, offering detoxification and long term rehabilitation. Only four such in-patient units have so far been established in London and have provided a service for both voluntary agencies and out-patient clinics. The overall emphasis, however, has largely been on out-patient treatment (Ghodse, 1983). It is not clear whether this has been due to a reluctance on the part of addicts, the availability and quality of facilities, or a belief among doctors that out-patient withdrawal is preferable. In recent years there has been an upsurge in admissions to psychiatric hospitals of patients suffering from drug-related disorders. Between 1979 and 1983 the admission rate rose by 76% (DHSS, 1985).

Very little information is available on how the specialist in-patient units function. Most studies have incorporated data from these units with that from out-patient clinics (Love & Gossop, 1985). Studies that have dealt with hospital in-patients alone have been primarily concerned with the natural history of drug dependence.

The overall impression has been that these patients are highly disturbed individuals with a poor prognosis (Willis, 1969; Wiepert *et al*, 1978). Edwards (1979) has noted that the lack of current information on what actually happens to an addict once admitted for treatment has handicapped the discussion of future policies on the treatment of drug dependence in the UK. This study attempts to fill that gap by describing the experience of a major in-patient unit in London.

Method

Data was collected on all patients admitted to the Drug Dependence In-Patient Unit at Tooting Bec Hospital during 1984. This is a 26-bed in-patient unit shared between the Drug Dependence Treatment Clinics of St Thomas' and St George's Hospitals to which patients are admitted for a variety of reasons: some seek detoxification with the aim of leaving the unit drug-free, while others are admitted because of a crisis or a chaotic situation and require stabilisation of their drug usage. The medical staff of both units use a common format when recording the assessment of patients on first contact, which is usually in the out-patient clinic. This information, together with the in-patient records and relevant correspondence from clinicians, social workers and probation officers was analysed.

Results

Age and sex of patients

During 1984 there were 253 admissions to the in-patient unit, representing 198 patients of whom 37 were admitted more than once during the year.

Males constituted 60% (119) of patients. Forty-three per cent of the patients were aged 30 or over and only 8% were younger than 20.

Past history

In 38% of patients there was a history of major disruption in early life, involving death or separation of parents. Delinquency before the age of 15 was present in 51%, and 56% had left school without sitting any exams. For 56% of patients, the last partner with whom they cohabited was also dependent on drugs. For 89% there had been at least one previous criminal conviction, while 28% had six or more convictions. During the six months prior to their first or only admission, 73% of patients were unemployed.

Source of referral

Nearly two-thirds of the admissions (156; 62%) were referred from drug dependence out-patient treatment clinics, just over half of these (84; 33% of admission) from the 'home' clinics of St George's and St Thomas' and the rest (72; 28%) from other clinics that lack an in-patient facility of their own. Other sources of referral were general practitioners (37; 15%), other hospital departments and agencies (24; 9%), and self-referral (27; 11%). The source of referral was not known in 9 cases (4%).

Motivation for admission

Only 114 patients were able to give clear reasons for seeking admission. Of these, interpersonal relationships (parents, spouse, girlfriend or children) accounted for 54% (62 admissions). Physical complications of addiction accounted for 23 admissions (20%), retaining a pregnancy for 9 admissions (8%) and an attempt to obtain or retain employment for 12 admissions (11%). Although legal proceedings were said to be a motivational factor in only 8 cases (7%), 63 patients were in fact facing an impending court-case and the desire for a favourable psychiatric report for the court may have contributed to their decision to enter hospital.

Drugs of abuse and dependence

Table I shows the drugs used regularly in the month prior to admission, and it is evident that use of more than one drug is common. Drugs were obtained from a variety of sources, with 83 admissions (33%) receiving drugs from a drug dependence unit and 27 (11%) from NHS general practitioner and/or a private doctor. Only 35 admissions (14%) denied obtaining drugs illegally on the black market. It was found that 157 (62%) admissions had injected drugs intravenously.

TABLE I

Drugs used regularly in the month prior to admission

Drugs	*Admissions*	
	Number	*Percentage*[1]
Heroin	153	61
Methadone	118	47
Diconal	6	2
Other opiates	24	10
Barbiturates	23	9
Benzodiazepines	59	23
Cocaine	19	8
Amphetamines	26	10
Cannabis	59	23
Hallucinogenes	1	0
Alcohol	62	25

[1] Percentages do not add up to 100% since some individuals used more than one drug

Purpose of admission

Although the majority of patients were admitted for detoxification, 61 admissions (24%) were for the stabilisation of opiate-dependent individuals on a regular dose of methadone. Four drug dependent patients who were also epileptic were admitted for stabilisation on phenobarbitone. Occasionally, other reasons may have prompted or contributed to the admission: 24 admissions (10%) followed an overdose or a self-inflicted wound, while in 11 (4%) there had been a social crisis. In addition, 59 admissions (23%) were found to have infections which required antibiotic treatment. These were mostly skin sepsis arising from non-sterile injection techniques; 31 (12%) were found to have abnormal liver function tests.

In-patient management

On admission, a history was taken of all drugs of abuse and dependence, and a physical examination was carried out. No drugs were administered at first, but the patients were carefully observed and examined at frequent intervals. Only if there was clear, objective evidence of the abstinence syndrome was the appropriate drug administered in moderate dosage, to be repeated when withdrawal signs recurred. Signs of opiate abstinence include dilated pupils, gooseflesh, lacrimation, rhinorrhoea, restlessness and drug-seeking. Signs of barbiturate abstinence include tremor, tachycardia with increased heart-rate on standing, orthostatic hypotension, nausea and vomiting, sweating, irritability and agitation. Over a 24–48 hour period of observation it was thus possible to establish the required daily dose of the drug, which could then be administered regularly in divided doses. Once the patient was stabilised in this way, the dose of drug was gradually reduced according to a standardised regime. If a patient was physically dependent on two substances (usually opiate and sedative), stabilisation on methadone and sedative was followed by sedative withdrawal and then opiate withdrawal; alternatively, the dose of both substances was reduced, stepwise, simultaneously.

All patients admitted for opiate detoxification (151) were found to be physically dependent on opiates, and 112 (74%) successfully completed the detoxification course; of the 39 who failed to do so, 29 left prematurely of their own accord, while the rest were discharged for reasons such as violent behaviour or illicit drug use on the ward. There were 18 patients admitted for barbiturate detoxification and again all were found to be physically dependent; 17 completed detoxification. However, out of the 25 admissions for benzodiazepine withdrawal, only 15 actually required diazepam; 14 of them completed detoxification. Moreover, out of 32 patients admitted for alcoholism, only 19 required treatment; all of these completed the withdrawal schedule. Epileptic seizures were experienced by 4% of admissions prior to stabilisation on sedatives, and by 1.6% during sedative detoxification. None of the patients had fits after completion of their withdrawal schedules.

In addition to the drug regimes, patients were offered a variety of other treatments on the ward. They took part in therapeutic groups or in community meetings designed to deal with day-to-day problems on the ward. Art therapy, relaxation classes and individual counselling were also provided.

Outcome

Once patients had successfully completed their withdrawal schedules, they were encouraged to give careful consideration to their immediate future. Those who were especially vulnerable to relapse were asked to consider entering a residential rehabilitation programme, and social workers helped them to choose an appropriate plan for their future. The choice of entering a therapeutic community was made by 5% of all admissions, and 38% decided to opt for out-patient follow-up in the drug dependence clinic. The decision to discharge themselves against medical advice was made by 46% of admissions, although two-thirds of these had successfully completed their withdrawal schedules. Patients were advised to remain for a minimum of two weeks following detoxification, because it was felt that immediate return to the community held a high risk of relapse, but many were impatient after the weeks spent on a closed ward, and left against medical advice. The disposal of the remaining 12% of admissions varied. Many returned to the care of their general practitioners, while some moved to a different geographical locality.

The duration of the admissions varied widely: 6% of admissions ended within the first 24 hours while 7% lasted longer than three months. A third of all admissions remained in hospital for more than four weeks.

Discussion

Patients admitted to hospital for the treatment of drug dependence are generally those who are too severely dependent for out-patient management. Although estimation of the severity of dependence is a complex issue, one indication of the severity of dependence of these patients was that more than a third of all admissions in 1984 were re-admissions within that 12-month period.

Although 43% of the admissions were aged 30 years or more, a contemporary study of referrals to a London out-patient drug dependence clinic showed that only 35% of patients were in this age group (Love & Gossop, 1985). In the UK as a whole, only 22% of addicts notified to the Home Office in 1984 (and whose age was known) were aged 30 years or more, and 15% were under the age of 20 years. These differences may arise because London, with a longer history of drug abuse, has an older population of addicts than occurs in other parts of the country. Alternatively, or in addition, a possible explanation for the difference may be that clinicians were reluctant to admit younger patients to an environment where they would be in prolonged and close contact with seasoned addicts, or again, younger patients may not perceive in-patient treatment as relevant to themselves.

The purpose that lay behind the majority of admissions was the treatment of opiate dependence. All these patients were found to be physically dependent on opiates, and 74% of those admitted for opiate detoxification completed their withdrawal schedule. For such a disturbed group of patients this is a high success rate and similar to that reported by Gossop *et al* (1986).

The pattern of addiction to sedatives has altered over the years, with the decline in barbiturates being replaced by a rise in benzodiazepine abuse. Indeed, benzodiazepines were identified in the urine of 60% of patients attending a drug dependence out-patient clinic, and 76% of these had obtained their drug illegally (Beary *et al.,* 1987). Altogether, out of 37 patients admitted for sedative (benzodiazepine and/or barbiturate) detoxification, 28 were dependent on opiates. The proportion of patients who gave a history of barbiturate usage and the proportion who were subsequently found to be addicted were roughly the same, whereas only 15 patients (25%) of the 59 who gave a history of regular benzodiazepine use showed signs of physical dependence. Most of those who proceeded on to sedative detoxification completed their schedules, probably because it was unit policy not to allow patients to leave in the middle of a withdrawal schedule because of the risk of fits.

Most drug addicts are users of more than one drug, and alcohol is one of the substances frequently abused; it may then become one of their drugs of dependence. Stimulants are also commonly abused, and it is noteworthy that cocaine was used almost as frequently as amphetamines, probably reflecting the recent rise in its use.

In-patient treatment tended to fall into two stages; the initial period of drug withdrawal, when the patient was mainly preoccupied with his or her symptoms, and the subsequent period of recovery and planning for the future. Owing to the lack of facilities, patients entering the second stage were forced to remain on the same locked ward. Many resented this continued restriction and many of the self-discharges occurred during this phase.

The in-patient unit of the drug dependence clinic provides a valuable service, particularly for those addicts who are the most seriously ill. Of those who entered hospital, a high proportion were successfully stabilised or withdrawn from drugs. However, this should be regarded as only the first step in the rehabilitation of the patient, and this study does not provide information about the long term outcome for the patients concerned. Nevertheless, a re-structuring of the in-patient facilities to mirror more accurately the two-stage nature of the treatment offered may improve the outcome as far as long term rehabilitation is concerned. The recent rise in drug addiction has resulted in more resources becoming available to treatment agencies, but the bulk of these have been allocated to community projects. Equal importance should be attached to improving in-patient facilities, so that a better standard of care can also be offered to the more severely ill and disturbed patients that these units serve.

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The Munchausen Syndrome and its Symbolic Significance:
An In-depth Case Analysis

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The 'gains' of Munchausen syndrome cannot readily be understood on a rational level, and are presumed to be intra-psychic. The presenting fabrications often follow a remarkably similar pattern, and some psychodynamic insight might be gained by analysis of the general themes and contents. The case of one typical patient is reported here, and the aetiology and symbolic significance discussed.

Munchausen's syndrome was first described by Richard Asher in 1951. It is a condition in which patients repeatedly present themselves at different hospitals, often far apart, often using different names, making up colourful histories, and describing various fictitious signs and symptoms in order to gain admission to hospital. Once admitted, they submit themselves to painful and dangerous investigations, operations, and treatment, disappearing only when they are found out. Asher wrote that "The most remarkable feature of the syndrome is the apparent senselessness of it all. Unlike the malingerer who may gain a definite end, these patients often seem to gain nothing except the discomfort of unnecessary investigations and operations". Furthermore, these patients sometimes die as a result of their 'dicing with death' (Enoch & Trethowan, 1967).

Much has been written about these patients' self-destructiveness, and various interpretations have been attempted. Although their manipulation appear to be conscious, the underlying motivations must be unconscious; the gains cannot be understood on a rational level and must be presumed to be intra-psychic. Various interpretations of this behaviour have been made, including a need to suffer and perhaps to die, a desire to be the centre of attention, the adoption of a passive controlling role, the cheating of an authority or parent figure, dependency needs, erotic desires, symbolic castration, hatred of doctors, maternal mishandling of early childhood illness, abnormal care-eliciting behaviour, a desire to obtain shelter, money, or drugs, psychopathy, depression, and a desire for a new life with a new name (Berney 1973, Blackwell 1968; Cramer *et al*, 1971; Enoch & Trethowan, 1979; Henderson 1974; Ireland *et al*, 1967; Menninger 1934; O'Shea *et al*, 1984; Reed 1978; Spiro 1968). Certainly it would appear unlikely that the syndrome is a simple case of hospital addiction, need for board and lodging, attention-seeking behaviour, dependency needs, or abnormal care-eliciting behaviour, as these patients inevitably discharge themselves following discovery, even if offered an alternative. In-depth analysis of cases has therefore proved difficult.

However, these patients' fabrications often follow a curiously similar pattern, and some psychodynamic insight might be obtained by closer examination of their general theme and content. The most common story, usually accompanying an overdose or some other variant of self-destructive behaviour, is of a feigned and dramatic bereavement. These stories often bear a striking resemblance to tragic opera, and indeed, these patients frequently exhibit a preoccupation with the operatic world, e.g. giving their occupation or that of the deceased relative as 'opera-singer', or claiming some other connection with the opera or the ballet. We have recently seen a Munchausen patient who gave this typical story and who, following discharge, corresponded briefly with the hospital chaplain. The theme of these communications was primarily the opera and the ballet. It seemed interesting to examine these leads more closely, in an attempt to shed light on this fascinating condition.

Case Report

Paul K, a male aged 46, was admitted following a paracetamol overdose, which he claimed had been precipitated by the death of his wife and only daughter in a road traffic accident in Tunisia, three weeks earlier. He described his shock when the news of the death was broken to him, and also told of the horror of having to examine the bodies in the mortuary. He expressed gratitude at being able to tell the story to the doctors and nurses, and said that he recognised his need to speak to people in this way. His past history included a story of a massacre on his father's plantation in Kenya, in which two servants were decapitated.

On examination, he was anxious and shaky, but was described by the nursing staff as being very pleasant. However, when attempts were made to contact the patient's reported general practitioner and place of work, it was discovered that neither of these had ever heard of him. He discharged himself before this could be discussed with him, but left a letter with the hospital chaplain, together with a list of what he felt were the significant events in his presumably real life; he thanked the chaplain for his care, and apologised for any trouble that he had given. In his view, the answer to his conundrum is not known by any medical or psychiatric mind, but that one person who has the answer is a 19th century Italian composer, Gaetano Donizetti in *L'Elisir d'Amour* (*Elixir Of Love*).

The list of attitudes and events suggested that he had never received or given love, but that ambition drove him to conform to social norms and to marry and have children until, at the age of 42, a life crisis involving guilt caused him to sacrifice his ambition. Three years later, he described his own 'death', by which he presumably meant a symbolic and emotional death resulting from, or possibly causing, the losses of his wife's companionship, his own drive, and his ambitions for his children. He concluded that love and grief were absent, and that this resulted in guilt.

A few days later, the chaplain received another letter from the patient, from Manchester, stating that he had not found any answer in *L'Elisir d'Amour* after all, but that he was going to try the ballet *Romeo and Juliet* instead. Two days later, another letter arrived, saying that *Romeo and Juliet* had also proved negative, but that he was going to Paris to try *Tristan and Isolde*; this story, like *Der Fliegender Holländer* (The Flying Dutchman) and *Götterdämmerung* (Twilight of the Gods), was based on Wagner's Theory of *Liebestod* or 'love though death'. The last card arrived three days later, and stated that Paris had also been negative, but that he was moving on to Munich to try Richard Strauss's opera *Tod und Verklärung* (*Death and Transfiguration*).

The operatic themes

To understand the patient's meaning and solve the riddle, it is necessary to know something about the operas and ballet mentioned by him, though the story of *Romeo and Juliet* requires no explanation.

L'Elisir d'Amour is a comic opera written by Donizetti in 1832. Nemorino drinks a love-potion in order to win Adina, but in the meantime, accepts the hand of another. The news then spreads that Nemorino's uncle has died and left him a legacy, and in consequence, he is besieged by the attentions of all the girls in the village. Adina also becomes conscious of her love for him, and after some misunderstanding, the lovers are united.

The next group of operas are all by Wagner: *Tristan and Isolde, The Flying Dutchman*, and *Götterdämmerung*. The story of *Tristan and Isolde* opens on board a ship, where Isolde, who is being taken to Cornwall against her will to marry King Mark, resolves that she and her military escort, Tristan should die. She tricks him into drinking a death-potion, which she shares with him, but unknown to them both, a love-potion has been substituted instead. In the face of certain death, they become conscious of their mutual love, and are able to confess it. However, due to the substition of the potion, they do not die, and arrive safely in Cornwall, where the frustrated lovers long for death, since only through that can they be united and their love realised. When discovered in an embrace with Isolde, Tristan fails to defend himself, and is badly wounded, dying in Isolde's arms, and Isolde's *Liebestod* starts. This is the longing and readiness for death, combined with love-ecstacy, which results in Isolde's transfiguration and death, as she sinks to the ground over Tristan's body.

The Flying Dutchman contains the same theme of redemption through love and death. The Dutch captain was condemned to sail the seas forever, unless redeemed by the love of a woman who will be faithful to him unto death. He meets Senta and becomes betrothed, but due to a misunderstanding, believes that she has been unfaithful to him, and orders his ship to set sail. Senta, determined to save him, throws herself into the sea, crying "See, I am faithful to you unto death", and at the same moment, the ship sinks, and he and Senta are seen rising into the heavens, clasped in an embrace.

The third opera is *Götterdämmerung*, the tragedy which culminates the *Ring Cycle*. The Gods and the World are doomed through greed and corruption, and can only be saved by the return of the Rhinegold, now made into a ring, to the Rhine-maidens. Siegfried who possesses the ring, is betrothed to Brünnhilde, a Valkyrie.

However, he is given a magic potion, which causes him to forget Brünnhilde, and ask for the hand of Gutrune; Brünhilde therefore conspires to kill Siegfried, and reveals that he is only vulnerable to attack in the back. He is consequently murdered while two ravens, the symbols of transformation, circle above his head. His dying words are of Brünnhilde's awakening, and he calls out that he is wakening her again and that this time her eyes are open forever, implying an eventual rebirth following her impending death. The implication of life after death is continued when an attempt is made to tear the ring from Siegfried's finger, and the dead man raises his hand threateningly.

This leads to the theme of redemption. Brünnhilde knows that redemption can only be achieved by the return of the ring to the Rhine-maidens, and resolves to do this. She commands that she and Siegfried should be burnt on a pyre, and takes the ring. The flames engulf them, and at the same time, the Rhine overflows its banks and surges forward with the Rhine-maidens on its waves, so that the ring is returned to them. The fires are seen to devour Valhalla, thus allowing the rebirth of a more hopeful phase.

The last work has the telling name *Death And Transfiguration*. This tone poem depicts the bodily dissolution of a hero who in his last moments sees the happiest events of his life pass before his mind's eye. A poem by Alexander Ritter was later published with the score, describing the *'lamento e trionfo'* of the hero's death. The main theme has been described as "not resignation but contented leave taking".

Discussion

The basic theme underlying all these stories, most of which are derived from ancient mythology, seems to be that of transformation, redemption, and the triumph of love, achieved either through the voluntary embracing of death or imbibing magical potions. This theme is one that recurs time and again in the traditions, religions, myths, and legends of many different cultures, from the primitive sacrifices of Stone-Age man, to Phoenix arising from the ashes, through to Christ redeeming us by his death and subsequent reincarnation.

Robert Donnington, in his book *Wagner's Ring And Its Symbols,* writes: "These outward deaths are often depicted literally in dreams, fantasies, myths and legends, but their true meaning is almost certainly inward pain and death". Wagner himself wrote: "The period in which I have worked in response to my intuition starts with *The Flying Dutchman* and if in these works there is any underlying poetic theme, it must be looked for in the supreme tragedy of renunciation, the abnegation of the will, which is shown there as necessary and unavoidable, and alone capable of achieving redemption". Donnington understands redemption to be a synonym for transformation—"None of Wagner's images in *The Ring* actually point to annihilation as our own Western thoughts conceive it. One after another of them points to rebirth in the sense of transformation"; this would appear to be true in the suicide or voluntary sacrifice of life of Tristan and Isolde, Senta and the Dutchman, and Siegfried and Brünnhilde, in each of the three Wagnerian operas referred to, as well as that of Romeo and Juliet in the ballet mentioned by the patient.

It is interesting to attempt to relate some of this to what we know of Munchausen patients. Enoch & Trethowan (1979) said of Munchausen syndrome that "the disorder is characterised by a relentless tendency towards self-destruction", and it would seem to be this which chiefly distinguishes the syndrome from malingering and hysteria. Time and again, these patients put themselves into situations in which they suffer. One can only speculate about the psychodynamic mechanisms, but it is tempting to attempt to explain the relentless masochistic self-punitiveness and 'dicing with death' as an unconscious attempt to appease the gods, prove overwhelming remorse, and obtain absolution and redemption by killing the old self or a part of it, to allow transformation from despised self to ego-ideal, and hence the attainment and inspiration of elusive love and perfect happiness. Menninger (1934) described certain patients in whom "the capacity for dealing with reality is so poorly developed, that they proceed as if they could actually kill themselves and not die". Pseudologia phantastica may be yet another form of this calculated risk-taking, and in addition the difference between reality and the rather grand abilities, qualities, and qualifications which these patients claim for themselves probably also reflects the discrepancy between self-image and ego-ideal and the need to inspire admiration. It may also satisfy the need, arising from this poor self-image, to deceive 'intelligent' professionals.

Snowden *et al* (1978) said of their Munchausen patients: "In some cases it seemed as though the patients were seeking a new life with a new name to get away from themselves and their problems", and Deutsch (1955) that "In one act he searches for a new identity and denies his own real identity". Such an instinctive and powerful belief in atonement and salvation may well spring from the forces of an ancient and deeply-rooted collective unconscious which, over the ages, has given rise to a variety of myths and is now surfacing again and being acted out in a primitive and concrete way. Indeed, there would seem to be too many similarities in the content of the Munchausen stories for this to be purely coincidental. The feigned death of spouse or family may also have as its source, as in so many of these operas, the need for the symbolic sacrifice and death of both partners before unity and love can be achieved.

Cramer *et al* (1971) wrote of Munchausen patients: "Their pathomimicry may be a last ditch attempt at warding off further disintegration". Ireland *et al* (1967) noted that the self-destructive behaviour may "serve to allay intolerable anxiety", and "focal self-destruction may represent atonement for an overwhelming sense of guilt." Menninger (1934) speculated that "Guilt prevents them from accepting love", and also suggested that the behaviour may be "an anti-suicide, a last-minute attempt at saving the whole by sacrificing a part". There is also an element of passive suicide, in that the actual responsibility for the destructiveness is relinquished to the surgeon; it may be this which make it necessary to use the hospital as a stage. Menninger compares this to an animal with its foot caught in a trap, who gnaws off his own leg to get away. Sometimes a more direct attempt of annihilation is made: "The Munchausen patient, at a time of unusual stress, resorts to surrender of the whole in the form of attempted suicide" (Ireland *et al*, 1967). Justus *et al* (1980) carried out an MMPI on a Munchausen patient which concluded: "There are also indications that he is harbouring a great deal of angry affect over which he may be experiencing considerable guilt. As such it may be turned inwards in the form of depressed affect and/or self punitiveness". Guilt would certainly seem to be prominent in our patient, who himself concluded "Guilt positive" at the end of his list of life-events.

When a diagnosis of Munchausen syndrome is made, there is almost always some implication or allegation of anti-social behaviour, but when viewed from this wider perspective, ego-weakness turns to ego-strength in a transformation every bit as dramatic and magical as the ones already described. The accompanying deceit, which stimulates so much contempt and anger, then appears not as a primary goal, but as an incidental by-product of a powerful instinctive drive towards intra-psychic gains.

It is difficult to assess how much of the Munchausen behaviour is conscious or unconscious, since much depends on the observer's point of view, and the syndrome probably lies on the borderline between the two. However, the importance of making this distinction is uncertain. In general, we tend to equate 'voluntary' with 'conscious', but in our everyday life, all of our 'voluntary' actions are to some extent influenced by unconscious factors. We are also accustomed to assume that unconscious falsifications of illness indicates psychopathology, while the possession of insight into that falsification indicates a voluntary element, and therefore psychopathy. It is possible that this falsification may be partly conscious but not wholly under the 'patient's' control, and that it indicates neither psychopathy nor necessarily psychopathology. Spiro (1968) has suggested of the Munchausen patient that "he knows when he is acting, but even with this knowledge he can not stop acting". He likens these patients to alcoholics and drug addicts: "Seemingly wilful acts are determined by unconscious factors and environmental ones to produce a psychiatric illness of profound dimensions". On the other hand, Szasz (1961) refers to "the logical dilemma of the existence of an alleged entity called mental illness which even when deliberately counterfeited is still mental illness". The problem lies largely in the fact that what we call illness is to a great extent selected by society and doctors.

The role of a sick person in society is also relevant. It has been said that everyone exaggerates or feigns ill-health at times, for a variety of reasons, including the avoidance of minor responsibility, denial of failure, and to evoke caring within our family and social network. Parsons (1951) has pointed out that once a person's illness has been medically sanctioned, that person occupies a special role, and is relieved of the usual demands and obligations made on him, while Mechanic (1962) has shown that medical facilities are more likely to be used by persons highly stressed in terms of loneliness and nervousness. The doctor's role complements this, in that he assumes responsibility for the patient's illness and cure (which may include responsibility for the intra-psychic cure, in the form of surgery), and provides a relatively easy way for the patient to communicate and temporarily relieve his sense of isolation. Traditionally in a doctor-patient relationship, the patient has needs and the doctor offers services which go beyond technical medicine.

It is also likely that the aetiology of Munchausen syndrome is multi-factorial, and that no single psychopathological factor accounts for all cases. However, it may be appropriate to view the behaviour in some cases as adaptive, rather than maladaptive in a psychodynamic sense, and it would be interesting then to interpret the patient's behaviour to them in these terms.

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Anorexia Nervosa in Males
A Comparison with Female Patients

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The demographic features, clinical picture, antecedent events and family background in 13 male patients with anorexia nervosa are compared with those in a control group of 39 adolescent female anorectics; the general picture of the illness in males is similar to that in females. Focusing on a group of males, usually less vulnerable to anorexia nervosa, can highlight possible predisposing factors.

Anorexia nervosa (AN) is predominantly an illness of girls and young women, but it is estimated that 5–10% of all cases occur in males (Hay & Leonard, 1979; Andersen & Mickalide, 1983; Crisp & Burns, 1983). It is of value to present a series of male cases for two reasons. Firstly, males presenting with AN are still relatively rare, and most published accounts consist either of individual case reports or of small series of patients. Further investigation of their features is necessary, particularly in comparison with typical female patients. A more important reason, though, concerns aetiological factors: the rarity of the illness in males may indicate that they have a degree of protection from the condition, and it could be argued that predisposing or precipitating factors will therefore be more prominent in those males who do actually develop it. While the possibility of there being qualitative sex-determined differences in aetiology between males and females with AN cannot be ruled out, from the clinical and diagnostic point of view the illness in the male is generally seen to resemble that in the female (Beumont *et al*, 1972; Vandereycken & Van den Broucke, 1984). If this can be confirmed, it seems reasonable to assume that the same major aetiological factors also operate in both sexes.

The investigation of a series of male patients provides an opportunity to identify such factors. This paper presents the features of 13 male patients with AN, and compares them with a series of 39 typical adolescent female anorectics, studied in a similar way.

Method

By a manual search and with the help of the Oxford Record Linkage Study, an attempt was made to identify all male patients seen since 1967 in the Oxford psychiatric hospitals who had received a diagnosis of AN. Their case-notes were then scrutinised, and if the criteria for a diagnosis of AN were met, relevant details were recorded. The diagnostic criteria were modified from those of Feighner *et al* (1972) and were

(a) a distorted implacable attitude to food, eating or weight
(b) weight loss of at least 25% of original weight, or the attainment of a weight 25% or more below that expected. Expected weight was calculated from standard actuarial tables (Geigy, 1962) for patients aged 15 and over, and from height and weight standard charts (Tanner & Whitehouse, 1975) for younger patients
(c) the absence of any medical or other primary psychiatric disorder which might account for the anorexia or weight loss
(d) the presence of at least one of the following: loss of sexual interest, lanugo, bradycardia (sustained at ⩽60/min), overactivity, bulimia, vomiting.

Findings for these patients are compared, where relevant, with those for 39 females in a series of adolescent patients presented in more detail elsewhere (Margo, 1985, 1987).

Results

Demographic features

Between 1967 and 1983, 13 male patients fulfilled the diagnostic criteria. All were Causasians. Because they came from a number of settings, it was difficult to ascertain what proportion of the total number of AN cases they represented. However, the three adolescent patients seen up to 1979 represented 7% of all adolescent anorectic patients seen at the Warneford Hospital and Highfield Adolescent Unit in this period and diagnosed according to the same criteria.

Twelve of the 13 patients were referred in the second half of the period studied (1976–83). Age of onset of illness ranged from 13.8 to 35.1 years, with a mean of 20.0 years. Seven patients were aged under 18 at onset; three were 18–25, and three were over 25 years. Eleven patients had never married; two were married, one with no children and the other with three.

Five patients came from social classes I and II (Registrar-General's classification; Office of Population Censuses and Surveys (1985)) and eight from social classes III, IV and V; this was not significantly different from the expected distribution for Oxfordshire. Not only did this group of patients fail to manifest the usual upper social class predominance of AN, but the males showed a statistically significant relative preponderance of lower social class patients compared with the female group ($\chi^2 = 2.71$, d.f. = 1, one tailed $P = 0.05$).

Clinical features

Weight losses of up to 50.4% of original weight were recorded (mean 33.7%) and weights were up to 47.5% less than expected (mean 35.4%). In 3 patients lanugo was recorded as a clinical feature; loss of sexual interest was noted in 6, bradycardia in 7, overactivity in 8, bulimia in 3, and vomiting in 4.

The only statistically significant difference in the occurrence of clinical features in males and females was the notably more common presence of overactivity in males (62% against 26%; $\chi^2 = 4.08$, d.f. = 1, two tailed, $P < 0.05$).

Of the nine patients whose heights were recorded, six were under the 50th percentile (Tanner & Whitehouse, 1975). Four of these were under the tenth percentile (binomial test, $P = 0.008$), including two whose heights were less than the third percentile. This is in contrast to the female group, in which there was a significant excess of tall girls (over the 90th percentile).

The time from onset of illness to referral to a specialist agency was variable – from 1 to 57 months. Although the proportion of cases referred to a specialist agency within six months of onset was similar to that for girls (50% and 57% respectively), the average delay before referral was far longer for males – 12.5 months, compared with 7.4 months. This was because when males did have a delay of over six months before referral, it tended to be a long one – over a year in two-thirds of these cases.

Antecedent events

In seven patients it was possible to identify major life events in the year preceding onset which were thought to be of relevance to the development of the illness. Examples were loss or threat of loss of friendships, illness of a parent, or disruptions in family relationships caused by events such as an unexpected pregnancy in the mother or the relationship between father and sister leading to the latter being placed in care. In addition, three patients complained of being teased because of their size (in one case this was small stature and lack of physical development rather than obesity) and two patients were reported to have suffered a 'flu-like illness shortly before the onset of AN. These findings are similar to those in the female group.

Premorbid obesity was defined as a weight at some time before the onset of the illness that was 25% or more above that expected for the patient's age and height, or (if height was not known) a weight over the 97th percentile for his age: it was a feature in four patients. This was much higher than the 10% in the female group, but the difference did not reach statistical significance.

Family background

Age of parents. Information was available for 12 sets of parents. Ages of mothers at birth of their anorectic children ranged from 20 to 39, with a mean of 28.3 years. Those of fathers varied from 25 to 40, with a mean of 31.3 years. The mean difference in parental ages was 3.1 years. Unlike the mothers of the female patients, the parents of this group were not older than expected, according to the available national statistics.

Family composition. Unlike the female group, these patients come from larger than expected families, the mean number of children being 3.25, against an estimated expected number of 2.4 (Farid, 1974). Only one was an only child, while five came from families of four or more children. The female: male ratio among the siblings of the patients was 12:17. As in the female group, anorectics did not appear to occupy a particular place in the birth order.

Family history of mental illness. A positive history of mental illness (defined as the identification of a condition for which there was evidence that some form of active management had taken place) was present in a parent or sibling of five patients (38.5%). If grandparents and parental siblings were included, a positive history was found in 14 relatives of six patients (46.2%). The corresponding percentages in the female group were 23.1% and 30.8% respectively. In all cases in which a diagnosis was available, the illness seemed affective in nature.

Abnormalities in the family setting. Such abnormalities (as defined in Margo, 1987) were noted in five patients. These included problems such as chronic parental marital difficulties, the death of a parent and problems with a stepmother and stepsiblings, and chronic mental illness or physical handicap in a parent. In addition, one patient had a mother who had become a persistent dieter before the onset of his own illness. These results are similar to those found in the female group.

Discussion

As in the female group, there was an increase in the referral rate from the mid-1970s, although whether this reflects an actual increase in the prevalence of AN remains unclear (Margo, 1985). Bruch (1974) believed that AN in males was pre-pubertal in onset, but this has been refuted by a number of studies which have shown that, as in females, the condition in males begins mainly in adolescence (Beumont *et al*, 1972; Dally & Gomez, 1979; Crisp & Burns, 1983; Vandereycken & Van den Broucke, 1984). The latter was confirmed in this study, although three patients were over 25 at onset. The male patients in this series failed to manifest the usual upper social class predominance seen in female patients; this finding does not accord with a recent comparison of male and female patients (Burns & Crisp, 1984), and its validity is uncertain.

Of the clinical features, overactivity was significantly more often recorded in the male group than in the female control group, and appears to be a common symptom in males with AN (Beumont *et al*, 1972; Taipale *et al*, 1972; Bruch, 1974; Hogan *et al*, 1974; Sheppard *et al*, 1984; Ziesat & Ferguson, 1984). This may be because physical activity and exercise are more culturally acceptable methods of losing weight for males than females. The incidence of loss of sexual interest in this series was relatively low, compared with that in other studies (Beumont *et al*, 1972; Dally & Gomez, 1979; Crisp & Burns, 1983). A major reason for this is that in five of the 13 patients, information on this point was not available in the case-notes. Clinicians ought not to be shy of pursuing questions about sexual interest, particularly as patients often welcome or are indifferent to their loss of libido while ill, and may not volunteer it as a symptom (Crisp & Burns, 1983).

It was notable that in four of the nine cases in whom heights were recorded these were under the tenth percentile; this contrasts with the excess of very tall girls in the female group. Reports of heights in other series of male anorectic patients are inconsistent: although heights were unremarkable in some series (Dally & Gomez, 1979), Bruch (1974) reported that three of her six primary cases were of very short stature. A number of case reports suggest that AN stunts growth, particularly in males (Toms & Crisp, 1972; Root & Powers, 1983) – i.e. that short stature in anorectic patients ought to be seen as secondary to the illness. Although this is sometimes true, it does not appear to be so in all cases and could not be shown to apply in any of those reported here; in three of the four short patients the stature clearly antedated the onset of the illness. One of the patients dated the beginning of his preoccupation with his size and appearance to being teased by his peers about his small stature. While females in our culture are under pressure to control their size and be slim, males prefer to be larger and heavier (Hsu, 1983). Thus the small male, like the large or very tall female, risks becoming preoccupied about his size and deviation from what he considers a desirable physique. This dissatisfaction with appearance may lead to psychological conflicts or low self-esteem being focused on the body, and may in turn be a risk factor for the development of AN, control over the body becoming confused with control over wider issues.

The long delay before referral to specialist services in those patients in whom the illness was not recognised early is a matter of some concern: long duration of illness is associated with poor prognosis, both in males specifically (Burns & Crisp, 1984) and in AN generally (Morgan *et al*, 1983). Resistance to treatment among male patients (Crisp & Toms, 1972) and the lesser degree of public and medical alertness to the condition in males are probably the major causes of this problem.

As in females, significant life events appeared to be common antecedents of the illness. Premorbid obesity was more common than in the female group; a similar high incidence was reported in a number of other studies of males, although definitions of obesity vary (Falstein *et al*, 1956; Beumont *et al*, 1972; Hogan *et al*, 1974; Anderson & Mickalide, 1983).

Unlike those of the females, the mothers of the male patients were not older than expected. However, relationship and other difficulties within the family were shown to be common here, as in the female group. Many studies of males with AN have commented on the high prevalence of such difficulties, e.g. severe parental marital disharmony (Taipale *et al*, 1972; Sreenivasan, 1978; Dally & Gomez, 1979; Hay & Leonard, 1979; Sheppard *et al*, 1984). A history of psychiatric illness in the families was frequent, affecting almost half the patients: since this is a case-notes study, the true figure may be even higher. The positive family history was on the mother's side in five cases and on the father's once; when parents were involved, four were mothers and one a father. A similar pattern has been reported elsewhere (Crisp & Toms, 1972; Dally & Gomez, 1979). Psychiatric illness in the family – particularly in a parent – has a potential pathogenic effect over and above the purely genetic, such as by disrupting family relationships or causing episodes of parental separation.

From this series, the impression that AN in males is essentially similar to the condition in females is largely confirmed. The second object of the study was to examine whether factors of aetiological significance might be more clearly highlighted in a male group of patients. Possible factors would be those prominent in the female group and occurring at least as commonly, and probably notably more commonly in the male group. According to this argument the aetiological significance of, for example, the finding that anorectic females have older mothers (Margo, 1987) is called into question by the failure to confirm this finding in the male sample. On the other hand, features such as significant life events and abnormalities in the family are found consistently in the backgrounds of many anorectic patients, and were prominent again here. However, such factors as life events may often act as precipitants, so that the underlying causes of vulnerability need to be sought elsewhere. AN is unlikely to have one single or necessary aetiological factor, but instead would seem to be predisposed to by a number of vulnerability factors, whose relative importance cannot yet be determined. This study does point to two, in addition to those already mentioned – a family history of psychiatric illness, and a deviation from the perceived 'normal' body shape.

The importance of a family history of psychiatric illness emerged strongly in the male patients: nearly half had such a history, predominantly of the affective type. This supports the view that there may be some relationship between AN and affective disorder (Cantwell *et al*, 1977; Winokur *et al*, 1980; Hudson *et al*, 1983), although the nature of the relationship is complex and needs further elucidation (Hsu, 1983). If the inherited or environmental effects of such a family history contribute to a general psychological vulnerability, the question still to be answered is why such a vulnerability should under stress manifest itself as AN. It is possible that anything preoccupying a vulnerable individual with dissatisfaction over bodily appearance will in time lead to the body becoming the focus for all his or her emotional conflicts, and predispose to the development of AN: premorbid obesity appears to be a not uncommon feature in male anorectics, and often serves as such a factor. The possible role of short stature in males (or large stature in females) in this regard has already been discussed.

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Specific Cross-Gender Behaviour in Boyhood and Later Homosexual Orientation[1]

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Data from a group of males aged 13 to 23, who as children exhibited extensive cross-gender behaviour, was analysed. In boyhood they frequently played with dress-up dolls, role-played as females, dressed in girls' clothes, stated the wish to be girls, primarily had girls as friends, and avoided rough-and-tumble play. The majority of the group evolved a bisexual or homosexual orientation; two types of behaviour, boyhood doll play and female role-playing, were found to be associated with later homosexual orientation. The findings suggest developmental associations between specific types of boyhood cross-gender behaviour and the objects of later sexual arousal.

When interviewed as adults, male homosexuals recall patterns of boyhood feminine behaviour more often than do male heterosexuals. They commonly report, as children, having: role-played as females, worn girls' or women's clothes, avoided male playmates, avoided conventional rough-house boyhood games, and wanted to be girls (Harry, 1982; Bell *et al*, 1981; Whitam, 1977; Saghir & Robins, 1973). These investigators suggested that such cross-gender behaviours are likely to endure in the form of adult homosexual orientation, but their studies used retrospective data, which are likely to involve errors of selective recall or reconstruction as subjects report their pasts. By contrast, two reports have utilised follow-up data on previously 'feminine, boys. In one, interviews with 16 adult patients described in medical records made during childhood as showing 'feminine' behaviour revealed two to be homosexual, three to be transsexual, and one a transvestite (Lebovitz, 1972). However, the extent to which these adults were representative of all males previously described as 'feminine' in those records is not clear. In the other report, a group of 55 males seen earlier for 'feminine' behaviour in boyhood or early adolescence was re-evaluated; about two-thirds of the group was reported to be homosexual (Zuger, 1984), but it is not clear how many who are currently homosexual were pre-adolescent when initially evaluated, and there is no report of how many of the 'homosexuals' are exclusively homosexual, predominantly homosexual, or bisexual. Most importantly, from the standpoint of the present study, there is no report of the association between specific features of boyhood 'femininity' and later sexual orientation.

The findings presented here examine whether specific boyhood gender-typed behaviours are related to later sexual orientation. We assessed a group of adolescent and young adult males (aged 13 to 23) who had displayed cross-gender behaviour in boyhood; each boy had been previously evaluated before the age of 12.

Sample

Our data derive from a clinical group of 66 families evaluated at the UCLA Gender Identity Clinic between 1969 and 1974. Families were evaluated after parents presented with the complaint that their sons were 'feminine', and were included in the group if the boys displayed extensive cross-gender behaviour. Most of the boys, more than occasionally, stated their wish to be girls or to grow up to become women. They avoided the traditional sex-typed activities of boyhood (such as rough-and-tumble play and sports), preferentially role-played as females, and preferred the clothes, toys, companionship, and games of girls (Green, 1974, 1976). They were anatomically normal (not intersexed). A comparison group of 56 families was solicited by newspaper advertisement, and selected by demographically matching each family to a family with a 'feminine' boy. Families were matched by age of son, sex and age of siblings, and race, religion, education, and marital status of parents. Fifteen years after the collection of initial interview and questionnaire data began, follow-up data on sexual orientation have been collected on about two-thirds of the boys (previously 'feminine' boys $n=44$; comparison boys $n=35$). Contact was lost with or terminated by about one-third of the families.

Method

Following Kinsey *et al* (1948), sexual orientation was rated on a seven-point scale ranging from exclusive heterosexuality (0) to exclusive homosexuality (6). On this scale, 1 is incidental homosexuality, but nearly all heterosexuality.

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[1] An earlier version of this paper was presented at the annual meeting of the American Sociological Association in San Antonio, Texas, August 1984.

TABLE I

Items related to boys' atypical sex-typed behaviour

Category	*Variable name*	*Wording*	*Typical scoring*	*Whether from interview (I) or questionnaire (Q)*
Doll/role play	ROLEPLAY	Does he play "house" or "mother-father" games? What role does he take?	1 = always male, never female 6 = always female, never male	I
	PLAYHS	When playing house my son usually assumes the role of . . .	1 = father, brother, himself, or other male 3 = mother, sister, or other female	Q
	DOLLPLAY	What are his favourite (next favourite) toys?	1 = no dolls, but trucks, etc. 6 = constant play dolls)	I
	DOLPLAY	My son plays or has played with dolls . . .	1 = never 4 = favourite toys	Q
Cross-dressing	XDRESS	I have observed my son or have received reports of my son wearing an article of feminine clothing (including jewellery or makeup) . . .	1 = never 3 = frequently	Q
	NOXDRS	I have observed my son or have received reports of my son dressing up in feminine clothing the following number of times:	Score = actual number	Q
	IMPROVS	My son has improvised (designed from other materials) the following feminine clothing:	0 = none 1 = one type 2 = two or more types	Q
	INTEREST	Does he seem to show any unusual interest or preoccupation with types of cloth or textures? (To mother): Does he comment on your clothing?	1 = no interest in cloth or clothes 6 = constantly comments on and touches materials, calls mother's clothes pretty	I
	COMMENT	My son comments on his mother's clothing styles. . .	1 = never 3 = frequently	Q
Female peers	FRIENDS	Who is his best (next best) friend? Does he seem to prefer boys or girls as playmates?	1 = all male friends 5 = all female	I
	RELATE	My son relates best to . . .	1 = boys or adult males 2 = girls or adult females	Q
Rough play	SPORTS	My son's participation in sports is best described as . . .	1 = more than average 3 = less than average	Q
	RUFHS	My son's participation in the rough-and-tumble of boyhood is best described as . . .	1 = more than average 3 = less than average	Q
Be girl	BEGIRL	My son has stated his wish to be a girl . . .	1 = never 3 = frequently	Q

TABLE II

Zero-order correlations among items related to boys' atypical sex-typed behaviour[1]

PLAYHS	DOLLPLAY	DOLPLAY	XDRESS	NOXDRES	IMPROVS	INTEREST	COMMENT	FRIENDS	RELATE	SPORTS	RUFHS	BEGIRL	
0.47***	0.24**	0.20*	–	0.32*	–	–	–	–	–	–	–		ROLEPLAY
	–	0.25**	–	0.36***	0.19*	0.34***	0.23**	–	–	–	–	–	PLAYHS
		0.45***	–	–	–	0.20*	–	–	–	–	–	–	DOLLPLAY
			–	–	0.20*	0.25**	0.26**	–	–	–	–	–	DOLPLAY
				0.58***	0.38***	–	0.25**	–	–	–	–	0.28**	XDRESS
					0.33***	0.29**	0.27**	–	–	–	–	0.24**	NOXDRES
						–	0.39***	–	–	0.23**	–	–	IMPROVS
							0.46***	0.21*	–	–	–	–	INTEREST
								0.24**	–	–	–	–	COMMENT
									0.63***	–	–	0.22*	FRIENDS
										–	–	0.24**	RELATE
											0.48***	0.17*	SPORTS
												0.28**	RUFHS

1. Only correlations with $P<0.10$ are presented.
*$P<0.10$; **$P<0.05$; ***$P<0.01$

From 2 upwards there is more than incidental homosexuality, or bisexuality. Subjects were rated both for fantasy and interpersonal behaviour. Since the majority of our data were on boys in their teens (median age = 18), many of the boys were still sexually inactive with respect to interpersonal genital sexual behaviour. There are thus more ratings of sexual fantasy than of sexual behaviour.

Sexual fantasy ratings were derived by one of us from interviews discussing masturbation fantasies, nocturnal erotic dream imagery, and genital responsivity to pornography. The percentages of masturbation and dream fantasy content involving male or female partners, and the proportion of times erections would occur to homosexual or heterosexual pornography, were converted into appropriate placement on the seven-point scale (increments of approximately 14% for same-sex partners elevate the fantasy rating by 1 point).

Sexual behaviour ratings were derived from interview data regarding post-pubertal interpersonal genital experiences. Here, at least one same-sex experience results in a score above 0. As with the fantasy ratings, the proportion of experiences with male or female partners was converted into appropriate placement on the seven-point continuum (Green, 1985, 1987).

Data on boys' cross-gender behaviour, or 'femininity', derived from two identical questionnaires, designed and administered by one of us to each parent, and from three semi-structured audiotape recorded interviews (one with the couple one with each parent separately) by one of us that were transcribed and blind-coded. Questionnaires and interviews were administered prior to any therapy that a child or family may have had and that might have modified behaviour. Some interview items were asked again in different form on the questionnaires. Table I lists the 14 items rated; they fall roughly into five categories of cross-gender behaviour. Items are grouped here partially on the basis of content and partially on the basis of their inter-correlations.

Results

Regarding sexual fantasy, of the 44 males who were previously 'feminine' boys, eight had a Kinsey rating of 6 (exclusively homosexual), eleven had a rating of 5, seven a rating of 4, none a rating of 3, four a rating of 2, two a rating of 1, and twelve a rating of 0 (exclusively heterosexual). Thirty of this group also reported interpersonal sexual behaviour. Of these eight had a Kinsey rating of 6, six a rating of 5, seven a rating of 4, one a rating of 3, two a rating of 2, one a rating of 1, and five a rating of 0. The remaining 14 had not yet engaged in sufficient sexual behaviour to warrant a rating.

Of the 35 males who were previously 'masculine' boys, none had a Kinsey rating of sexual fantasy between 2 and 6 (bisexual to homosexual), and two had a rating of 1 (incidentally homosexual, but predominantly heterosexual). Of the 22 previously 'masculine' boys who reported interpersonal sexual behaviour, all had a rating of 0. The remaining 13 had not yet engaged in sufficient sexual behaviour to warrant a rating.

This high incidence of homosexual orientation among adolescents or young adults who displayed cross-gender behaviour in boyhood grants further legitimacy to the thesis that such types of behaviour are precursors of adult homosexuality.

TABLE III
Correlations of sexual orientation ratings with atypical sex-typed behaviour, with and without controlling for age at time of initial evaluation

Behaviour	*Sexual fantasy rating*		*Sexual behaviour rating*	
	Zero-order	*Partial*	*Zero-order*	*Partial*
Role/doll play				
ROLEPLAY	0.24*	0.25*	0.30*	0.30*
PLAYHS	0.11	0.13	0.18	0.18
DOLLPLAY	0.36**	0.36**	0.22	0.22
DOLPLAY	0.25*	0.22*	0.21	0.20
Cross-dress				
XDRESS	−0.09	−0.08	0.11	0.12
NOXDRS	−0.07	−0.03	−0.03	−0.02
IMPROVS	−0.17	−0.16	0.02	0.03
INTEREST	0.09	0.10	0.21	0.21
COMMENT	0.15	0.11	0.14	0.13
Female peers				
FRIENDS	0.19	0.26*	0.22	0.25
RELATE	0.06	0.09	−0.08	−0.07
Rough play				
SPORTS	−0.04	0.03	0.02	0.05
RUFHS	0.10	0.13	0.16	0.16
Be girl				
BEGIRL	0.04	0.11	−0.05	−0.03

*$P<0.10$; **$P<0.05$

Table II shows the correlations found between the cross-gender behaviour items listed in Table I. Correlations within each grouping of items are to the lower left hand side of the line. The fact that some correlations above this line are significant indicates the inter-relatedness of the types of cross-gender behaviour. For example, female role-playing and dress-up doll play often involve cross-dressing or an interest in feminine clothing. Desire to be a girl may be accompanied by cross-dressing and the ability to relate better to girls than to boys. However, the correlations within each of the five groupings in Table I are strong enough to permit their use as a basis for later generalisations.

Table III displays our principal findings: the zero-order correlations between the 14 measures of boyhood sex-typed behaviour and Kinsey sexual orientation ratings in adolescence and young adulthood within the 'feminine boy' group. The variables related to later sexual orientation are those items that fall into the role-playing and doll play category and the peer group cateogry. Doll playing has the strongest association of the three, and is related to sexual fantasy. Female role-playing tends to be related to both fantasy and behavioural ratings. Female peer group tends to be related only to fantasy, and then only when age at initial evaluation is controlled for. Cross-dressing is not associated with Kinsey ratings, and sexual orientation seems unrelated to the degree of rough-and-tumble play or sports, or the reported frequency of the stated desire to be a girl.

Some of the variables in Table III are associated with the boys' age at evaluation. For example, female role-playing, doll play, and cross-dressing are much less typical of a seven year-old than a four year-old. Futhermore, if a seven year-old cross-dresses, the chances are that he also cross-dressed at four. However, the reverse may not be as likely. Therefore, the zero order correlations were controlled for age at evaluation. To do this, a regression of variable X was run on age, and the residuals taken. The residuals are variable X adjusted for the linear effects of age. If we control the zero order correlations in Table III for age at evaluation, the correlations with role-playing and doll playing remain relatively unchanged. Thus, the associations do not appear to be a chance function of the age at which the boyhood measures were taken.

Discussion

Conclusions drawn from these data must be carefully qualified, taking into account the nature of the groups. Only boys who had atypical amounts of most of the five types of cross-gender behaviour were accepted for evaluation of the UCLA Gender Identity Clinic. Yet without taking into account variations in these boys' sex-typed behaviour, we know that the odds are about two to one that these boys would develop more than incidental homosexual fantasies in adolescence or young adulthood, and have more than incidental homosexual experiences.

Given that a boy does exhibit atypical amounts of cross-gender behaviour, the best predictors of later homosexual fantasies are, on the basis of our data, the degree to which he plays with dress-up dolls and (to a lesser extent) role-plays as a female or has females in his peer group. For later homosexual behaviour, the best predictor is role-playing as a female. Within the 'feminine boy' group cross-dressing is a poor predictor in this regard, as is the degree to which he participates in rough-and-tumble play or sports, and states the wish to be a girl. However, each behaviour is predictive of later sexual orientation, when comparing the two groups.

Since about half of transvestite males, who derive sexual arousal from dressing in women's clothes, begin cross-dressing prior to adolescence (Prince & Bentler, 1972), we predicted that some of the cross-dressing boys would become adult transvestites. Despite considerable cross-dressing among the boys, however, no one in our group developed transvestism. The reason for this may be the small probability of sampling a pre-transvestite, given the lower incidence of transvestism compared to homosexuality in the general population, and the small number of boys in our group. Transsexuals, too, report extensive childhood cross-gender behaviour (Benjamin, 1966). Despite a similarly low incidence of transsexualism in the general population, one of the previous 'feminine' boys, who is erotically attracted to males, also desires a sex-change operation (Green, 1987).

The development of adult sexual orientation appears most likely to involve interplay among a variety of intervening factors, such as innate features, relationship to parents, medical background, and parental concern over cross-gender behaviour (Green, 1987). Here we have only looked at a small portion of the developmental picture, but have found that a variety of types of boyhood behaviour appear characteristic of pre-homosexual males.

Comparing our two groups of males, several types of boyhood behaviour were more commonly found in the pre-homosexual or bisexual males. These included the frequently stated wish to be a girl, frequent cross-dressing, preferential role-playing as a female, a primarily female peer group, a preference for dress-up doll-play, an avoidance of rough-and-tumble play, and an avoidance of sports. However, within the 'feminine' boy group, it was primarily role-playing as a female, playing with dress-up dolls, and a female peer group that were associated with later ratings of sexual orientation. Here, the more these types of behaviour were engaged in, the higher (more homosexual) was the later rating of sexual orientation.

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CSF Monoamines in Autistic Syndromes and Other Pervasive Developmental Disorders of Early Childhood

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Spinal fluid concentrations of the three major monoamine metabolites were examined in 25 infantile autistic children and 12 children with other childhood psychoses, and were contrasted with results obtained in normal children and in groups of children with neurological and neurodevelopmental disorders. Autistic children showed absolute and relative increases of the dopamine metabolite homovanillic acid. The group with other childhood psychoses also showed an increase in HVA level; in this group there were also indications of high levels of serotonin and norepinephrine metabolites. The results are discussed in the context of a pathogenetic model for autism involving hyperfunction of dopaminergic nerve fibres in the brain stem-mesolimbic system.

Infantile autism – as defined by DSM–III (American Psychiatric Association, 1980) – and the other childhood psychoses are currently thought to be caused by hereditary factors or brain damage, singly or in combination (Folstein & Rutter, 1977; Ornitz, 1983; Coleman & Gillberg, 1985). Biochemical dysfunction is likely to be of importance, and several early studies on peripheral blood have indicated hyperfunction of serotonin (Hanley *et al*, 1977; Ritvo *et al*, 1970). Later studies have indicated hyperfunction of dopamine, as judged by measurements of homovanillic acid (HVA) and endorphin in the cerebrospinal fluid (CSF) (Winsberg *et al*, 1980; Gillberg *et al*, 1983; Gillberg *et al*, 1985). We recently reported preliminary findings of raised HVA levels in the CSF of 13 autistic children (Gillberg *et al*, 1983). We now present results of CSF monoamine analyses on 37 psychotic children, 25 of whom were diagnosed as suffering from infantile autism.

Subjects

Infantile autism

Twenty-five children (20 boys and 5 girls) diagnosed by an experienced child psychiatrist as suffering from infantile autism (IA) were included in the study. They were consecutive autistic patients at a university clinic of child psychiatry in Western Sweden, where the majority of autistic children in that part of the country are assessed. All cases had: (a) early onset (before 30 months of age); (b) severe disturbance of social relatedness, involving lack of reciprocity and a period in infancy of autistic aloneness; (c) severe disturbance of language, expressed as muteness or echolalia with reversal of pronouns, plus impaired comprehension of mime, gesture and spoken language; and (d) severe behaviour disturbances involving insistence on sameness, development of elaborate repetitive routines, and odd play behaviour. All the children, who ranged in age from 1–16 years of age (mean = 8.1, s.d. = 4.3), fulfilled DSM–III and Rutter's (1978) criteria for infantile autism and fitted the clinical picture outlined by Kanner & Eisenberg (1956).

A number of the autistic children showed additional handicaps, such as mental retardation or epilepsy (Table I), and two children were on medication (clomipramine) at the time of the study.

Other childhood psychoses

Twelve children (8 boys and 4 girls) fulfilled DSM–III criteria for childhood onset pervasive developmental disorder; i.e. they showed many of the symptoms typical of autism, but the age of onset could not be objectively proven using medical record data to be prior to 30 months (which does not preclude the possibility of early symptoms). These children will be referred to as suffering from 'other childhood psychoses' (OP). Some of the children in this group also were multiply handicapped, but only two (both girls) were on any kind of medication (one on valproate and one on carbamazepine). The age range in this group was 3–13 years (mean = 8.3, s.d. = 3.3).

Control groups

The control group for IA consisted of 20 children (15 boys and 5 girls), matched for sex and age (within one year, or two months for two children aged under four) with the first 20 of the 25 autistic children. The mean age at examination in this group was 8.3 years (s.d. = 4.3).

The control group for OP consisted of 10 children (6 boys and 4 girls) matched for sex and age (within two years) with the first 10 of the 12 children with other childhood psychoses. (The psychotic children under four years were matched for age with children who were two months younger.) The age range in this group was 3–13 years (mean = 10.0, s.d. = 3.5).

TABLE I
Characteristics of the IA and OP groups of children

Group	*Case no.*	*Sex*	*Age: years*	*IQ*	*Epilepsy*	*Other major handicap/abnormality*[1]
Autism	1	M	1	~35		Fragile (X) (p22)[2] (relevance unknown)
	2	M	3	54		Brain stem dysfunction[2]
	3	M	4	80		Temporal lobe spike and wave activity[4]
	4	M	4	95		Brain stem dysfunction[3]
	5	M	4	~20		—
	6	M	4	~20		—
	7	M	5	50		Brain stem dysfunction[3]
	8	M	5	40		Fragile (X) (p22)[2], (relevance unknown)
	9	M	6	46		Metabolic disorder[3]
	10	M	6	67		Brain stem dysfunction[3]
	11	M	6	56		—
	12	M	6	~30		—
	13	M	6	~25		Brain stem dysfunction[3]
	14	M	7	85	+	Fragile (X) (q273)[2], Brain stem dysfunction[3]
	15	M	9	56	+	Fragile (X) (q273)[2]
	16	M	11	70		Brain stem dysfunction[3]
	17	M	13	60		—
	18	M	14	20		—
	19	M	14	65		Temporal lobe spike and wave activity bilaterally[4]
	20	M	14	~35		Moderate increase of low frequency activity[4]
	21	F	10	30	+	Temporal lobe spike and wave activity on left side, clomipramine treatment
	22	F	10	67	+	Neurofibromatosis
	23	F	13	80		—
	24	F	14	100		—
	25	F	15	60	+	—
Other psychosis	1	M	3	~30	+	Temporal lobe discharge,[4] carbamazepine treatment
	2	M	4	45		—
	3	M	7	80		—
	4	M	8	45		—
	5	M	8	42		Neurofibromatosis
	6	M	9	65		CSF protein signs of degeneration
	7	M	9	68	+	Bilateral temporal lobe spike and wave activity[4]
	8	M	11	67		Fragile (X) (p22)[2] (relevance unknown)
	9	F	8	—		—
	10	F	10	132		—
	11	F	12	~15	+	Rett syndrome
	12	F	13	66	+	Bilateral synchronous spike and wave activity,[4] plus temporal lobe spike and wave activity, sodium valproate treatment

1. Results of computerised tomography scans and neurochemical tests not included.
2. Chromosomal culture in folic-acid-depleted medium.
3. According to examination of auditory brain stem responses.
4. According to EEG in the waking state.

All the children in these control groups had had a lumbar puncture because a disorder affecting the central nervous system (febrile virosis, pneumonia or otitis with meningismus ($n=16$), syncope ($n=2$), Bell's palsy ($n=1$), retinal degeneration ($n=1$), headache ($n=3$), paraesthesia, hysteria? ($n=1$), obsessions ($n=1$), tics ($n=1$), and other transient neurological problems ($n=4$)) had been suspected; these disorders were later ruled out. None of the children in these groups were on medication when their CSF samples were taken.

Mental retardation group

A group consisting of 10 non-autistic, non-psychotic mentally retarded (MR) children, aged 3–14 years (mean = 8.0, s.d. = 2.7) were also examined. Four of these also had cerebral palsy (CP), and one child had the combination of MR, CP and epilepsy. The epileptic child was on phenytoin; no other children were on medication at the time of the study.

Progressive encephalopathy group

Five children (3 boys and 2 girls) with severe mental retardation and unequivocal signs of deterioration were referred to the progressive encephalopathy group. They were without psychotic symptoms. The age range was 4–13 years (mean = 9.2, s.d. = 4.3). None of these children were receiving drugs.

Meningitis group

Seven children (4 boys and 3 girls) were referred to the meningitis group; CSF specimens for monoamine analysis were obtained in the acute febrile state of the disease before any kind of treatment had been started. The causative agents were Haemophilus influenza in one case and possibly viral in the remaining six cases. The age range in this group was 2–16 years (mean = 7.6, s.d. = 4.7).

Method

Lumbar puncture/CSF collection

Lumbar punctures were performed under local anaesthesia. All children in the study and the comparison groups had been allowed to move about freely before the lumbar puncture, and there had been no other restriction. Two children in the IA comparison group had been confined to bed because of a virus infection and pyrexia. No premedication was given. The first 3 ml of CSF was collected from all children in the study, and was kept frozen until the monoamine analysis was made.

The lumbar punctures were performed by the same child psychiatrist in 30 of the 37 cases with psychosis, because he was well known to the children and doctors not well acquainted with the problem of psychosis in childhood often feel uneasy in such situations. In the majority of the cases with psychosis the child was also accompanied by a therapist or teacher whom they knew well, who would sit by the child's side and talk or sing to him or her during the procedure. The majority of the lumbar punctures were performed in the afternoon, especially in the psychosis group and their comparison groups. Some of the punctures (e.g. those done on acute indication) were performed at other times.

Determination of monoamine metabolites

Mass fragmentographic determination of HVA and 5-hydroxyindole-acetic acid (5-HIAA) was performed on 0.2 ml samples of CSF in a procedure modified from that described by Swahn *et al* (1976). To each sample of CSF, 0.4 nmol of deuterated HVA and 0.2 nmol of deuterated 5-HIAA were added as internal standards. The pH of the sample was adjusted to about 2 with formic acid, and after saturation with sodium chloride the metabolites were extracted with two portions of diethyl ether (0.5 ml). The solvent was evaporated under nitrogen, and the pentafluoropropionyl derivatives were prepared by the addition of 20% pentafluoropropanol in pentafluoropropionic anhydride (50 µl) and heating to 75°C in a sealed tube. The process of forming derivatives was repeated once; the sample was then evaporated to dryness *in vacuo* and finally dissolved in ethyl acetate (25 µl). About 5 µl of the solution was used for the analysis.

Hydroxymethoxyphenylglycol (HMPG) was assayed on a second sample of 0.2 ml CSF. After the addition of deuterated HMPG (0.1 nmol) and saturation with sodium chloride, the sample was extracted with two portions of ethyl acetate (0.5 ml) instead of diethyl ether. After evaporation, a trifluoro-derivative was made by the reaction of the sample dissolved in ethyl acetate (25 µl) with trifluoroacetic anhydride (25 µl) for one hour at room temperature. All determinations were done in duplicate on a LKB 2091 gas chromatograph/mass spectrometer. The recovery of the three metabolites varied between 75% and 90%, and the coefficient of variation, determined from duplicate analyses, between 3.6% for HVA and 6.8% for 5-HIAA.

Results

Infantile autism group

Mean CSF HVA concentrations were significantly higher in the autism group than in the IA control group ($P < 0.001$); 18 of the 20 control group children had lower HVA concentrations than their corresponding autism proband. 5-HIAA and HMPG concentrations did not differ significantly between the two groups (Table II).

The concentrations of HVA and 5-HIAA relative to that

TABLE II

HVA, 5-HIAA and HMPG CSF concentrations in the IA and control groups

	Infantile autism group (n = 25): nmol/l		*Control group (n = 20): nmol/l*	
	Mean	*s.d.*	*Mean*	*s.d.*
HVA	460*	146	299*	91
5-HIAA	129	45	114	38
HMPG	72	13	65	16

*$P < 0.001$.

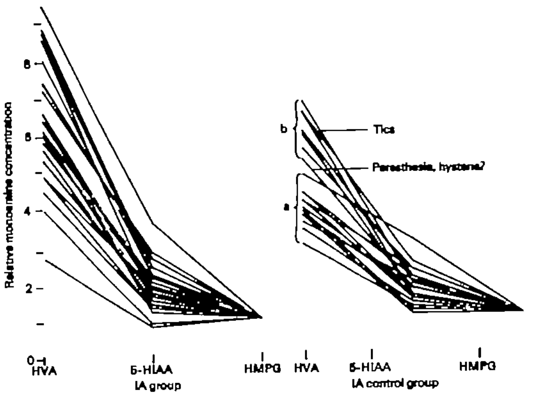

FIG. 1. HVA and 5-HIAA concentrations in the IA and control groups, relative to HMPG concentration.

TABLE III

HVA and 5-HIAA concentrations (relative to HMPG = 1) in the IA, OP and control groups

Group	*Relative HVA concentration*		*Relative 5-HIAA concentration*	
	Mean	*s.d.*	*Mean*	*s.d.*
IA ($n=25$)	6.4*	1.6	1.9	0.6
IA control ($n=20$)[1]	4.7*	1.2	1.8	0.5
OP ($n=12$)	7.4	4.3	2.1	1.2
OP control ($n=10$)	4.9	1.8	1.8	0.9

1. Including 2 cases where ratios were calculated on basis of mean HMPG values.
*$P<0.001$.

TABLE IV

HVA, 5-HIAA and HMPG CSF concentrations in the OP and control groups

	Other childhood psychosis group ($n=12$): nmol/l		*Control group ($n=10$): nmol/l*	
	Mean	*s.d.*	*Mean*	*s.d.*
HVA	513*	219	263*	174
5-HIAA	148	57	103	46
HMPG	73**	12	61**	8

*$P<0.01$; **$P<0.05$.

of HMPG, which showed the least variation of the metabolites in all the study groups, are shown in Table III and Fig. 1. Not only is HVA raised in the autistic group, but the relative excess of this metabolite as compared with 5-HIAA is marked. The IA control group appears to consist of 2 subgroups: one (labelled (a) in Fig. 1) with an almost linear relationship between the three metabolites, and another (labelled (b) in Fig. 1) with a more conspicuous discrepancy between relative HVA and 5-HIAA concentrations. At least two of the seven cases in subgroup (b) had psychiatric problems. It might be that subgroup (a) is in fact the 'normal' comparison group.

The mean HVA:HMPG ratio was significantly higher in the autism than in the IA control group ($P<0.001$). No difference between the groups emerged in respect of 5-HIAA:HMPG ratios.

Other psychosis group

Mean CSF HVA concentrations were raised in the OP group compared with the OP control group ($P<0.01$) (Table IV, Fig. 2). Eight of the 10 OP control group children had lower HVA concentrations than their corresponding psychotic probands. CSF 5-HIAA and HMPG concentrations tended to be higher in the psychotic group, but the differences fell just short of statistical significance in respect of 5-HIAA.

Other groups

The HVA concentrations in the MR group (mean = 271, s.d. = 109 nmol/l) were close to those of the control groups, but 5-HIAA and HMPG levels were slightly higher (mean = 130, s.d. = 59 nmol/l and mean = 80, s.d. = 50 nmol/l respectively). The HVA and 5-HIAA concentrations were much higher in the progressive encephalopathy group than in the control groups (mean = 605, s.d. = 114 nmol/l and mean = 276, s.d. = 116 nmol/l respectively). There was no difference with regard to HMPG concentration (mean = 70, s.d. = 9 nmol/l). In the meningitis group the mean HVA concentration was almost as high as in the progressive encephalopathy group, but there was considerable individual variation (mean = 598, s.d. = 366 nmol/l). The 5-HIAA concentration was also raised, compared with the control group (mean = 167, s.d. = 64 nmol/l), but the HMPG concentration was in the normal range (mean = 75, s.d. = 14 nmol/l).

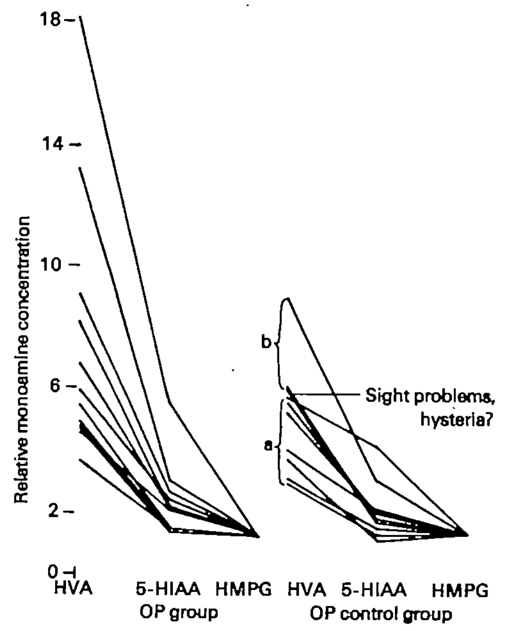

FIG. 2. HVA and 5-HIAA concentrations in the OP and control groups, relative to HMPG concentration.

Age, IQ and sex correlations with monoamine concentrations In all the study groups there were clear-cut age trends; monoamine concentrations generally declined with increasing age of the child (Table V). This appeared to hold true especially if the children were under the age of five or if they were of short stature for other reasons.

IQ did not correlate in any meaningful way with HVA concentrations, either in the autism group, or in the group with other childhood psychoses. However, it appeared that in the autism group high levels of 5-HIAA correlated with low IQ ($r = -0.45$, $P < 0.05$).

TABLE V

Pearson product-moment correlation between age and monoamine concentrations in the IA, OP and control groups

Group	*HVA*	*5-HIAA*	*HMPG*
IA ($n=25$)	−0.56**	−0.66**	−0.07
IA control ($n=20$)	−0.62**	−0.74**	−0.47*
OP ($n=12$)	−0.58*	−0.63*	−0.16
OP control ($n=10$)	−0.85**	−0.64*	−0.34

*$P < 0.05$; **$P < 0.01$.

The girls tended to show slightly lower monoamine CSF-concentrations than the boys. However, all differences were statistically insignificant. Since there were relatively few girls the results are not conclusive. Furthermore, the girls were somewhat older than the boys, and the differences might be accounted for by age effects.

Discussion

We have previously presented preliminary evidence indicating dopamine hyperfunction in the CSF of autistic children (Gillberg *et al*, 1983). This new extended study on 25 autistic children and 12 children with other psychoses of early childhood adds further support for the hypothesis (Maurer & Damasio, 1982; Coleman & Gillberg, 1985) that dysfunction of CNS dopaminergic pathways might be important in autism and autistic-like conditions.

However, several things must be borne in mind. Firstly, although in this context the group was fairly large, the number of autistic children included was too small for firm conclusions to be drawn. The number of children in the other psychosis group (and its control group) was much too small for the results in that group to serve as anything other than a basis for tentative suggestions.

Secondly, the control groups of children did not consist of perfectly healthy children, although a majority of them were free of psychiatric disorders. This means that we have not provided hard proof that levels of HVA in CSF are really higher in autistic or psychotic children than in normal children. However, we consider our control groups acceptable, and feel that ethical considerations would preclude a CSF study on children without even any kind of acute ailment.

Thirdly, high dopamine levels should not be taken as ultimate proof of primary central nervous system dysfunction. However, studies by Sedvall *et al* (1980) on adults provide support for the notion that altered monoamine metabolism in the CSF indicates primary central nervous system vulnerability.

Finally, one might argue that factors not intrinsic to the psychotic disorder (e.g. food, drink, or activity level) might cause the deviant results. We found no such meaningful correlation, but some factors may have been missed.

It appeared that isolated elevation of dopamine was typical of autism, and that in the other psychosis group all three metabolites tended to be raised. Several studies have shown that about one-third of 'autistic' children have raised levels of blood serotonin. The tendency found in our study towards high CSF 5-HIAA levels in the other psychosis group is therefore of some interest. However, in our autistic subjects high CSF 5-HIAA levels correlated significantly with mental retardation but not with autism. This might mean that hyperfunction of 5-HIAA in the field of childhood psychosis is a non-specific finding linked with the IQ rather than with the psychiatric abnormality of the child. This line of reasoning has been supported by a number of authors (see Young *et al*, 1982; Coleman & Gillberg, 1985).

There was no indication in the present study that the high HVA levels could be accounted for by mental retardation. In the autistic group, high HVA levels correlated with autism and not with low IQ. Furthermore, the mentally retarded group without autistic features showed results compatible with those of the normal control groups.

High monoamine metabolite levels in the CSF showed an inverse relationship with age both in the psychosis and the control groups. There was no suggestion that the age trend could explain the difference between index and control cases. Nevertheless, 'immaturity' of the nervous system in the psychotic children might turn out to be one important factor, even though the results in the small mental retardation group tended to contradict such an interpretation.

A number of authors (Young *et al*, 1982; Maurer & Damasio, 1982; Coleman & Gillberg, 1985) have hypothesised – on the basis of animal research, results from adult psychiatry and simple case studies of autistic syndromes in childhood – that hyperfunction of dopaminergic pathways arising in the brain-stem and projecting rostrally to mesolimbic structures in the temporal and frontal lobes might be crucial in the pathogenesis of certain symptoms in autism. Coleman & Gillberg (1985) have proposed a theory implicating bilateral mesolimbic dysfunction or damage as essential in producing the core symptoms (incomprehension of human mime and gesture, inability to relate reciprocally to other people) in infantile autism. Mesolimbic areas might be dysfunctional *sui generis*, but may also be dysfunctional because of lack of input from the brain-stem, which in turn might be caused either by primary brain-stem damage (leading to denervation hyperfunction of the dopaminergic pathways connecting the brain-stem with the mesolimbic structure) or by primary dysfunction of the dopaminergic nerve fibres. We found evidence of brain-stem dysfunction in 28% of our autistic group (and in none of the other psychosis group) and of bilateral temperal lobe dysfunction in 12% (8% in the other psychosis group).

It is unclear whether there exist real differences in respect of CSF monoamine contents between children with autism and those with other childhood psychoses. Our results indicated specific dysfunction of dopamine in autism and non-specific elevation of all the studied metabolites in the other psychosis category, but further studies are needed before any well-founded hypotheses can be proposed. The ratio of HVA:5-HIAA:HMPG might prove to be more important than absolute values for these metabolites. For instance, the HVA:HMPG ratio was greater than 6 in 64% of the autism cases, in 44% of the other psychosis cases (in spite of the fact that this group showed the highest absolute values), and in 15% and 10% respectively of cases in the comparison groups.

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Relationship between Interictal Psychopathology and the Type of Epilepsy

Results of a Survey in General Practice

J. EDEH and B. TOONE

Psychiatric morbidity was assessed in a sample of 88 adult epileptic patients drawn from general practices in South London. Using the Clinical Interview Schedule, 48% emerged as psychiatric cases. When either total CIS score or caseness status was used for comparison, group differences were evident; patients with temporal lobe epilepsy and focal non-TLE did not differ, but each was significantly more impaired than those with primary generalised epilepsy. The groups also differed in their psychiatric symptom profiles. The results suggest that the increased prevalence of interictal psychopathology commonly associated with TLE may also be a feature of other forms of focal epilepsy.

Much has been written on the nature of psychiatric illness in epilepsy, and few would dispute that there appears to be a substantial increase in psychiatric morbidity among epileptic patients. However, our understanding of this subject has been formed by studies of patients in the settings of hospital, institution, or clinic. Although often of high quality, these offer a limited perspective of a highly selected clinical population and one moreover that is selected for, among other factors, vulnerability to psychiatric illness. Prevalence measures derived from such unrepresentative samples may therefore seriously overestimate psychiatric morbidity among epileptic patients. Other issues may also be obscured: one that remains a source of controversy is the alleged vulnerability of patients with temporal lobe epilepsy (TLE) to psychiatric illness.

The central role of the limbic system in the integration and expression of emotional experience has long been recognised (Papez, 1937; Yakovlev, 1948; McLean, 1970) and the increased prevalence of psychiatric disorder among epileptic patients is also well established (Pond & Bidwell, 1959; Gudmundsson, 1966; Rutter *et al*, 1970). The early observation of an association between anterior temporal lobe foci and the presence of major psychiatric illness (Gibbs *et al*, 1948) was therefore not unexpected, and was in accordance with common clinical experience. Further studies (Pond & Bidwell, 1959; Currie *et al*, 1971) have given evidence of an unusually high prevalence of psychiatric disorder in patients with TLE, while an association has also been reported with more specific behavioural syndromes and disabilities, e.g. sexual deviation (Kolarsky *et al*, 1967), personality disorder (Bear & Fedio, 1977), and psychosis (Slater *et al*, 1963). However, a number of studies (Small *et al*, 1962; Stevens, 1966; Standage & Fenton, 1975) have reported essentially negative findings, so that the issue remains controversial and unresolved.

Methodological shortcomings may explain some of these discrepancies. In early studies in particular, diagnostic criteria for both epilepsy and psychiatric disorder were ill-defined, and as a consequence major inconsistencies in the reported prevalence of different disorders are not infrequent, e.g. psychosis in TLE (Ervin *et al*, 1955; Stevens, 1966). The choice of investigatory techniques has also been criticised (Bear & Fedio, 1977). Questionnaires such as the Minnesota Multiphasic Personality Inventory, standardised against an unselected population and designed to measure trait rather than state, may be too insensitive to elicit the more specific personality characteristics observed in TLE and ill-fitted to record current mental state.

However, the choice of unrepresentive patient samples remains the most likely source of error (Stevens, 1966). Poor treatment response in newly referred patients is associated with the presence of complex partial seizures and with neuropsychiatric findings (Reynolds *et al*, 1983); such patients are less likely to be referred back to their general practitioners for continuing care and will in time come to make up the greater number of clinic attenders (Edeh, 1984). Epidemiological findings support this view (Gastaut *et al*, 1975; Hauser & Kurland, 1975).

Any attempt to compare temporal lobe and non-temporal lobe epileptics with respect to psychiatric morbidity will therefore be prone to sampling error.

These biases can best be avoided by a comprehensive survey of a population of unselected epileptic patients in the community, but regrettably there has been little published work in this area. Pond & Bidwell's (1959) study of 14 general practices appears in retrospect a landmark that has not yet been repeated. Excellent though this study was, the availability of investigatory facilities was then limited and specialist opinion not always sought. The criteria for diagnosis of epilepsy subtypes would not satisfy present-day requirements, while instruments for measurement of psychiatric morbidity in a community were not yet available. The latter was expressed in terms of the proportion of patients who had sought or received psychiatric treatment – the "conspicuous morbidity" (Kessel, 1960). The aim of the present study was to estimate both the conspicuous and hidden morbidity in an unselected general practice-based population. Application of rigorous diagnostic criteria for temporal lobe and non-temporal epilepsy should permit a comparison of these epileptic sub-categories in terms of psychiatric disturbance.

Method

We examined the prescribing arrangements of 14 general practitioners working in five group practices in South London (Lambeth, Croydon, and Bromley); 103 patients (aged 16 years and over) out of a total registered adult population of 29 822 (prevalence rate 3.45/1000) regularly received repeat prescriptions of anticonvulsant medication. All satisfied the operational criteria for the definition of epilepsy of Gunn & Fenton (1969): "a history of at least three epileptic attacks in any two-year period and the continuation of anticonvulsant therapy, if the period did not immediately precede the time of examination".

Eighty-eight of the sample agreed to participate in the survey. All practice records were scrutinised for information regarding history of epilepsy, previous investigations, and current treatment, while further information on 79 patients was obtained from hospital casenotes. Each patient was seen at home in the evening for psychiatric assessment using the Clinical Interview Schedule (CIS) (Goldberg *et al*, 1970), and was subsequently invited to attend hospital for clinical investigations; these were carried out at the Maudsley Hospital within 7–14 days of the interview. The home visits and clinical investigations were completed in 15 months.

Psychiatric morbidity

Psychiatric ratings on the CIS were derived from ten symptoms commonly reported in general practice and from 12 manifest abnormalities observed by the psychiatrist. Each of the 22 ratings were made on a five-point scale: O for normal; 1 for sub-clinical disturbance or trait; and 2, 3 and 4 for mild, moderate, or severe clinical disturbance respectively. As recommended by Goldberg, a total CIS score was calculated for each patient by adding together the ratings for all ten reported symptoms and double the ratings for all 12 manifest abnormalities.

A patient scoring 13 or more was classed as a case (Benjamin *et al*, 1982; Hobbs *et al*, 1983). Each case received a clinical diagnosis according to ICD-9 (World Health Organization, 1978).

Social correlates of psychiatric disorder were assessed using the Schedule for Social Maladjustment and Dysfunction (Clare & Cairns, 1978); this will be the subject of a separate paper.

EEG and CT scans

A 16 channel electroencephalographic (EEG) recording was carried out on all patients, using the 10–20 international system of electrode placement. All records were assessed by established methods of visual interpretation by a consultant clinical neurophysiologist. Computerised tomography (CT) was carried out using an EMI CT 1010 Head Scanner; enhancement, when indicated, was achieved with intravenous iodinated contrast medium (sodium iothalamate – Conray 420, 1 ml/kg body weight). The CT scan films were assessed independently by a consultant neuroradiologist.

Diagnosis and classification of epilepsy

The type of epilepsy was diagnosed on combined clinical and EEG considerations (Gastaut, 1969; Parsonage, 1983). Patients with primary generalised epilepsy (PGE) lacked a preceding aura, localising event during or after the seizure, a history of cerebral insult, or focal neurological signs; EEG was normal or showed generalised spike, polyspike, or spike-wave complexes with bilateral distribution and without a sharp or slow wave focus.

Partial epilepsies were differentiated by the presence of an initial event of localising significance and an EEG spike, sharp or slow wave focus. Patients with TLE presented features of complex partial seizures or abnormal interictal EEG records showing spike, sharp, or slow wave focus, and phase-reversal over the anterior or mid-temporal areas (Ervin, 1967; Driver, 1970).

Coding and statistical analysis

All the survey data were entered into coded questionnaires and punched onto cards, which were verified for punching errors. Analysis was subsequently carried out on the University of London's AMDAHL 470/v8 computer, accessed through the King's College Hospital Medical School terminal system. Significant differences across the epilepsy sub-categories were determined using the Kruskal-Wallis one-way analysis of variance (ANOVA) (Siegel, 1956); comparison between groups used the Mann-Whitney U test or the chi-squared test as indicated.

TABLE I

Characteristics of the epilepsy sub-categories

	PGE *(n=41)*	*Focal non-TLE* *(n=22)*	*TLE* *(n=25)*
Sex			
Male	27	13	14
Female	14	9	11
Age			
Mean	41.65	46.09	43.48
s.d.	18.61	20.17	14.98
Age of onset			
Mean	24.00	24.90	20.68
s.d.	19.41	22.87	14.79
Duration of epilepsy			
Mean	17.65	21.18	22.80
s.d.	11.52	14.78	12.86
Seizure frequency			
Controlled	17 (41%)	4 (18%)	5 (20%)
Uncontrolled	24 (59%)	18 (82%)	20 (80%)
No. of drugs prescribed			
One	23 (56%)	11 (50%)	9 (36%)
Two	16 (39%)	11 (50%)	15 (60%)
Three or more	2 (5%)	0	1 (4%)
CT scan			
Normal	32 (78%)	9 (41%)	14 (56%)
Abnormal*	9 (22%)	13 (59%)	11 (44%)

*$\chi^2 = 6.78$ ($P<0.01$) for differences between PGE and focal non-TLE.

Results

Patients were grouped into three epilepsy sub-categories: TLE ($n=25$), focal non-TLE ($n=22$) and PGE ($n=41$) according to the criteria described above. Fifteen patients refused to participate in the study, or agreed to be interviewed but failed to attend for hospital investigations. According to their casenotes, six would appear to have PGE and six partial epilepsy. Seven had a history of psychiatric illness (four cases with neuroses, and one each with personality disorder, periodic psychotic episodes, and mental subnormality); thus, they did not seem to differ markedly from those in the study.

Characteristics of the groups

The results are summarised in Table I. The male female sex ratio was approximately the same for all three groups. Mean age, age of seizure onset, and duration of epilepsy were broadly similar. Control of seizure was better in patients with PGE compared with either those with focal non-TLE or TLE, but the difference did not reach statistical significance. More patients with PGE were on a single anticonvulsant drug, compared with those with TLE and focal non-TLE.

In the PGE group 22% of the patients had abnormal CT scan, compared with 59% of those with focal non-TLE; this difference is significant ($\chi^2=6.78$, $P<0.01$), but comparison of CT scan abnormalities between patients with PGE and TLE or between focal non-TLE and TLE did not reveal a significant difference.

History of previous psychiatric referral

Twenty-seven patients (31%) had a history of psychiatric

TABLE II

Comparison of the epilepsy sub-categories on the CIS items

CIS items	*ANOVA** *H*	*P*	*Inter-group*** *differences*
Reported symptoms			
Irritability	5.644	0.05	PGE vs TLE; $P<0.02$ PGE vs focal non-TLE; $P<0.01$
Impaired concentration	9.854	0.007	PGE vs TLE; $P<0.01$
Depression	6.314	0.043	PGE vs TLE; $P<0.02$
Anxiety	5.715	0.05	TLE vs focal non-TLE; $P<0.02$
Manifest abnormalities			
Depressed affect	6.815	0.03	PGE vs TLE; $P<0.02$ TLE vs focal non-TLE; $P<0.05$
Intellectual impairment	7.652	0.02	PGE vs focal non-TLE; $P<0.01$

*Kruskal-Wallis H statistics, corrected for ties.

**Mann-Whitney U test.

referral – six with PGE, ten with focal non-TLE, and eleven with TLE ($\chi^2 = 4.83$, NS). The difference between focal epilepsy (TLE and non-TLE) and PGE was significant ($\chi^2 = 7.94$, d.f. = 1, $P < 0.01$).

Psychiatric symptom profile

Statistical analysis was performed on the CIS ratings in two stages: the Kruskal-Wallis one-way analysis of variance was applied across the groups, and where a significant difference was observed at the one in twenty level significance, further analysis was carried out using the Mann-Whitney U test to determine differences between groups.

Difference across groups (Table II). Significant differences across groups were demonstrated for mean ratings on irritability, depression, anxiety and impaired concentration; among manifest abnomalities, depressed affect and intellectual impairment had significant differences.

Other CIS items rated under reported symptoms (somatic, fatigue, sleep disturbance, phobias, obsessions and depersonalisation)and under manifest abnormalities (spontaneity, suspiciousness, histrionic behaviour, anxiety, elated or flat affect, delusions, hallucinations, excessive concern with bodily functions, and depressed thoughts) failed to show any significant difference across the epilepsy sub-categories.

Difference between groups. On the reported symptoms the TLE group rated more highly on anxiety than the focal non-TLE group and more highly on irritability, depression and impaired concentration than the PGE group.

On manifest abnormalities the TLE group scored more heavily on depressed affect than either the PGE or focal non-TLE groups. The focal non-TLE group was more intellectually impaired than the PGE group at the one in one hundred level.

Severity of psychiatric disturbance

Figure 1 shows the distribution of the total CIS scores for the three epilepsy sub-categories. Differences between patients with focal non-TLE and TLE were not significant, but the differences between those with PGE and TLE, and between those with PGE and focal non-TLE were statistically significant at the one in twenty level.

The proportion of patients regarded as normal (rating 0) and those with varying degrees of psychiatric disturbance (ratings 1–4) did not differ between the groups with partial epilepsy, but significant differences were observed between PGE and focal non-TLE and between PGE and TLE (Table III).

Diagnostic categories

Psychiatric diagnosis according to epilepsy subtypes is shown in Table IV. The majority of patients who received a psychiatric diagnosis were thought to be either cases of anxiety or depressive neuroses. Other diagnostic categories made up less than 24% of the psychiatric cases, or less than 15% of the whole sample. Anxiety neurosis was evenly distributed among the epilepsy sub-categories, but there were more patients with depressive neurosis in the TLE group compared with those with focal non-TLE and PGE.

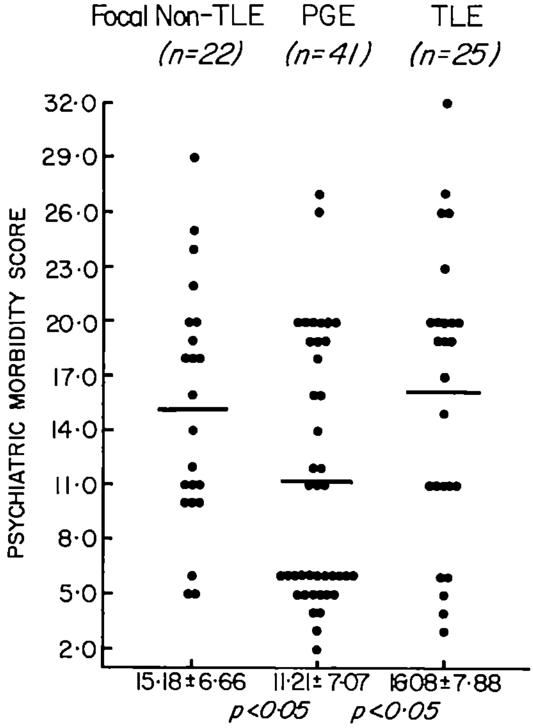

FIG. 1 Distribution of total CIS scores for the epilepsy sub-categories. Means and standard deviations are given at the base of each column, and the means are also indicated by a rule on the graph.

Discussion

The high prevalence of psychiatric morbidity in epilepsy has been frequently reported; although Pond & Bidwell (1959) reported a prevalence of 29% in general practice, Pond (1981) criticised this figure as an underestimate. It represents only conspicuous psychiatric morbidity (Kessel, 1960), and is consistent with the proportion of cases in the current survey (30.7%) known to have had previous out-patient psychiatric referral. With the use of the CIS, 42 (47.7%) of the sample emerged as 'cases', and according to clinical diagnosis, three-quarters had some form of affective neurosis.

The assertion (Gibbs, 1951) that TLE is more associated with increased psychiatric morbidity than other forms of epilepsy provoked a controversy that has persisted until the present day. It is possible to attribute at least some of this disagreement to methodological shortcomings. In particular, the

TABLE III
Type of epilepsy and severity of psychiatric disturbance

	*Severity of psychiatric disturbance**	*PGE (n=41)*	*Focal non-TLE (n=22)*	*TLE (n=25)*	*Total*
0	Normal	21 (51%)	3 (14%)	5 (20%)	29
1	Sub-clinical	5 (12%)	7 (32%)	5 (20%)	17
2	Mild	13 (32%)	8 (36%)	10 (40%)	31
3	Moderate	2 (5%)	3 (14%)	4 (16%)	9
4	Severe	0	1 (4%)	1 (4%)	2

*The following comparisons were made of the size of the normal group compared with the size of the group comprising severities 1–4: PGE versus focal non-TLE ($\chi^2=7.05$, $P<0.01$); PGE versus TLE ($\chi^2=5.10$; $P<0.05$); focal non-TLE versus TLE (NS) (Fisher's exact test).

TABLE IV
Type of epilepsy and psychiatric diagnosis

Diagnostic category	*PGE*		*Focal non-TLE*		*TLE*		*Total*
	n	*%*	*n*	*%*	*n*	*%*	
Normal	26	63	10	46	10	40	46
Anxiety neurosis	6	15	3	14	4	16	13
Depressive neurosis	6	15	4	18	9	36	19
Other neurosis	1	2	0	0	0	0	1
Schizophrenia	0	0	1	5	0	0	1
Affective psychosis	0	0	0	0	1	4	1
Organic psychosis	0	0	1	5	1	4	2
Personality disorder	2	5	0	0	0	0	2
Mental subnormality	0	0	3	14	0	0	3
Total	41		22		25		88

great majority of studies have reported on hospital-based epileptic populations, but it has become increasingly apparent that such samples are unrepresentative. This observation is borne out by data from our own study: partial epilepsy with complex seizures was significantly over-represented among hospital clinic patients compared with clinic non-attenders (i.e. patients managed by general practitioners) (Edeh, 1984). Psychiatric morbidity, using either CIS scores or caseness as criteria, was greater in the first group, and we have attempted to avoid this error by studying an unselected community population.

Nevertheless, there are limitations in the study design. Unlike the survey of 14 general practices (Pond & Bidwell, 1959; Pond *et al*, 1960) which sampled over a wide area and from both urban and rural districts, this study was limited to South London; a wider spread would have been desirable, but was not possible for administrative reasons. Even so, a balance between inner-city (Brixton) and outer suburbs (Beckenham and Croydon) was achieved which seems fairly representative of the metropolitan population. The point-prevalence (3.45/1000) is comparable with other studies that used similar inclusion criteria (Kurland, 1959; Gudmundsson, 1966; Hopkins & Scambler, 1977), and suggests that ascertainment was reasonably complete. The clinical characteristics, in so far as they could be determined, of the minority of patients who refused to co-operate with our survey did not clearly differ from those studied.

The methodology of clinical assessment has also generated controversy. Criticism has been levelled at the use of instruments that fail to distinguish between state and trait and between the evaluation of abnormal mental states and the assessment of personality structure. In this study the emphasis was on clinical assessment, and the CIS was chosen because it combines a careful but unstructured enquiry into the patient's past history with a detailed and systematic assessment of current symptomatology and mental state.

The findings suggest a greater prevalence of psychiatric morbidity in the focal group, especially those with temporal lobe foci, than in the PGE group. This is so when comparison is based on an unstructured account of past and present psychiatric illness, and also when current symptoms and manifest abnormalities are compared. The TLE group was rated more highly than the focal non-TLE lobe group on measures of anxiety and depressed affect and more than the PGE group on irritability, depression, depressed affect, and impaired concentration. In terms of global severity of psychiatric disturbance, the focal groups did not differ; each scored more highly than the PGE group.

How may the increased psychopathology in the temporal lobe group be explained? Structural brain damage, as demonstrated by abnormal CT scan appearances, was common in the focal epileptic groups (there was, across the groups, an insignificant trend towards increased psychiatric morbidity among patients with CT scan abnormalities); seizure control was poorer. In neither instance were the comparisons between the TLE and PGE groups significant.

There have been few community studies of psychiatric morbidity in epilepsy, and our findings are not easily compared with those of Pond & Bidwell (1959), the other British study. Their choice of broad inclusion criteria resulted in prevalence figures that were unusually high (6.2/1000, almost twice that of the present study). Only 39 of 245 patients (an unexpectedly small proportion) were thought to have TLE, and these mainly on clinical grounds, as few had had EEGs; 20 were thought to have "serious psychological disorders", but only 11 showed the "typical slowness, stickiness, moodiness and psychotic tendencies that constitute the so called epileptic personality". Eight (20%) had already been in mental hospitals, compared with 7% for the group as a whole – a figure itself considerably higher than in the general population.

Our study did not employ specific measures of personality structure, such as had been designed by Bear & Fedio (1977), who claimed that "a consistent change in behaviour, thought and affect appeared to be the specific consequence of a temporal epileptic focus". That assertion, then supported only by a comparison of temporal lobe epileptics with a group of patients with peripheral neurological disturbance, would appear premature. Other workers (Masters *et al*, 1984) comparing temporal lobe with other forms of epilepsy, psychiatric illness, and normal control groups failed to detect trait characteristics of the temporal lobe group. In the present study, two patients were thought to have personality disorders; both were diagnosed as having PGE.

The majority of 'cases' were thought to have a minor affective disturbance, usually of an anxious or depressive nature; this is in line with other recent published work from this country (Standage & Fenton, 1975; Robertson & Trimble, 1983). Earlier American studies gave greater prominence to psychosis (Ervin *et al*, 1955; Stevens, 1966) – a discrepancy partly explained by international variations in selection and case-finding criteria.

Our findings suggest, therefore, rather than a distinction between temporal lobe and non-temporal lobe epilepsy, a separation between focal epilepsy, regardless of site of focus, and PGE. With respect to the percentage of subjects who had received psychiatric treatment and to the global psychiatric morbidity scores, the two focal groups fared similarly. The reasons for the differences between focal and primary generalised epilepsy are not clear. The presence of brain damage, implicit in the diagnosis of focal epilepsy and further demonstrated by CT scanning, the relatively poor seizure control, and even the wider prescription of drugs, may each be regarded as a stress factor, insignificant in isolation but exerting a cumulative effect. Rodin *et al* (1976) have suggested a multifactorial explanation for the behavioural change in patients with TLE, particularly the high prevalence of multiple drug prescription and multiple seizures: these data provide qualified support for their view.

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Response of Depressive Symptoms to Nortriptyline, Phenelzine and Placebo

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The effects of nortriptyline, phenelzine, and placebo on 13 symptoms of depression were compared in 75 patients, aged 55 or over, who were suffering from major depression. Nortriptyline and phenelzine were more effective than placebo in treating depression mood, guilt feelings, suicidal ideation, agitation, anxiety, loss of energy, and a.m. diurnal variation of mood. Nortriptyline was better than phenelzine or placebo in improving middle/late insomnia. Most of the symptoms did not show significant improvement until the fourth week of treatment.

Many of the studies dealing with the efficacy of antidepressants have expressed clinical improvement in terms of global scores and scales. These measures often rate changes in the depressive symptom complex *en masse* without taking into account whether a particular symptom is more or less responsive to a particular drug. While these global measures may accurately reflect overall changes in the patient's condition, on an individual basis, a patient may feel better in most respects yet may be bothered by a particular symptom that persists, or may be so pained by a symptom that its resolution is more urgent than treatment of the remainder of the depressive syndrome, which will improve in the deliberate way that often characterises antidepressant response. Although there have been studies examining the ability of certain symptoms to predict antidepressant response, there may be a difference between those symptoms that are useful in predicting response and those that are actually improved during treatment (Overall, 1975). Nelson *et al* (1984) correlated the improvement of items on the Hamilton Rating Scale for Depression with plasma levels of desipramine in an attempt to select symptoms that were prime indicators of the antidepressant effect. Improvement in symptoms such as depressed mood, hopelessness, guilt feelings, middle/late insomnia, loss of energy, somatic anxiety, and loss of appetite were felt to best indicate that the drug was working. However, this study did not have a placebo control, and other items that were not correlated with desipramine levels also improved over time. Other studies (Raskin *et al*, 1970; Montgomery & Asberg, 1979; Bech *et al*, 1981) have also arrived at lists of symptoms that are contended to resolve in close parallel with the action of the antidepressant. Common among the lists are depressed mood, anxiety, guilt feelings, decreased interest, and loss of energy.

In addition to the search for particular symptoms that are especially responsive to antidepressants, there have been efforts to see whether certain symptoms are more responsive to either of the two major groups of antidepressants, tricyclics and monoamine oxidase (MAO) inhibitors. Some reports have stated that tricyclics are the better treatment for endogenous symptoms (anhedonia, loss of appetite, late insomnia, a.m. diurnal variation, retardation), while MAO inhibitors are the treatment of choice for anxiety and other 'atypical' or non-endogenous symptoms (initial insomnia, increased appetite, p.m. diurnal variation, mood reactivity) (Ravaris *et al*, 1980; Nies, 1984). Others have found no difference between those symptoms that improved by tricyclics and those that improved by MAO inhibitors (Kay *et al*, 1973; Paykel *et al*, 1982).

A third issue that has not been well studied is the chronology of symptom resolution. The clinical improvement with antidepressants is often not an all-or-nothing phenomenon; symptoms often resolve at different rates. Perhaps there is a predictable sequence of symptom resolution with antidepressants in general, or with a particular class of antidepressant. This would be helpful in optimising the selection of an antidepressant for a particular patient.

Previously (Georgotas *et al*, 1986), we reported on the comparative efficacy of nortriptyline, phenelzine and placebo in depressed elderly in terms of global scores and scales. We found that both nortriptyline and phenelzine were significantly better than placebo, but not significantly different from each other. The outcome measures used were the total score on the Hamilton Rating Scale, the total score on the Zung

Self-Rating Scale, and Clinical Global Impressions Scale. The present report uses individual items of the Hamilton scale to examine which symptoms of depression are best treated with antidepressants as compared with placebo, whether some symptoms are better treated with either nortriptyline or phenelzine, and what is the order of symptom improvement.

Method

The study was conducted at the Depression Studies Program of New York University, an outpatient research facility for the study and treatment of affective disorders. Men and women, 55 years of age and above, complaining of depressive symptoms were evaluated for entry into this study. During the initial screening, the treatment plan and study requirements were fully explained, and written informed consent was secured. Patients included in the study were independently diagnosed by two psychiatrists as suffering from a major depressive disorder as defined by the Research Diagnostic Criteria (RDC) (Spitzer *et al*, 1977). A score of 16 or greater on the 21-item Hamilton Rating Scale for Depression (Hamilton, 1967) was also required for inclusion. Patients were excluded if they showed evidence of moderate to severe dementia, drug or alcohol dependence, a serious neurological disorder, or other pre-existing major psychiatric disorders. None of the patients accepted had psychotic features.

The patients were subsequently given a complete physical examination, ECG, and clinical laboratory evaluations to rule out active medical problems. Patients with chronic illnesses under control were included provided that they met the other criteria and had obtained clearance from their personal physicians. All patients accepted for the study were placed on dietary restrictions for MAO inhibitors.

Patients who met the entry criteria were administered placebo during a single-blind washout period of at least seven days. All antidepressants had been discontinued for at least one week prior to the placebo washout period. None of the patients had been treated with neuroleptics, lithium, carbamazepine, or MAO inhibitors during the year prior to this study. Any patient not meeting the entry criteria at the end of the washout period was not admitted to the double-blind portion of the study, which lasted seven weeks. Patients who were admitted were randomly assigned to one of three treatment conditions: nortriptyline (NT), phenelzine (PE), or placebo (PL). All three substances were in identical capsules which contained either 25 mg of NT, 15 mg of PE, or an inert substance. The initial dose of one capsule for three days was increased to two capsules on days 4–7. At the end of the first treatment week, the daily dose was increased to three capsules. Blood samples obtained at the end of week 2 were assayed for either NT plasma level or MAO platelet inhibition. Patients on NT who attained a plasma level in the range 50–160 ng/ml remained on three capsules. In patients whose levels were below this therapeutic window, the dosage was raised to four capsules, and if, on the basis of the sample taken at the end of treatment week 3, therapeutic levels had still not been achieved, the dose was raised to five capsules. A similar regimen was followed for patients on PE: those who did not attain a 70% or greater MAO inhibition by week 2 were prescribed a dose of four capsules, and the dose was raised to five capsules if the specified percentage MAO inhibition had not been achieved by week 3. The dose of placebo was also adjusted. In the event of side effects which interfered with functioning to a significant degree, dosage was decreased, irrespective of percentage platelet MAO inhibition or NT level. The doses were adjusted by the non-blind psychiatrist.

Patients completing fewer than 28 days of treatment for any reason were excluded from the final data analysis, except when significant drug-related adverse reactions required withdrawal of medication, or when there was a significant deterioration of clinical condition in any patient treated for at least 14 days. These patients were not included in the analysis because we felt that their treatment had not been given an adequate trial for determination of its efficacy or safety. All patients completing at least 28 days of active drug treatment were included in the final data analysis, except for those who failed to achieve NT levels within therapeutic range (50–160 ng/ml) or platelet MAO inhibition greater than 70% for at least two weeks, and those who were removed from the study due to other reasons not related to psychiatric pathology or study drugs, such as intercurrent illness, evidence of noncompliance, or alcohol or drug abuse. The therapeutic range for NT of 50–160 ng/ml was chosen based on the work of Sørensen *et al* (1978), and on our own previous study (Georgotas *et al*, 1983), which found that this range is applicable to the elderly. Previous studies (Robinson *et al*, 1978; Georgotas *et al*, 1981) have found optimal response with MAO inhibition ≥80%, and this was our aim in this study; however, in our recent experience with elderly patients, some cannot tolerate doses necessary for ≥80% inhibition and yet have a satisfactory response to PE with platelet MAO inhibition 70–80%.

At the end of the placebo washout period and during each weekly visit of the seven-week treatment period, patients were rated on the Hamilton Rating Scale by a psychiatrist blind to the patient's treatment condition. The symptoms to be studied were extracted from items on the Hamilton scale. The score of each item was taken as a quantitative measure of the severity of a symptom. In some cases, a symptom was represented by a combination of two Hamilton items. The 13 symptoms studied and their corresponding item(s) on the Hamilton scale were the following: depressed mood, 1; guilt feelings, 2; suicidal ideation, 3; initial insomnia, 4; middle/late insomnia, 5+6; retardation, 8; agitation, 9; psychic/somatic anxiety 10+11; appetite disturbance, 12; loss of energy, 13; weight loss, 16; and diurnal variation of mood – both a.m. and p.m., 18. The differences in effect of the three treatment groups on these symptoms were investigated using a multiple regression analysis with a repeated measures design as described by Pedhazur (1982). In this method, the criterion variable (depressive symptom) was regressed on the predictor variable (treatment) for all weeks of treatment. The pretreatment Baseline score for each criterion variable was partialled out of the analysis. A vector representing subjects by the means of their criterion variable over the treatment period was then entered into the regression equation. Consequently, the degrees of freedom associated with the subjects' vector and with the residual sum of squares were adjusted in determining the *F* ratio for testing statistical significance. Week-by-week comparisons of the Hamilton items used an analysis of covariance (ANCOVA) with the pretreatment Baseline score as the covariate.

Results

Patient sample

The double-blind treatment phase was completed by 90 patients, but only 75 were included in the data analysis. The reasons for excluding 15 patients from the data analysis were as follows: three (1 NT, 2 PE) did not comply with the protocol, two (1 PL, 1 PE) were terminated after a few days because of failure to obtain medical clearance for pre-existing cardiac problems, one (PE) was hospitalised within the first week because of clinical deterioration, five (1 PL, 2 NT, 2 PE) withdrew for reasons unrelated to treatment after two weeks or less and without having reached adequate NT levels or percentage platelet MAO inhibition, one (PE) was excluded for not having reached adequate percentage platelet MAO inhibition after seven weeks, one (PE) terminated after one week because of clinical improvement, and two (PE) self-terminated after one and three weeks because of failure to improve.

Of the 75 patients who met inclusion criteria for data analysis, 56 finished seven weeks of treatment. Reasons for the premature termination of 19 patients were as follows: 12 (11 PL, 1 NT) terminated because of clinical deterioration, three (1 PL, 2 PE) terminated because of clinical improvement, and four (2 NT, 2 PE) terminated early due to side effects.

Patients in the NT group had mean NT levels after week 2 in the range 82.6–95.3 ng/ml; the mean values of platelet MAO inhibition for phenelzine-treated patients were 68.3–81.1% after week 2, and exceeded 80% after week 5.

Patients were discouraged from taking other psychotropic medication during the study; however, 18 patients (24%) took small doses of sedatives/hypnotics for brief periods: seven PL patients (1 chloral hydrate, 3 flurazepam HCl, 2 temazepam, 1 chlordiazepoxide HCl), six NT patients (1 chloral hydrate, 2 lorazepam, 2 temazepam, 1 flurazepam HCl), and five PE patients (2 flurazepam HCl, 3 temazepam).

Overall treatment effect

Repeated measures analyses were performed comparing changes in the scores of selected items from the 21-item Hamilton Rating Scale over the seven-week treatment period among the three treatment groups; the pretreatment Baseline score for each item was partialled out of the analyses. The items investigated were the following: depressed mood, guilt feelings, suicidal ideation, initial insomnia, middle/late insomnia, retardation, agitation, psychic/somatic anxiety, appetite disturbance, loss of energy, weight loss, and diurnal variation—both a.m. and p.m. Items from the Hamilton scale representing depressed mood, guilt feelings, suicidal ideation, middle/late insomnia, agitation, anxiety, loss of energy, and a.m. diurnal variation had significant ($P < 0.05$) overall treatment effect, signifying that there were significant differences among the nortriptyline, phenelzine, and placebo groups in the outcome of these symptoms. No significant treatment effect was found for initial insomnia, retardation, weight loss, or p.m. diurnal variation, indicating that changes in these symptoms during the seven weeks were not significantly different among the three treatment groups. Pairwise comparisons of those items with significant overall effects were performed using a similar repeated measures model. Both nortriptyline and phenelzine were found to be significantly superior ($P < 0.05$) to placebo in reducing the scores on the Hamilton items for depressed mood, guilt feelings, suicidal ideation, agitation, anxiety, loss of energy, and a.m. diurnal variation of mood. No significant treatment effect on these items was found in comparing nortriptyline-treated patients with phenelzine-treated patients. However, nortriptyline was significantly better ($P < 0.05$) than either phenelzine or placebo in alleviating middle/late insomnia over the seven-week treatment period.

In summary, nortriptyline and phenelzine were significantly more effective than placebo in treating depressed mood, guilt feelings, suicidal ideation, agitation, anxiety, loss of energy, and a.m. diurnal variation of mood. Nortriptyline was significantly better than either phenelzine or placebo in treating middle/late insomnia. No significant differences were found in the effects that nortriptyline, phenelzine, and placebo had on the symptoms of retardation, appetite disturbance, weight loss, and p.m. diurnal variation of mood.

Chronology of symptom resolution

For those items on the Hamilton scale that had a significant overall treatment effect (depressed mood, guilt feelings, suicidal ideation, middle/late insomnia, agitation, psychic/somatic anxiety, loss of energy, and a.m. diurnal variation), weekly ANCOVAs, partialling out the Baseline score, were performed. The purpose of this was to compare the effects of the three treatments on a particular symptom on a week-by-week basis during the seven weeks of treatment. A statistically significant overall treatment effect ($P < 0.05$) at a particular week was followed by pairwise ANCOVAs. One of the first symptoms to show significant improvement was suicidal ideation. Nortriptyline patients had significant improvement in suicidal ideation as compared with placebo patients at week 2 ($P = 0.002$), and phenelzine patients had significant improvement in this symptom as compared with placebo patients at week 3 ($P = 0.014$). The next symptoms to be resolved were guilt feelings, psychic/somatic anxiety, and loss of energy. At week 4, patients treated with either nortriptyline or phenelzine had significant improvement ($P \leq 0.01$) in these symptoms as compared with patients treated with placebo. Not until week 5 was depressed mood significantly better ($P \leq 0.01$) in antidepressant-treated patients as compared with placebo patients. Nortriptyline-treated patients showed significant improvement ($P < 0.01$ in middle/late insomnia beginning at week 5; at no time were phenelzine-treated patients significantly better than placebo with regard to this symptom. The treatment effect on a.m. diurnal variation did not become significant until week 7, when those patients treated with either nortriptyline or phenelzine had significant resolution ($P<0.05$) of this symptom over patients treated with placebo. The above results are summarised in Table I.

In summary, with the exception of suicidal ideation, which had significant improvement beginning at week 2, the symptoms began showing significant treatment changes at week 4. For most of the Hamilton items, nortriptyline and phenelzine were significantly more effective than placebo, and both drugs became effective at the same time. The exceptions were suicidal ideation, for which the improvement over placebo became evident one week earlier in the nortriptyline group than in the phenelzine group, and middle/late insomnia, which responded significantly better with nortriptyline than with either phenelzine or placebo.

TABLE I
Chronology of improvement of depressive symptoms

Symptoms	*Week at which improvement became significant**
Suicidal ideation	2 (NT>PL), 3 (PE>PL)
Guilt feelings	4 (NT and PE>PL)
Anxiety	4 (NT and PE>PL)
Loss of energy	4 (NT and PE>PL)
Depressed mood	5 (NT and PE>PL)
Middle/late insomnia	5 (NT>PL), 7 (NT>PE)
Agitation	6 (NT and PE>PL)
Diurnal variation, a.m.	7 (NT and PE>PL)

*$P<0.05$ by ANCOVA with Baseline value as covariate.

Discussion

In the above study, the effects of nortriptyline, phenelzine and placebo on 13 symptoms of depression were investigated. The symptoms of depressed mood, guilt feelings, suicidal ideation, middle/late insomnia, agitation, anxiety, loss of energy, and a.m. diurnal variation showed significant improvement in patients treated with antidepressants as compared with patients treated with placebo. No significant difference in treatment response was found in the symptoms of initial insomnia, retardation, weight loss, and p.m. diurnal variation. That the former nine symptoms responded better to antidepressants than to placebo is not surprising and, in fact, is an elaboration on our earlier findings (Georgotas *et al*, 1986). The lack of a difference in response for the other four symptoms may reflect several things. First, although many of our patients complained of weight loss prior to entering our study, very few of our patients actually lost weight during the seven weeks of treatment, even if they were not clinical responders. This may explain the failure to find a significant treatment difference in weight loss. Although patients were generally forbidden to use other psychiatric drugs during the study, in some cases of severe insomnia, patients were given mild hypnotics for short-term use; in addition, some patients had long-standing complaints of initial insomnia that antedated the onset of illness and, in fact, some patients who responded still complained of difficulty falling asleep. These may explain the lack of a statistically significant difference in the response of initial insomnia to the three treatments. The failure of retardation and p.m. diurnal variation to respond differently may be due either to methodological limitations or to the lack of an actual drug/placebo difference.

When the responses to nortriptyline and to phenelzine were compared, eight of the nine symptoms studied were treated with equal efficacy by either drug. The effect of nortriptyline on symptoms appeared to be similar to that of phenelzine, regardless of whether the symptoms were endogenous or non-endogenous. The exception was middle/late insomnia, which was significantly improved by nortryptyline as compared with phenelzine. Except for the latter symptoms, these results corroborate other studies (Kay *et al*, 1973; Paykel *et al*, 1982) that found no difference in symptom response between tricyclics and MAO inhibitors. Even the purported selective anti-anxiety effect of MAO inhibitors (Ravaris *et al*, 1980) was not supported by this study. Other things being equal, nortriptyline and phenelzine appear to be equally effective in the general treatment of major depression, with nortriptyline better than phenelzine in improving middle/late insomnia.

Most of the symptoms of depression did not show significant improvement until the fourth week of treatment. The exception was suicidal ideation, which improved by the second week in nortriptyline patients and in the third week in phenelzine patients. Otherwise, the order of symptom resolution was similar for both drugs. There did not appear to be differences in the time of resolution between, say, psychic versus somatic symptoms, or endogenous versus non-endogenous symptoms. It should be emphasised, however, that although differences between drug and placebo patients began to appear at week 2, some symptoms such as agitation and a.m. diurnal variation were not significantly improved until the sixth or seventh week of treatment. Thus, in addition to the lag period before the onset of antidepressant action, the actual time to complete recovery from the onset of action may be as long as a month.

The above findings emphasise again that both nortriptyline and phenelzine are effective in treating major depression in depressed patients over the age of 55. Except for nortriptyline's superior effect on middle/late insomnia, neither drug appears to be especially selective in relieving certain symptoms of depression. In addition, the resolution of the symptom complex may be stretched over a period of four or five weeks. Given the usefulness these results have in adjusting our expectations of the effects of antidepressants, further research is needed to refine this information into clinically useful guidelines.

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Anorgasmia from Clomipramine in Obsessive-Compulsive Disorder

A Controlled Trial

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Forty-six patients with obsessive-compulsive disorder undergoing a double-blind controlled study of clomipramine and placebo were interviewed to assess changes in sexual function. Of 33 patients with previously normal orgasm, nearly all of the 24 on clomipramine developed total or partial anorgasmia; none of the 9 on placebo did so. Anorgasmia persisted with minimal tolerance over the five months that clomipramine was taken. Men and women were equally affected. Sexual side-effects are easily missed without a structured interview, and can detract from the value of drug treatment.

It has been known for many years that psychotropic drugs can disrupt sexual function (Singh, 1961; Shader, 1964; Blair & Simpson, 1966). Many case reports have implicated tricyclic antidepressants (TCAs), and in particular clomipramine, as a cause of low libido, delayed or absent orgasm, and absent or retrograde ejaculation (dry orgasm) (Beaumont, 1973, 1977; Couper-Smartt & Rodham, 1973; Nininger, 1978; Thoren *et al*, 1980; Yassa, 1982; Quirk & Einarson, 1982; Sovner, 1983). Premature ejaculation is sometimes treated with TCAs (Eaton, 1973; Correa & Traldi, 1977; Abraham & Porto, 1978), although a controlled investigation of clomipramine did not show a useful effect (Goodman, 1980).

The pathophysiology of these effects is not well understood. Various sites of action have been implicated, both central (serotonergic or prolactinaemic) and peripheral (anticholinergic, or alpha-adrenergic blocking) (Beaumont, 1973; Everitt, 1977; Barnes *et al*, 1979; Kulik & Wilbur, 1982; Sovner, 1983; Shen, 1983).

Little is known about the incidence of sexual side-effects of drugs. In an uncontrolled study of 51 depressed patients, 65% of whom had prior sexual dysfunction, a low dose of clomipramine adversely affected sexual function in 20% and improved it in 50% (Beaumont, 1977). Manufacturers' reports and pharmacological textbooks suggest that sexual side-effects from antidepressants are minor. Controlled studies of clomipramine for obsessive-compulsive disorder (OCD) confirm this impression (Marks *et al*, 1980; Ananth, 1980; Insel *et al*, 1983), although Thoren *et al* (1980) do mention anorgasmia as a side-effect.

During a replication of a double blind controlled study comparing a 35 week course of clomipramine with placebo in OCD, some patients complained about sexual side-effects. This led us to examine all the patients in a structured interview.

Method

Sample

Forty-six patients with severe OCD (ICD-9 (World Health Organization, 1978) and DSM-III (American Psychiatric Association, 1980) criteria) were randomly assigned to clomipramine or to placebo in a 3:1 ratio in a controlled double-blind study of clomipramine and exposure treatment. Of these, 33 (24 on clomipramine, 9 on placebo) had enough prior regular sexual activity with orgasm to enable assessment of the subsequent sexual side-effects of medication; of the 33, 21 (16 on clomipramine, 5 on placebo) had no previous sexual dysfunction.

Procedure

Patients were informed that they would have either drug or placebo, but were not warned of any sexual side-effects. The drug was provided in 25 mg capsules, starting with one per night and increasing by one capsule every three nights to eight capsules per night. Complaints about side-effects were met with reassurance, and any attribution to the drug noted without comment.

Assessments

Patients and assessor were initially blind as to drug condition. A psychiatric interview and a urine screen for psychotropic drugs were administered at the start of the study (week 0). The self-rated Beck Depression Inventory (Beck *et al*, 1974) (13 items, score range 0–39) and the assessor-rated Hamilton Rating Scale for Depression (Hamilton, 1969) (17 items, score range 0–52) were administered in weeks 0 and 8, as was the self-rated Physical Symptom Questionnaire (Marks *et al*, 1980) (12 items, score range 0–36). Drug dose and plasma levels were checked in weeks 4 & 8. The following assessments were made of sexual function: (a) spontaneous complaints of sexual dysfunction; (b) changes in sexual relationships; (c) effect of being tense or being sedated on sexual function; (d) effect of OCD on sexual function; and (e) attribution of sexual dysfunction and of side-effects; these data were recorded whenever complaints arose, and also at week 8.

A structured interview enquired into changes in sexual interest and urge, ability to fantasise, spontaneous sexual fantasy, changes in erection or lubrication, frequency of sex, sexual sensation, length of time to orgasm, and the intensity, enjoyability, occurrence of and ease of orgasm at intercourse and at masturbation, pain at orgasm or when emptying the bladder, and occurrence of dry orgasm. Most patients did not know whether they had sexual dreams, and information on these was left out of the results.

Results

The mean age of the 46 patients was 31 (s.d. 9.4 years). There were 25 men (13 single, 11 married, 1 divorced) and 21 women (9 single, 11 married, 1 divorced).

Sexual function before treatment

This information was obtained at routine assessment, so the dysfunction recorded may be an underestimate. Twenty-one patients (9 men, 12 women) had apparently unproblematic heterosexual sex lives; 16 were put on clomipramine and 5 on placebo.

Sexual problems in the remaining 25 patients (mean age 30, s.d. 9.6 years) are summarised in Table I. For inclusion the shyness, reduced libido, phobia about sex, or guilt had to be sufficient to grossly interfere with their ability to form sexual relationships or have coitus. Sexual deviation is also recorded. The mean age of the 11 virgins was 26 (s.d. 6.9 years). One patient who had not had sex for many years had a wife who had vaginismus and was still a virgin; they refused treatment for this problem. Another patient with low libido and his wife were only able to have sex when drunk. Of these 25, 12 had enough sexual activity to orgasm to be included; eight were put on clomipramine and four on placebo.

Change in sexual function

Marital relationships remained stable. Change in sexual function could not be assessed in 13 of the 46 patients; of these 13, information from one was unreliable, another only had sex after drinking, one had anorgasmia, two had missing data and eight had no sexual activity during the 35 weeks of the trial. The following results come from the remaining 33 patients (mean age 32) who had enough sexual activity to orgasm to allow us to assess the effect of clomipramine. They include the 21 patients with unproblematic sex lives.

Placebo patients ($n = 9$; 4 men, 5 women). Three developed slight sleepiness that interfered minimally with sexual function, and one increased in sexual interest. There were no spontaneous or elicited complaints of change in sexual function or orgasm. They were on a mean of 175 mg (range 100–200 mg) of placebo.

Clomipramine patients ($n = 24$; 17 men, 7 women). The results are summarised in Table II. There were no com-

TABLE I
Analysis of the 25 patients who had sexual problems before treatment began

	Female (n=9)	*Male (n=16)*	*Total*
Virgin	+ + + + +	+ + + + + +	11
In permanent relationships: no sex <4 years	+	+ +	3
Extreme shyness	+ + +	+ + + +	7
Low libido	+ +	+ + + +	6
Anorgasmia	+ + +		3
Previous sex therapy	+		1
Premature ejaculation		+ +	2
Phobic of sex	+	+	2
Extreme guilt		+	1
Erectile problems		+ ?	1
Paedophilia		+ + +	3
Homosexuality		+ +	2
Transvestism	+	+	2
OCD affecting sex function		? ? ? +	1

TABLE II

Sexual side-effects in the 24 previously orgasmic clomipramine patients

	Female (n=7)						*Male (n=17)*																	*Total*
Sleepiness interfering with sex function	+				+																			2
Interest in sex reduced		O		O		+				O				O	O			O	O	O				1(+8)
Frequency of sex/masturbation reduced		O		O		+	+			O								O	O					2(+5)
Erection/lubrication interfered with				O		+									+			O						2(+2)
Sexual sensation reduced			+	O		+		+			+			+				O						5(+2)
Less intense orgasm	+		+			+		+	+		+		+			+		+	+	+	+		+	13
Less satisfaction with orgasm	+			+		+		+	+		+		+			+		+	+	+	+		+	13
Orgasm reduced or absent	*	*	*	*	*	+		+	*	*	*	+	*	*	*	*	*	*	*	*	*	+	*	22
Increased difficulty or time to reach orgasm	+	+	+	+	+	+		+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	22
Pain at orgasm								+	+		+		+											4
Pain emptying bladder							+	+	?															2
Spontaneous complaints of sexual dysfunction		+								+			+	+		+	+		+	+				8
Previous sexual problem or deviation	+						+	+	+		+	+			+			+						8

+ = Problem present
O = Problem the patient considered secondary to disinterest due to anorgasmia
* = No orgasm at all

plaints of sexual dysfunction from sedation or inability to fantasise sexually. Spontaneous sexual thoughts did not change. Eight of the nine patients (7 men, 2 women) reporting decreased interest in sex attributed it to difficulty in achieving orgasm; five of the eight were among the eight who spontaneously complained about drug-induced sexual dysfunction. Seven (4 men, 3 women) patients said they were having less coitus or masturbating less often – five attributed it to difficulty in reaching orgasm. Only 2 of the 17 men reported problems with erection and 2 of the 7 women with lubrication; these effects were slight. Seven patients (4 men, 3 women) reported reduced sexual sensation, two attributing it to disinterest due to anorgasmia.

Of the 24 patients on clomipramine, 22 reported much greater effort, took longer and were often unable to achieve orgasm; 18 of these 22 (13 men, 5 women) were unable to attain orgasm at all even after prolonged effort, although all 24 had been orgasmic previously. One woman could occasionally achieve orgasm with masturbation, but not with coitus, during which she had always been orgasmic previously. The other four (3 men, 1 woman) were intermittently anorgasmic while on the drug. Most (number not recorded) of the 14 in whom clomipramine reduced intensity and enjoyability of orgasm attributed it to the effort involved in reaching it. Four men reported pain at orgasm, and 3 men had pain on micturition which lasted two or three weeks and disappeared although they continued clomipramine. No man reported orgasm without ejaculation (dry orgasm).

The mean dose of clomipramine at the time of interview was 140 mg (range 25–200 mg). Most patients noticed marked difficulty in achieving orgasm within the first few days and often at the first sexual activity after starting the capsules, when on a dose no higher than 25–50 mg. Total anorgasmia was usual after 100–150 mg. Most found it hard work trying to reach orgasm and became frustrated and disinterested in sex. Some wondered if they had developed a permanent sexual problem, and were relieved to discuss this. Only two patients developed any tolerance at all; after 2–3 months they became able with great difficulty to attain orgasm occasionally while remaining on clomipramine. Five patients insisted on reducing and occasionally stopping their drug due to anorgasmia.

Return to normal sexual function occurred within three days of stopping clomipramine in all except one man. Although he had been symptom-free before starting clomipramine he developed slight impotence and loss of libido after drug withdrawal, which improved three months later without treatment. Of the 24 patients on clomipramine only 2 found orgasm unchanged: one refused to take more than 25 mg, as she experienced vague but intolerable side-effects, and the other was a transvestite who had been masturbating

20 times a week; he was asked to reduce this to twice a week and became able to attain orgasm again, despite a dose of 150 mg of clomipramine.

Physical Symptom Questionnaire

Neither the 9 placebo nor the 24 clomipramine patients rated themselves as being significantly different at week 8 compared with week 0, either on the total scores or on the item enquiring about sexual difficulties. Of the ten who reported no side-effects on this self-rated questionnaire, three nevertheless spontaneously complained of anorgasmia, and eight of them reported complete anorgasmia at interview.

Depression ratings

There were no significant differences (*t*-tests), at week 0 or at week 8, between the clomipramine and placebo groups on either the Beck or Hamilton questionnaires.

Laboratory tests

Urine screen at week 0 confirmed that all patients were free of any psychotropic drug. Plasma levels at week 4 and week 8 confirmed that no placebo patient was on clomipramine. In patients on clomipramine, plasma levels of drug and metabolite reached therapeutic levels: clomipramine 97 ng (s.d. 48 ng) at week 4 and 89 ng (s.d. 79 ng) at week 8; and desmethylclomipramine 190 ng (s.d. 102 ng) at week 4 and 225 ng (s.d. 105 ng) at week 8). Nearly every instance of severe anorgasmia began within the first week when plasma levels must have been very low.

Discussion

Sexual function before treatment

We were surprised at the amount of sexual pathology in our obsessive-compulsive patients; 24% of our sample were virgins at a mean age of 26 and another 9% had not had sex for years. We do not know how this proportion compares with that in other anxiety disorders or in normals.

Sexual side-effects of clomipramine

Patients were free of other drugs. Sexual function in the nine placebo patients remained unchanged. Within days of starting clomipramine 92% of the patients (96% of those on more than 25 mg per day) developed great difficulty in achieving orgasm, most to the point of being anorgasmic; 70% became totally anorgasmic by the time their dose of clomipramine had reached a therapeutic level. Men and women were equally affected.

The sexual problem from clomipramine solely concerned orgasmic and ejaculatory capacity, not other aspects of sexual function. Clomipramine did not reduce frequency of spontaneous sexual thoughts, ability to fantasise, or interest in sexual intercourse; patients were able to achieve erection or lubrication normally. Clomipramine-induced anorgasmia led to patients feeling frustrated, and many found sex less enjoyable. A few patients on clomipramine developed pain at orgasm or on micturition. Depression scores in both groups were comparably low before and during treatment, so the anorgasmia after starting clomipramine could not have been due to depressed mood. Sedation was minor and did not affect sexual function.

Tolerance hardly ever developed. The main interview was carried out at week 8, although most patients had also been assessed earlier. In the 6 months that patients were on the drug only 2 of 22 became at all tolerant to it, and even they found that orgasm was more difficult to achieve than when off the drug.

Delay in achieving orgasm began early, possibly by the time of the first sexual activity after starting clomipramine. Return to normal was equally fast, occurring within 1–3 days of stopping the drug. Other reports confirm this (Nininger, 1978; Quirk & Einarson, 1982; Schwarcz, 1982; Shen, 1982). Although it is common knowledge that antidepressants can retard ejaculation it is not generally realised quite how much clomipramine impairs orgasm in both sexes. The extent of this handicap is emphasised by our present findings and substantially diminishes the therapeutic value of clomipramine. It is not surprising that five of our patients reduced and occasionally stopped the drug because of its sexual side-effects. Similar problems might be found on closer study of other antidepressants. If such were found, this would diminish the utility of these drugs.

McLean *et al* (1983) reported orgasm on yawning in two patients on clomipramine. Our sample made no such report during an intensive interview on sexual behaviour, although we did not enquire directly about it.

Assessment of sexual dysfunction in drug trials

Given that 96% of our sample who were all previously orgasmic developed anorgasmia on clomipramine, there have been surprisingly few reports of this side-effect in the many controlled trials of the treatment of obsessive-compulsive, phobic and depressive disorders (reviewed in Marks, 1983). Numerous case reports implicate TCAs and monoamine oxidase inhibitors (Friedman *et al*, 1978; Rapp, 1979; Lesko *et al*, 1982; Christenson, 1983; Pohl, 1983) in sexual dysfunction.

Sexual side-effects are probably common and under-reported in drug trials because they are so easily missed. Most patients report sexual side-effects only when directly and specifically questioned about them, and not on simple questionnaires. The physical symptoms questionnaire that we used to elicit side-effects did not ask directly about orgasm, only that patients should rate 'sexual difficulties' on a 4-point scale from 'no problem' to 'severe'. Clomipramine and placebo patients gave similar answers to this question; 36% of those with drug-induced anorgasmia at intervew did not admit to any sexual difficulty on the questionnaire in spite of their concern about it and even though some were secretly reducing their dose of clomipramine in an attempt to regain their lost orgasmic capacity. In a previous controlled trial (Marks *et al*, 1980) this questionnaire also failed to detect any difference in sexual side-effects between clomipramine and placebo patients. It is evident that more direct and specific questioning is needed to detect these. About a third of patients on clomipramine spontaneously complained of sexual problems, and this prompted us to interview all the patients in more detail.

The failure of patients to report anorgasmia is remarkable. The same failure of reporting is found with thioridazine; 50% of patients in the study of Kotin *et al* (1976) had problems with ejaculation and 33% had dry orgasms, yet in the 17 years that thioridazine had been used prior to that study (12–15 million patients) only 31 cases of ejaculatory failure were reported to the manufacturers and there were only ten published articles reporting 18 cases.

The blindness of assessors to drug condition has been questioned, as the dry mouth of patients on TCAs is immediately evident to the experienced observer (Couper-Smartt & Rodham, 1973; Leyburn, 1967). Nevertheless, in a previous trial examining clomipramine in OCD assessors remained blind to drug condition (Marks *et al*, 1980). However, once alerted to the range of side-effects, including anorgasmia, assessors in the present study were able to guess the patients' drug condition. The patient's blindness, too, must necessarily be questioned in trials of this kind. The problem of maintaining rater blindness will be discussed in more detail in our main report of this trial.

Mechanism of anorgasmia from clomipramine

Identification of the mechanism of anorgasmia from clomipramine might well illuminate the normal physiology of orgasm. Anorgasmia begins within three days (perhaps hours) of starting clomipramine, and returns to normal three days after stopping it. The antidepressant effect takes 12–14 days, as does the uptake of clomipramine into fat; the anorgasmia therefore cannot be due to these factors. Nor is it likely to result from the sedation that can occur 2–3 days after starting clomipramine, because complaints of sleepiness were minimal and patients said this did not affect sexual function. Delayed orgasm is unlikely to be mediated by the central serotonin or noradrenaline reuptake blocking effect of clomipramine (Barnes *et al*, 1979). Although clomipramine has prominent anticholinergic effects, other anticholinergic drugs such as atropine do not necessarily cause anorgasmia even in doses large enough to cause pupil dilatation, tachycardia and extremely dry mouth (Wagner & Brindley, 1980). Moreover, similar anorgasmia is reported with monoamine oxidase inhibitors (Friedman *et al*, 1978; Rapp, 1979; Lesko *et al*, 1982; Christenson, 1983; Pohl, 1983).

Beaumont's (1973) suggestion that alpha-adrenergic blockade causes anorgasmia is probably wrong. Both phenoxybenzamine (Wagner & Brindley, 1980) and phentolamine (Brindley, personal communication) cause failure of external ejaculation (dry orgasm) but have no significant effect on the ease with which orgasm can be produced. Occasional case reports suggest that desipramine (which is largely noradrenergic) may not block orgasm in the way that many other TCAs do (Quirk & Einarson, 1982; Sovner, 1983); it does not have much anticholinergic, alpha-adrenergic blocking or serotonergic activity. We do not know how other TCAs affect sexual function. Clomipramine-induced anorgasmia does not result from reduced genital sensation, which patients who had delayed or absent orgasm reported to be negligible.

Evaluation of the literature of sexual dysfunction due to drugs would be easier if reports specified which aspect was disrupted, e.g. libido, orgasm or ejaculation, and whether a control group was included in the study.

In conclusion, a structured interview revealed that anorgasmia became the norm, not the exception, in obsessive-compulsive men and women on clomipramine.

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Outcome in Bipolar Affective Disorder After Stereotactic Tractotomy

Nine patients have been treated by subcaudate stereotactic tractotomy for bipolar affective disorder resistant to drug treatments. In the majority, after the operation there was a reduction in frequency and severity of depressive and manic episodes. There was a trend for the operation to have more effect on the manic than on the depressive phases. Drugs which had been inert previously sometimes became therapeutically useful after surgery.

The history and development of psychosurgery has been reviewed by Bridges & Bartlett (1977). Current techniques bear little resemblance to earlier methods. Side-effects are now unusual, and changes in personality are rare. The main indication for subcaudate stereotactic tractotomy (SST) has been unipolar affective disorder, and there has been some doubt about the suitability of this operation for patients suffering from bipolar illness. The main concern was that if there were any tendency to disinhibition after the operation, this might result in a shift of the illness towards hypomania (Bartlett *et al*, 1981).

We present nine case studies of patients suffering from bipolar illness, who were referred from Whitchurch Hospital, Cardiff to the Brook Hospital, London, where they underwent SST. Six of these have been observed for at least seven years following the operation. The patients had long histories of well defined phasic manic and depressive disturbances in mood. All were severely incapacitated by the disorder and were failing to benefit from the treatments available at the time and, in particular, no longer responded to lithium. Some had reached the stage of rapid recurrences with no or minimal periods in remission. In each case the uncertainty about the outcome was explained to patients and relatives.

Case reports

The patterns of illness for each patient are summarised in Fig. 1.

Patient 1 (Female, born 1931) The patient's first episode of depression followed the birth of her second child; the second followed a partial thyroidectomy. After two years of good health she relapsed into a mixed affective state. Treatment was only partially effective, and the development of rapid cycling illness led to SST (eight seeds bilaterally) being performed. One year after the operation she went into a complete remission for six years. All this point she became hypomanic, and a year later was prescribed lithium for a further attack. A recent depressive episode has responded well to a tricyclic antidepressant. None of the postoperative episodes have been as severe as those before surgery.

Patient 2 (Female, born 1917) This patient had a puerperal depression preceeded by a history of minor mood lability. She then developed recurrent depressive episodes, with one mild episode of hypomania; her first definite manic episode occurred at the age of 57. Since treatments became increasingly ineffective, she underwent SST (seven seeds bilaterally). In the first two years after the operation she had five episodes of depression, and she was referred for an extension. This was prevented by her poor respiratory function. However, the time spent in illness over the next two years reduced, and her depression was attenuated and is now responsive to tricyclic antidepressants.

Patient 3 (Female, born 1907) This patient had her first depressive episode when aged 53; 14 years later, following her first episode of hypomania, she developed a rapid cycling mood disorder and at the time of SST (five seeds bilaterally) she was continuously ill. After the operation she had mild depressive episodes about once per year, which responded to mianserin. She was on lithium until the age of 75, when this was changed to carbamazepine on account of drug induced tremor. Since SST there has been a reduction in severity and frequency in depressive episodes; a hypomanic episode did not recur for six years.

Patient 4 (Female, born 1921) Following the death of her parents, this patient suffered from frequent episodes of mania and depression, and SST (eight seeds bilaterally) was performed when she was 56 years old. The lesion was extended four years later (four seeds bilaterally). Following the first operation, hypomanic phases became less severe but depressive episodes continued with unabated severity, and remained unresponsive to drugs. Following extension of the operation, she developed continuous partial epilepsy. The computerised tomography scan showed embolic damage in the right frontal region; this was attributed to earlier embolic episodes due to mitral valve disease. The epilepsy was controlled, but rapid cycling of mood continued, although the manic episodes were less severe. She had a left-sided stroke eighteen months after the second operation.

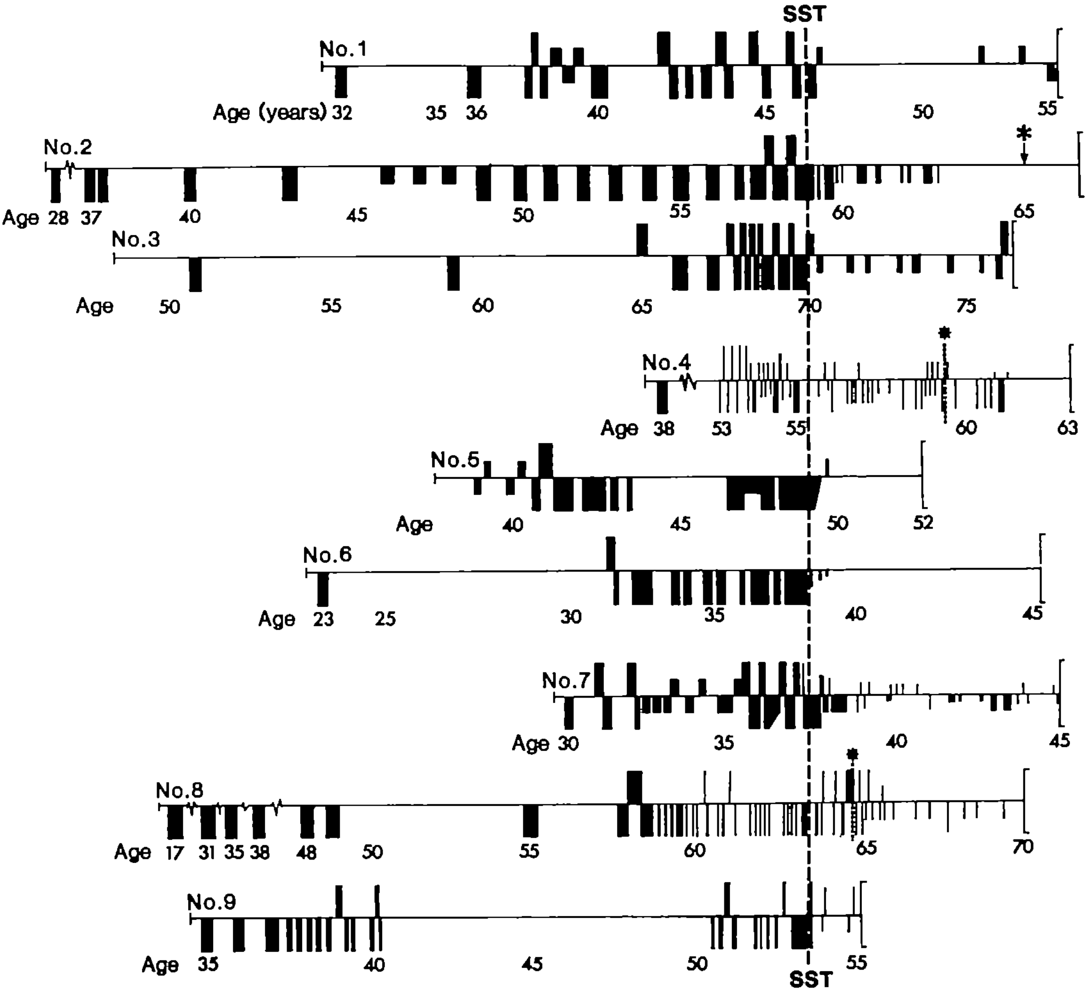

FIG. 1 Follow-up of nine patients. The time axis is in years, and represents the age of the patient. Entries above the axis represent manic episodes, and those below the axis depressive episodes; the degree of mania or depression is indicated by the height or depth of the marker. An asterisk shows the time of death; a star marks the time of extension of subcaudate tractotomy.

Patient 5 (Male, born 1933) This patient developed mood swings following a two-year history of anxiety symptoms. Because of his anxiety he was prescribed increasingly larger doses of chlorpromazine; attempts to withdraw this precipitated depressive episodes. Although the mood disorder was resistant to treatment, behaviour therapy partially controlled his phobic symptoms. Following two years of almost continuous depression, he underwent SST (ten seeds bilaterally). One year after surgery he was free from anxiety, phobic or affective symptoms. He has remained well to the time of follow-up.

Patient 6 (Female, born 1940) This patient's first depression followed the birth of her only child. Eight years later she developed a cyclical mood disorder which, despite treatment, progressed into almost continuous illness for the 18 months before SST (eight seeds bilaterally). Since the operation she has been free of major mood disorder, although after the death of her mother and the birth of a grandchild she developed a phantom pregnancy of nine months duration, which resolved spontaneously.

Patient 7 (Male, born 1940) This patient developed depression following the break-up of a homosexual relationship. He then had mood swings of increasing frequency, resistant to all treatment. Following SST (eight seeds bilaterally) manic phases were less severe, but depressive phases continued unabated. These have since responded to a combination of lithium, clomipramine and tryptophan. The substitution of carbamazepine for lithium has been beneficial. Stopping medication precipitated a further depressive episode.

Patient 8 (Female, born 1914) This patient's first depressive episode occurred after she failed her school certificate. Further depressive episodes occurred periodically until the age of 58 years, when she developed rapidly cycling affective disorder. Although treatment of acute episodes was partially successful they occurred with increasing frequency, and prophylaxis was impossible because of lithium intolerance. Continuation treatment with antidepressants was unhelpful. The SST (eight seeds bilaterally) had little effect, and an extension was performed a year later (left side four seeds, right side six seeds). She continued to have mood swings, but these now proved responsive to previously ineffective medication. In the past three years carbamazepine has increased mood stability.

Patient 9 (Female, born 1931) This patient developed agitated depression following a subarachnoid haemorrhage. After a time of rapid cycling she responded to a combination of a monoamine oxidase inhibitor, a tricyclic antidepressant and lithium, which was discontinued on admission to a general surgical ward. She then redeveloped resistant rapid cycling. The SST (ten seeds bilaterally) was performed when she was 54 years old, and follow-up is limited. Since the operation she has had five periods of illness, some of which were accompanied by persecutory delusions and auditory hallucinations. In the patient's opinion episodes of illness have been reduced in frequency and severity; in particular, the depression has been relieved more than the hypomania. However, before SST the patient never experienced first rank symptoms. The possibility of SST precipitating schizophrenic psychosis must be considered.

Discussion

Case studies of the type described here have a number of limitations. Data collection is retrospective and relies on case-notes. Those patients who have done best tend to have least contact with the psychiatric services, so that detailed information on them is unavailable. The following conclusions concerning outcome following SST are limited by the lack of a control group and the controversy which exists with regard to the course of affective disorder. Angst (1981) reports that this is one of decreasing cycle length and increasing number of episodes, whereas Bland (1985) suggests a more benign course where cycle duration remains constant. If this latter view is correct, then improvement following SST may be merely due to chance variations in vulnerability to illness or placebo effects.

The placebo response to such a dramatic therapy, however, may be less of a problem. It might be anticipated that placebo responses would be immediate and of relatively short duration, and this does not accord with the observed latent period before improvement appears. Those holding the view that affective illness of the kind described here does not deteriorate with time may ascribe this latent period to a result of the patient living through the latter part of a vulnerable phase. In general the patterns depicted in Fig. 1 do not support this conclusion.

With these reservations in mind, and bearing in mind the small sample size, the following tentative conclusions may be drawn.

(a) All patients have reported a reduction of frequency and severity of episodes of illness.
(b) Hypomanic episodes have been more sensitive to SST than depressive episodes, in that they have either stopped or occurred in a very attenuated form. In one case the converse may be true.
(c) In five patients, drugs which were ineffective before SST appeared to become effective subsequently; two patients continue to require no medication. Where carbamazepine has been used postoperatively, it may improve mental stability further.
(d) Instability of mood as seen before the operation may continue in the first year following SST.
(e) The recrudescence of mood cycling in the first patient following six years in remission, and the occurrence of a hypomanic episode in patient No. 3 after being well for six years, raises the question of whether this operation has a time-limited efficacy, due perhaps to ageing.
(f) The unsuccessful course of the fourth patient suggests that SST should not be used in patients with cerebral damage.
(g) Previous reports stated that SST is an effective treatment for anxiety (Göktepe *et al*, 1975; Strom-Olsen & Carlisle, 1971). A patient in whom anxiety and phobias were a prominent feature had early relief from these symptoms, but this was a single case and a complex situation.

Thus it would appear that, far from causing a shift towards hypomania, SST attenuates hypomanic episodes more effectively than depressive episodes, although many have experienced attenuation of both. The need to continue medication following operation points to the use of SST as an aid rather than a cure; this palliation may only be temporary, although for those patients who have had severe chronic illness it must be remembered that any respite is welcome. Where carbamazepine has been used, mental stability appears to have improved. The new generation of mood stabilising drugs such as carbamazepine and sodium valproate have not been in use until recently. It may be that these drugs will make SST unnecessary in the future.

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Bipolar Affective Disorder in Down's Syndrome

Recent literature reports suggest that the presence of Down's syndrome precludes the development of mania. A case of bipolar affective illness in a man with Down's syndrome is presented.

The presence of bipolar affective disorder in mentally handicapped people is well documented (Corbett, 1979; Hasan & Mooney, 1979; Heaton-Ward, 1977; Reid, 1972; Russell, 1985; Sovner & Hurley, 1983). An extensive literature review by Sovner *et al* (1985) identified 28 cases of major depressive illness but no reported case of mania in Down's syndrome, and they propose the hypothesis "that Down's syndrome may preclude the development of mania". We present a case that casts doubt on this hypothesis.

Case report

Mr W is a 26 year-old severely mentally handicapped non-verbal ambulant man with Down's syndrome. He is a resident in a bungalow unit with nine other severely mentally handicapped people. He is transported daily to a long term training centre, and visits his elderly parents' home most weekends. There is no family history of affective illness. He was admitted to residential care in spring 1983 because of the inability of his ageing parents to cope with his intermittent aggressive and disruptive behaviour. He is mischievous by nature, and has a tendency to upturn chairs and small furnishings in an attention-seeking manner.

In early July 1984 he became withdrawn and quiet, and would sit passively, with loss of interest in his surroundings and loss of appetite. He appeared less happy, cried frequently and was increasingly aggressive towards staff. During a three week period of observation no precipitating environmental features or physical illness could be identified. He lost 4 lbs in weight. A diagnosis of depressive disorder was made, and he was started on amitriptyline (25 mg at 6 p.m. and 50 mg nocte). This dosage produced excess sedation, necessitating reduction in the first week and continued reduction until discontinuation three weeks after commencement. His mood had improved somewhat while on the medication, and he returned to a normal mood at the beginning of September 1984.

In late September he suddenly began to show sleep disturbance: he slept for approximately 2 hours per night for six consecutive nights. He became increasingly destructive with constant hyperactivity, his vocalisations increased significantly, and he became loud and boisterous. He was aggressive with staff and had regular protracted episodes of inappropriate laughter, particularly when alone. His predeliction for overturning small furnishings expanded to large beds and tables, and he needed constant supervision. Staff described him as "wound up".

Physical examination (including dental assessment), biochemical investigation, and thyroid function tests were all normal. During a 10-day observation period no change was made in his normal daily programme, and his symptoms continued in the training centre and in the setting of the residental home. At weekends his parents found him totally unmanageable and commented on his "amazing energy". Hospital admission was considered, but he was treated in a residential setting with haloperidol (5 mg t.d.s.), thioridazine (100 mg nocte) and individual nursing. He improved significantly in the next four weeks: he slept better with no violent or explosive episodes, but continued to be hyperactive and mischievous, with unpredictable episodes of laughter and shouting. He continued a fluctuating improving course; medication was gradually reduced and finally discontinued in February 1985. Since then he has continued well, and his mood is normal in a residential, training and home setting.

A month-long period of mild but similar symptomatology in 1978 was later recalled by the patient's father.

Discussion

The diagnosis of psychiatric illness in mentally handicapped people presents a considerable challenge to the clinician, particularly when confronted with severely mentally handicapped people who have no language skills. Accurate diagnosis can be made by careful monitoring of behavioural, social and physiological parameters (Reid, 1972) and by comparing the patient's normal behaviour with that which he or she exhibits during the period of instability (Rivinus & Harmatz, 1979).

It could be argued that this manic episode was induced by tricyclic antidepressant medication. This argument seems improbable, as the initial dosage was small and was reduced further before discontinuation. Total duration of tricyclic treatment was 3 weeks, and the patient was medication-free for 6 weeks prior to the onset of mania.

In view of the association between Down's syndrome and thyroid disease (McCulloch *et al*, 1983) thyrotoxicosis was considered, but was ruled out on physical examination and biochemical investigation.

The case presented meets ICD-9 (World Health Organization, 1978) criteria for the diagnosis of mania and demonstrates that bipolar affective illness and Down's syndrome can co-exist.

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Monitoring the Occurrence and Duration of Electroconvulsive Fits

During 100 routine psychiatric hospital electroconvulsive treatments the medical staff administering treatment experienced little difficulty in deciding that a fit had occurred. The judgement was based on simple clinical observation of a succession of clonic bodily movements, and high inter-rater agreement was found for the measurement of fit duration. The cuff technique was also used, but it tended to underestimate fit duration and is not recommended for routine ECT practice.

Pippard & Ellam (1981) refer to medical staff incorrectly accepting the tonic contraction of body musculature at the time of application of the electrical stimulus as evidence that a fit has occurred. It has also been suggested that 'missed seizures' with unilateral electroconvulsive treatment (ECT) might account for the bias toward bilateral ECT. The problem is that muscle relaxants may abolish the muscular activity associated with a seizure, so what should doctors accept as adequate evidence of a therapeutic convulsion?

Christensen & Koldbaek (1982) recommended that ECT should be monitored by concurrent electroencephalogram (EEG). They claimed that 43% of 156 treatments appeared to be inadequate if judged by clinical observation alone, but EEG recording proved that only 9% had really failed. This work has been criticised by Brumback (1983), who pointed out several difficulties of interpretation if only bifrontal EEG electrodes were used. EEG monitoring of ECT, although perhaps the best reference standard, involves the use of delicate and expensive recording apparatus, and takes a considerable time. Autonomic changes provide evidence that a fit is occurring, but do not indicate its duration (Watkins-Pitchford & Reynolds, 1984). Sørensen *et al* (1981) measured the duration of the fit by electromyography (EMG) and simultaneous EEG recording. Both were effective methods. The duration of convulsive activity on EMG recorded from the calf averaged 68% of the duration measured by EEG; when recording from the masseter muscles the corresponding figure was 89%.

A simpler solution was suggested by Addersley & Hamilton (1953): a sphygmomanometer cuff inflated round one arm to above systolic pressure prevents muscle relaxant injected into the other arm from reaching the muscles of the cuffed arm, which then shows unmodified movements during the fit. This technique was studied again by Fink & Johnson (1982), who compared the cuff method with EEG recording. The results were highly correlated, although the cuff estimates were 10% shorter, and they recommended the cuff technique as a valuable simple addition to standard clinical practice. All these authors implied that without a monitoring aid it was difficult to be sure that a bilateral convulsion had occurred. From past experience we held the view that there was usually no difficulty as long as the anaesthetist was prepared to vary the dose of muscle relaxant.

Method

The starting point of a fit cannot be determined clinically. All patients undergoing ECT demonstrate a tonic contraction when the electrical stimulus is passed, but this may be followed by an early or delayed start to the clonic phase. The starting point was therefore taken as the termination of the electrical stimulus. The endpoint was taken to be the cessation of clonic movements.

The treatment technique employed was routine on the unit at the time. All patients had fasted, but atropine was not given. Methohexitone (30–60 mg, i.v.) was followed by suxamethonium chloride (20–100 mg). Doses depended on age, sex and body build, and were modified in the light of the response at each treatment. Patients were gently hyperventilated with oxygen until relaxed. Brief pulse square wave stimuli were then applied from an Ectron Constant Current apparatus (1.5 ms, 40/s for 6 s at 850 mA nominal). Treatments were usually administered three times per week.

In 100 consecutive treatments involving 25 patients, a sphygmomanometer cuff was applied to the patient's upper arm and inflated to 40 mmHg above current systolic blood pressure. Anaesthetic and muscle relaxant were then immediately injected into the other arm. In any individual patient the cuff was applied alternately to the right and left arm. The doctor administering the ECT was asked to observe the whole body, and timed the fit with a stopwatch. One of us (PCB) timed both the fit duration in the cuffed arm and fit duration in the rest of the body. Seventy-nine stimuli were bitemporal, and 21 unilateral treatments were given using the temporo-parietal placement of Lancaster *et al* (1958). The majority of patients were suffering from depressive illness, and concurrent antidepressants, tranquillisers and benzodiazepine hypnotics were given.

Results

In only one treatment did a clearly visible bilateral convulsion fail to occur with a single stimulus application. The distribution of fit durations was smooth and slightly skewed (5th percentile 13 s, median 27 s, 95th percentile 59 s). Fit duration as measured by two observers regularly corresponded to within a few seconds (mean signed difference 0.26 s shorter for the doctor administering the treatment, s.d. 2.4, $P=0.28$, paired t-test). In 49 treatments the duration of the fit seen in the cuffed arm was shorter by 1–20 s than the duration of movements elsewhere in the body; in 45 treatments the duration was the same, and in 6 it was greater by 1–10 s. The clonic movements tended to persist longest in the face. On two different occasions in the same patient on bilateral treatment the cuffed arm showed no movement at all. Some of the longest fits were in patients on unilateral treatment, and the duration was similar whether the cuff was on the right or left arm.

Discussion

These results support the contention that clinical observation alone is adequate for monitoring the fit during ECT provided that the dose of muscle relaxant is not excessive. A record of the duration of the fit proves that it was at least closely observed, and may have prognostic value. The duration may be underestimated if the face is covered by an oxygen mask during the fit. It is of interest that fit duration measured in the cuffed arm was on average about 10% shorter than in other parts of the body: the same difference noted by Fink & Johnson (1982) when comparing the cuff technique with EEG recording. This may be because the fit is genuinely shorter in the cuffed arm, or because the arm becomes paralysed during the fit. The EMG findings of Sørensen *et al* (1981) support the former explanation, but Holst-Larsen (1976) has shown that suxamethonium persists in the circulation for several minutes in concentrations sufficient to cause paralysis, and during the fit violent muscular movements and increased blood pressure are likely to force blood into the cuffed arm however high the inflation pressure in the cuff. The cuff technique is therefore useful for confirming the occurrence of a fit, but may not reliably indicate its duration. It can be used as a guide to dosage of anaesthetic and muscle relaxant: an inadequately anaesthetised patient may make purposive movements with the cuffed arm, and if the dose of muscle relaxant is too large there may be movements of the cuffed arm but none elsewhere. Once the anaesthetist is working within the appropriate dose range the clonic movements which indicate a fit should be obvious without special aids. Their duration will give a good indication of the duration of convulsive activity in the brain, and if they are absent it should be assumed that a fit has not occurred.

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Recurrent Pseudocyesis and Hypomania

The case of a young woman presenting with three distinct episodes of pseudocyesis occurring concurrently with annual hypomanic illness is described, and the association between pseudocyesis and affective disorder discussed, with regard both to possible underlying psychopathology and to previously proposed psychoneuroendocrine mechanisms.

The term pseudocyesis, to describe the condition in which a woman believes she is pregnant and develops somatic manifestations of pregnancy, was introduced by Good (1823). The condition has been discussed, however, by Hippocrates (1839), who described 12 women "who imagine they are pregnant seeing that the menses are suppressed and the matrices swollen". Hardwick & Fitzpatrick (1981) have emphasised the need to distinguish pseudocyesis from delusional pregnancy, simulated pregnancy, or pseudo-pregnancy caused by an endocrine-secreting tumour such as the galactorrhoea-amenorrhoea-hyperprolactinaemia syndrome (Martin *et al*, 1977). Bivin & Klinger (1937) compiled data on 444 cases drawn from 20 countries and dating back to the 17th century; they reported that in 5% of cases there was at least one recurrence of the condition.

During the past 40 years pseudocyesis has been described under a variety of nosological categories (Cohen, 1982). Brown & Barglow (1971), commenting on the links between depression and alteration in gonadal function mediated through psychoneuroendocrine mechanisms, regarded pseudocyesis as an affective equivalent; they suggested that a specific type of depression may lead to pseudocyesis in individuals with a psychosomatic specificity or predisposition. Starkman *et al* (1985), however, found no consistent linkage between psychiatric and neuroendocrine abnormalities in pseudocyesis. Some form of loss may be an important factor in the subsequent development of pseudocyesis; this may be loss of love, of a love object, of self-esteem, or of fertility (Lapido, 1979). Barglow (1966) and Barglow & Brown (1970) have reported two cases in which a loss precipitated severe anxiety and subsequent psychosis and in which pseudocyesis seemed to be a restitutive factor. We report here an unusual case in which there has been an association between recurrent hypomania and pseudocyesis on three occasions.

Case report

K is a single, unemployed girl, aged 21; she was brought up in London and is of English parents. Her parents separated when she was seven years old, and she subsequently lived with her mother who, during this period, abused drugs (in particular amphetamines) and took frequent overdoses. Her mother later remarried, and had two more children before the dissolution of her second marriage. K has one older brother, to whom she is close, but described feeling envious of the attention he received from their father during childhood. She was denied access to her father during much of her childhood; the father remarked that "K never had a childhood of her own". He had been treated for mild depression as a young man, but was never admitted to hospital; there was no other clear family history of psychiatric illness.

K was described as an intelligent girl, but failed to sit 'O' levels as a result of anxiety symptoms. She left school at the age of 16, and at first lived with her father. For a year she worked as a secretary for a Government training scheme, and then in a school for maladjusted teenagers; the latter job ended when she developed anorexia nervosa and depressive symptoms and was admitted to a psychiatric unit. Since then she has been unemployed, except for brief periods of shop or receptionist work.

Her menarche occurred at the age of ten. She described having been raped at the age of 12 by a boy in his late teens; her relationships have been greatly disturbed, and she has been unable to establish lasting friendships. However, she leads an active sexual life and had an intra-uterine device (IUD) fitted three years ago. She has never been pregnant.

Following the deaths of her grandfather and a boyfriend, she was admitted to hospital in a florid hypomanic state at the age of 18. At this time, she had her first episode of pseudocyesis. She was noted to be heavily made up, overactive, elated, and sexually disinhibited. She displayed pressure of speech and flight of ideas, and was uncharacteristically irritable. She claimed to be 22 weeks pregnant, and refused to accept the finding of a negative pregnancy test. Her abdomen was grossly distended, and she described 'heaviness' in her breasts. She was noted to have had amenorrhoea for two months and galactorrhoea prior to her receiving any major tranquillisers. Physical examination and tests, including a screen for drugs, were negative. After ten weeks in hospital, where she was treated with haloperidol, and following an ultrasound scan which showed no uterine enlargement, she gradually accepted that she was not pregnant, but persisted in the belief that she had had a miscarriage. She appeared distraught that she was not pregnant, but denied that she had really wanted a baby. Her menstrual periods spontaneously returned and her abdomen gradually reverted to its normal dimensions, before the galactorrhoea stopped.

One year after the first episode K was readmitted to the ward, having been acting aggressively at the hostel where she had been living. She presented with a further episode of pseudocyesis, and in view of her florid hypomanic state and inability to accept hospital care was placed under Section 25 of the 1959 Mental Health Act. Her progress and recovery were similar to the year before, with the exception that she was able after some time to accept that she had had a 'phantom pregnancy'.

Following discharge she lived with her father and his common-law wife, the hostel having refused to accept her back. She spent much of her time in squats with friends, and was noted to have 'mainlined' amphetamines on two separate occasions without displaying any subsequent hypomanic symptoms. She regularly attended the outpatient department and also continued in regular counselling with the psychiatric social worker. She agreed to undergo lithium therapy, and regular serum level testing indicated her compliance. One year after her second hypomanic illness, and four months after commencing lithium, she developed a third hypomanic episode accompanied by pseudocyesis. There were no clear precipitants for this admission. There was no evidence of drug abuse in the six months prior to admission. At first, she was irritable; her periods stopped and her abdomen distended. She went to Ireland on holiday, and was compulsorily admitted to a hospital there, before being transferred back to our care. On admission she remained clearly hypomanic, and had to be compulsorily detained. She had florid symptoms of pseudocyesis, including galactorrhoea and oedematous ankles, but retained some insight and accepted she had a 'phantom pregnancy'. She was treated with haloperidol, and her mood settled over a period of several weeks, accompanied initially by a return of her menstrual periods and the deflation of her abdomen, and followed by the cessation of galactorrhoea. She was ambivalent about her wishes to have a baby: she denied being disappointed at not being pregnant and desiring to have a baby, but at times requested the removal of her IUD, saying she might like to have a baby the following year.

Discussion

K's history of emotional deprivation, leading to the subsequent development of a personality characterised by a strong craving for affection and attention, resembles the personality profiles of previously reported cases of pseudocyesis (d'Orban, 1982; Hardwick & Fitzpatrick, 1981). The psychopathology of pseudocyesis bears some resemblance to that described in anorexia nervosa (Bruch, 1973); the women concerned often have high dependancy needs and display feelings of personal ineffectiveness. In fact, K did develop anorexia nervosa prior to her first episode of pseudocyesis. The onset of mania and pseudocyesis appears to have been precipitated by the deaths of her grandfather and boyfriend. Brown & Barglow (1971) suggest that "such a loss may trigger a host of defensive and regressive phenomena or multiple affects and symptoms"; they also suggest that pseudocyesis be viewed as a depressive equivalent. A phantom pregnancy may therefore provide a means of compensating an individual with pre-existing strong dependency needs who has further decompensated through some form of loss. The association between mania and pseudocyesis in our case highlights the difficulties in distinguishing between depression and defences against depression. Mania itself has also been considered by some workers to be a defence against intolerable depression.

There is no clear evidence that either 5-hydroxytryptophan or catecholamine systems are changed in 'opposite' directions in mania and depression respectively, although some evidence suggests that these illnesses show some similarities biochemically (Green & Costain, 1981). Brown & Barglow (1971) suggest that the sequence of changes in pseudocyesis may be precipitated by alterations in availability of biogenic amines at the median eminence, eventually leading to alterations in ovarian function. They suggest that these physiological changes may be induced by psychological factors, mediated through alterations in the neuronal networks connecting the higher brain centres with the median eminence of the hypothalamus. Schildkraut *et al* (1967, 1977), in their comprehensive reviews of the literature, suggest that some depressions may be associated with a functional deficiency of noradrenaline at specific receptor sites in the brain, whereas elation may be associated with an excess of amine.

The finding of mania in association with pseudocyesis therefore makes the hypothesis that pseudocyesis is specifically related to a reduction in availability of biogenic amines at neuronal receptor sites less likely, unless there is a subgroup of individuals who have a deficiency of noradrenaline at these sites, and not an excess as previously suggested.

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Seasonality, Birth Complications and Schizophrenia in a High Risk Sample

It has been suggested that viral infections, more prevalent in the winter months, might increase pregnancy and birth complications, which might in turn be responsible for the excess of winter-born patients with schizophrenia. In this study it was found that the rate of schizophrenia among high risk winter-urban births who had suffered a perinatal complication was significantly greater than that for those who had not, thus supporting the hypothesis.

Births occurring in winter months have been shown to produce a slight excess of later-diagnosed schizophrenics. These findings have been quite robust, as evidenced by the convergent data from large population studies (reviewed by Dalén, 1975) and from samples drawn from countries in southern, tropical, and northern latitudes (Dalén & Roche, 1975); Parker & Neilson, 1976; Parker & Balza, 1977). This has led to the hope that an understanding of this relationship might suggest an aetiological hypothesis for some subgroups of schizophrenia. In the light of some suggestive findings (including the greater winter prevalence of viral infections), it has been proposed that viral infections might be involved in the aetiology of some forms of schizophrenia (Torrey, 1980).

In a longitudinal and prospective, high risk study (the index children have mothers with severe and chronic schizophrenia, and thus are at high risk for schizophrenia themselves), the degree of genetic risk and indicators of viral infection interacted to predict later schizophrenia (Machón *et al*, 1983; Mednick & Schulsinger, 1968). It was hypothesised that certain birth conditions (winter and urban birth) would increase the likelihood of contracting a viral infection. Genetically vulnerable individuals born in winter and in an urban environment should be more likely as foetuses or neonates to have suffered some central nervous system damage due to the infection; the effects of the infection would manifest themselves in a higher likelihood of psychological impairment, including schizophrenia. The highest rate of schizophrenia was observed in the high risk urban-winter birth condition (21%); the high risk base rate was 9%.

Videbech *et al* (1974) demonstrated that the increase of later-diagnosed schizophrenics among those born in the winter is strongly paralleled by a winter increase in still-births in the general population. They suggested that both of these effects might be produced by increased levels of pregnancy and birth complications during winter. Wrede *et al* (1980) provided supporting evidence: in a representative Finnish population of offspring of schizophrenic mothers, the high risk children had significantly more pregnancy and birth complications (PBCs) than the controls. These differences were almost entirely explained by the large number of PBCs in the winter-born high risk children.

Parnas *et al* (1982) reported that among high risk subjects, those who were later diagnosed schizophrenic had the most severe and the greatest number of PBCs. These findings are consistent with those of McNeil & Kaij (1978), who found increased levels of birth and neonatal complications among process schizophrenics. Within the context of a stress-diathesis model, the disadvantage posed by PBCs has been theorised as representing at least one important class of environmental stressors which interact with a genetic predisposition to increase the likelihood of later adult schizophrenia (Parnas *et al*, 1982; McNeil & Kaij, 1978). Based on these suggestive findings, additional analyses to examine the effects of PBCs were carried out on the data from the Machón *et al* (1983) study. It was hypothesised that foetal viral infections might be responsible for the increased level of PBCs observed in schizophrenia. PBCs might then be seen as a factor which mediates the relationship between the effect of winter and urban birth and subsequent development of schizophrenia.

Method and results

It was hypothesised that among high risk winter-urban born subjects, the highest rates of schizophrenia would occur among those with pregnancy and birth complications. The assessment of PBCs was as described in Parnas *et al* (1982). The results of the present analyses are presented in Table I.

The rate of schizophrenia among the HR subjects born in winter and in an urban area with pregnancy and birth complications is 30%. This figure represents an increase of 42% (from a rate of 21% to a rate of 30%) in the rate of schizophrenia when the dimension of pregnancy and birth complications (as measured by absolute number of complications) is introduced to the high risk winter-urban birth condition (see Appendix 1). Among the high risk urban births with PBCs there were a disproportionate number of schizophrenics born in winter months (30%) compared with those born in non-winter months (9%) (Fisher's Exact Test, $P<0.04$, one-tailed). These results were identical across the other two dimensions of PBCs: the most severe complication score (Fisher's Exact Test, $P<0.02$, one-tailed) and the weighted sum of complications score (Fisher's Exact Test, $P<0.025$, one-tailed). Among the high risk urban births with no complications, the rates of winter born schizophrenics (9%) were not significantly different from the non-winter born (7%) (Fisher's Exact test, $P<0.50$, one-tailed).

TABLE I

Rate of schizophrenia among high-risk subjects, distributed according to place and season of birth and to presence or absence of PBCs[1]

	Urban birth		*Not-urban birth*	
	Winter	*Not-winter*	*Winter*	*Not-winter*
PBC	30% (6 of 20)	9% (4 of 44)	0% (0 of 2)	10% (1 of 10)
No PBCs	9% (1 of 11)	7% (4 of 55)	0% (0 of 10)	0% (0 of 14)

[1] Only 16 of the 17 consensus-diagnosed schizophrenics reported in Machón *et al* (1983) are included in this analysis; one, belonging to the Not urban-Not winter cell, was dropped from the analyses due to incomplete PBC data. Of non-schizophrenics, the following numbers of subjects were dropped from each cell due to missing PBC data: respectively 2, 13, 7 and 17.

The observed and expected cell frequencies for the 'not urban birth' group in Table I are small, and so meaningful statistics were not feasible for these two sets of analyses given that only one person (out of 36) developed schizophrenia.

Individual pregnancy and birth complications (as opposed to the summed and scaled scores used above) were also examined. Analyses with these scores were not feasible, however, due to the small number of subjects in the respective cells.

Discussion

The presence of PBCs among the high risk winter-urban births appears to be associated with an increased probability of later adult schizophrenia. These data suggest that the relationship between the high risk urban-winter birth condition and subsequent schizophrenia might be mediated by the presence of PBCs in the gestational periods and deliveries of these individuals. There are several plausible interpretations of these findings. One might be that PBCs are related to winter births and that these in turn produce subtle brain damage, which later results in psychological impairment. A second interpretation might be that viral infections lead to increased levels of PBCs, which in turn damage the foetus and predispose the individual to schizophrenia. A third interpretation might be that the perinatal viral infection (or some unknown seasonal factor) directly attacks the central nervous system and as a side-effect increases PBCs. It is impossible to provide a test of these alternative explanations with these data, since no specific information on viral infections is available. Further research is needed to elucidate the nature of the relationship between perinatal viral infections and PBCs, as these might relate to subsequent adult schizophrenia.

Acknowledgement

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Appendix 1: Erratum

The following table (Table AI) should replace Table I of Machón *et al* (1983). Results and interpretation of these data remain the same as in the original paper.

TABLE AI

Per cent high-risk subjects diagnosed schizophrenic distributed by place and season of birth

*Urban births** *10%* *(15 of 145)*		*Non-urban births*** *3%* *(2 of 60)*	
Winter 21% (7 of 33)	*Not-winter* 7% (8 of 112)	*Winter* 0% (0 of 19)	*Not-winter* 3% (2 of 41)

*Fisher's Exact Test, $P<0.0442$, two tailed.
**Fisher's Exact Test, NS.

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Contents: The continuum of psychosis and the gene; AIDS panic; Not a case of pseudo-AIDS; Impact of psychiatric illness on the family; Behavioural neurology; Prognosis of depression in old age; Present State Examination Change Rating Scale; Folate, Vitamin B_{12} and posture; Diazepam in sub-acute organic states; Incidence of schizophrenia worldwide; Psychiatrists' views on treatment of depression; Afternoon radiator-sitting syndrome; Sub-clinical arteriosclerotic dementia.

The Continuum of Psychosis and the Gene

SIR: Owen & Nimgaonkar (*Journal*, April 1987, **150**, 566–567) give reasons why they are persuaded by some of the arguments (Crow, *Journal*, October 1986, **149**, 419–429) for a continuum concept, but do not accept that this reflects on the genetic basis of the recurrent psychoses. The points they raise illustrate some of the issues at stake between the 'classical' Kraepelinian view and a continuum theory.

In stating that none of the studies that show an excess of individuals with schizophrenia among the offspring of patients with affective disorder "employed modern diagnostic criteria" Owen & Nimgaonkar draw attention to a bias intrinsic to most investigations of the genetics of psychosis – the tendency to exclude cases which do not meet a particular preconception of the nature of the disease under investigation. Thus adherents of the Kraepelinian viewpoint have seized eagerly upon DSM-III and similar diagnostic decrees as an opportunity to purify their index samples of 'typical' affective or schizophrenic psychoses. In so doing they eliminate the intermediate states which in my view are the key to the problem. One of the points I made in my paper was that even when DSM-III criteria are adopted (as in the recent studies of Gershon *et al* of affective disorder and Kendler *et al* of schizophrenia) a substantial excess of cases with 'schizoaffective' disorder is seen among the relatives of patients with both prototypical psychoses. According to one's viewpoint these cases represent 'diagnostic error' or evidence for a continuum. When DSM-III criteria are applied they are excluded. Thus the bias inherent in a classical Kraepelinian perspective is further accentuated by application of criteria which are founded on the binary concept.

The point is illustrated by Owen & Nimgaonkar's reference to the work of Elsasser on the children of two psychotic parents. The combined results of Elsasser, Schulz and Kahn are shown in Table I.

Owen & Nimgaonkar quote the rates of illness in the children of parents with manic-depressive and schizophrenic illnesses. However, Elsasser also included a category of "atypical psychosis". Since this

TABLE I

The children of two psychotic parents[1]

No. of parental pairs	*Parental mating*[2]	*No. of children over 16*	*Psychotic children*[3]							
			S		*M*		*A*		*? Nature*	
			a	*b*	*a*	*b*	*a*	*b*	*a*	*b*
34	S×S	96	20	8	–	–	–	–	–	–
20	M×M	47	1	–	9	1	2	–	1	–
19	S×M	68	6	2	6	2	1	–	2	–
23	A×S	91	8	–	1	1	3	–	–	2
21	A×M	55	–	–	4	2	4	1	–	–
17	A×A	67	5	–	5	1	10	1	–	2

1. Adapted (the rates of non-psychotic disorders are excluded) from Table 12 of Slater & Cowie (1971).
2. S = schizophrenic, M = manic-depressive, A = atypical.
3. a = diagnosis certain, b = diagnosis uncertain.

category includes 78 (29%) of the total of 268 parents it is not of marginal significance. Among the offspring of the schizophrenic and manic-depressive matings one atypical and two psychoses of uncertain nature were recorded in addition to more typical schizophrenic and affective illnesses. Of more interest is the fact that among the offspring of manic-depressive/manic-depressive matings were observed two cases of atypical psychosis and one of definite schizophrenia, and among the offspring of atypical/atypical matings were observed five certain and one doubtful manic-depressive psychoses, 10 certain and one doubtful atypical psychoses and five definite schizophrenic psychoses. The pattern of transmission appears more complex and the separation between the prototypical psychoses much less clear when atypical psychoses are included.

There is no alternative to confronting the question of the genetics of schizoaffective psychosis. Angst *et al* (1983) have collected more information on this topic than any other group: "The underlying hypothesis of a continuum of psychoses from depression to schizophrenia is not disproved by our results. They show that on a descriptive level of symptoms and syndromes, taking into account the whole course of psychosis, the dichotomy into schizophrenic and affective psychosis is highly questionable. We do not only find transitional groups of schizoaffective patients but also marked affective symptomatology underlying or superimposed on schizophrenia".

An earlier study by Angst *et al* (1979) is relevant to Owen & Nimgaonkar's belief that schizoaffective disorder is no more than a result of the coincidence of schizophrenia and affective disorder in the same family. In 71 of these 150 cases of schizoaffective illness at least one other family member was affected with schizophrenia (S), schizoaffective (SA) disorder or affective (A) (unipolar or bipolar) illness. In only 17 (i.e. a minority) of these 71 families were the illnesses in the secondary cases placed in more than one category (S, SA or A) and in only eight cases did these include schizophrenia and affective disorder. Interestingly, affective disorder was found more frequently than schizophrenia (23:11) in parents of schizoaffective probands, whereas schizophrenia was slightly more frequent (11:13) in siblings. This is compatible with an inter-generational shift along the continuum.

It is little recognised that Kraepelin (1920) himself had doubts about the binary concept: "Perhaps it is... possible to tackle the difficulties which still prevent us from distinguishing reliably between manic-depressive insanity and dementia praecox. No experienced psychiatrist will deny that there is an alarmingly large number of cases in which it seems impossible, in spite of the most careful observation, to make a firm diagnosis... Nevertheless it is becoming increasingly clear that we cannot distinguish satisfactorily between these two illnesses and this brings home the suspicion that our formulation of the problem may be incorrect".

Thus Kraepelin himself was apparently abandoning the position which Owen & Nimgaonkar are defending.

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AIDS Panic

Sir: Jacob *et al* (*Journal*, March 1987, **150**, 412) report on five male patients with 'AIDS phobia'. Generalisations based on small numbers need to be interpreted with caution. Since 1984, we have systematically assessed all patients who were referred to psychiatric services with a clinical picture of panic disorder and a prominent fear of having developed AIDS but were negative for HTLV-III antibodies. Our findings are at variance with those reported by these authors. Of 24 patients in our sample (mean age 38.2 years; range 22–61; 14 females) only four admitted to being bisexual; all the rest were heterosexual. A majority (20) described a recent psychological precipitant, not always of a sexual nature; the latter was discovered in 14 patients, 10 of whom admitted to a recent illegitimate or 'unusual' sexual relationship and 4 to a homosexual contact. A detailed chronological catalogue of media reports was compiled, and an overlay plot of case inceptions showed that although a few cases did occur soon after a major media publicity campaign on AIDS, there was no significant correlation for the sample as a whole.

The mean duration of symptoms before presenting to psychiatric services was 4.5 weeks (range 3–8), and the total duration of symptoms was 6.2 weeks (range 3–22). The premorbid personalities of patients included obsessional (8), anxious/hysterical (6) and others (6); and in the rest we could detect no abnormality. Only half the patients had some form of mental disorder: obsessive-compulsive disorder (4), depressive illness (2), schizophrenia (2), and various anxiety syndromes (4), including panic disorder.

The clinical course varied considerably: less than half (11) showed complete remission of symptoms after simple reassurance or a couple of sessions of supportive therapy. At the end of three months, only five patients had persistent symptoms of AIDS panic. The most distressing symptoms were severe anxiety syndromes and depression (suicidal symptoms in five), and in four there was evidence of delusions or hallucinations. Seven patients required no specific treatment apart from reassurance in the out-patient clinic, 11 were seen regularly for supportive therapy, seven received benzodiazepines, two antidepressants, and two neuroleptics.

Detailed analysis has been submitted for publication, but the findings described above show that AIDS panic has a more complex aetio-pathogenesis than that suggested by Jacob *et al.* It illustrates the dangers of making sweeping generalisations about clinical phenomena which have many and varied contributions from constitutional as well as environmental factors.

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Not a Case of Pseudo-AIDS

SIR: *Case report.* A 30-year-old married serviceman presented with the complaint that he believed that he had AIDS. He had no previous psychiatric history and presented to his GP complaining of vague physical symptoms which he ascribed to AIDS. He denied any homosexual contact or any heterosexual contact outside marriage. He claimed that he had been exposed to risk of infection with the human immuno-deficiency virus in a fight with an unknown assailant. The history was not suggestive of AIDS, and there was no clinical evidence of the syndrome. Despite reassurance he presented on several further occasions with the belief that he had AIDS. He was therefore eventually referred for AIDS counselling and testing.

While awaiting counselling the patient again presented to his GP and on this occasion admitted to symptoms of depressive illness with lowered mood, sleep disturbance, loss of interest, and tearfulness. His GP made a diagnosis of depressive illness and started treatment with doxepin. Approximately one week later the patient's mood appeared to be lifting. He did not appear over-concerned about AIDS. However, before the negative results of testing were received, the patient hanged himself, having put his affairs in order and having taken precautions against discovery.

This patient was managed by his GP, who made a diagnosis of depressive illness and initiated treatment with antidepressants. Unfortunately the patient killed himself early in treatment. The point in question is the significance of the initial presenting symptoms of fear of AIDS and whether this man represented a case of 'pseudo-AIDS' as described by Miller *et al* (*Journal,* May 1985, **146,** 550–551).

Miller presented two cases of pseudo-AIDS, one with an anxiety neurosis and one with a depressive illness. It has long been recognised, however, that the content of psychopathology can be influenced by current themes. What is important in patients who present with concern about AIDS in the absence of the disease is not so much AIDS, but the underlying illness which gives rise to the symptoms. We should concentrate not on the question of AIDS, but on making the correct diagnosis. The significance of the presenting symptom of worries about AIDS is not that there is a specific pseudo-AIDS syndrome but that we should be aware that we may see large numbers of patients in whom AIDS figures as the content of the phenomenology. This is not something which is specific to AIDS, but is a result of the fact that AIDS is the most important new disease which we have to face in this century and is a major part of the public consciousness at this time, being prominantly portrayed in the media. We need to be aware, not only that the fear of AIDS may give rise to psychopathology, but, perhaps more importantly, that the psychopathology of illness may be coloured by the collective fear of AIDS which exists and which is likely to grow in the wake of the government's publicity campaign. We are likely to see patients who are suffering from a wide variety of psychiatric illnesses all presenting with worries about AIDS.

Miller states that there is a clear need for clinicians to be alerted to the likelihood of psychiatric complications arising from fear of AIDS in their homosexual patients. It appears likely that this advice is correct, but already out of date in one important aspect: AIDS is no longer purely a disease of homosexuals and drug addicts. Growing numbers of cases are arising as a result of heterosexual transmission. It is important that we remember that a wide range of psychiatric illnesses in heterosexual patients may also present with fear of AIDS.

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The Burden of Care: The Impact of Functional Psychiatric Illness on the Patient's Family

Sir: This article (Fadden *et al, Journal,* March 1987, **150,** 285–292) purports to review the literature on "the effect of severe mental illness on other members of the patient's family", but at no point mentions the word 'child'. Most definitions of 'family' include a reference to children, for example, "the household, or all those who live in one house (as parents, children, servants): parents and their children: the children alone" (Macdonald, 1972).

A number of authors have written about the impact of parental psychiatric illness on children. Rutter & Quinton (1985) found that these children had a higher rate of psychiatric disturbance than a comparison group, were more likely to experience discord, and were often exposed to hostile or anxious depressed behaviour. Rodnick & Goldstein (1974) studied maternal behaviour before and after schizophrenic breakdown and commented that even in those mothers with good premorbid functioning, the recovery of 'mothering function' was delayed for as long as six months after discharge from hospital. Significant intellectual deficits have been found in children whose mothers were depressed during their first year (Cogill *et al,* 1986). There is no doubt that some children are adversely affected by a parent's mental illness.

Fadden *et al* comment on the "high rates of divorce and separation in marriages where one patient [I presume they mean partner] is mentally ill." Wallerstein & Kelly (1976), among others, have examined the effects of divorce on children. Again, there is no doubt that some children suffer.

The link between parental psychiatric problems and emotional disturbance in children is not necessarily, or always, causative. It does exist. We must be aware that the children in families where a parent has a functional psychiatric illness are at risk.

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Behavioural Neurology

Sir: Reynolds, in his book review (*Journal,* March 1987, **150,** 421–422) asks the interesting question, "What is behavioural neurology?". I too have sought to discover the origins and boundaries of this discipline, and am usually led back to the writings of Geschwind and his colleagues. As Reynolds points out, behavioural neurology has rediscovered many of the localised neuropsychological syndromes described by the neuropsychiatrists of the late 19th century, and essentially attempts to understand disturbances of behaviour following identifiable brain lesions. The culmination of this line of thinking was represented by Geschwind's (1965) classic paper on disconnection syndromes. As such, behavioural neurologists are found largely in the USA and approach behavioural problems from a relatively strict localisationalist, neurological point of view.

This may be contrasted with the related discipline of neuropsychiatry, where holism and integration of cerebral function tend to be stressed. Neuropsychiatry adopts a more dynamic view of the relationship between the brain and behaviour, compared with the more static models adopted by behavioural neurology (Trimble, 1981).

A third, related discipline is biological psychiatry, the boundaries of which I have discussed recently (Trimble, 1987). Biological psychiatry attempts to understand psychopathology in relationship to underlying disturbances of brain function, and concentrates more on primary psychiatric conditions such as affective disorder and psychoses than either neuropsychiatry or behavioural neurology.

Finally, organic psychiatry examines "cognitive, behavioural and emotional consequences of cerebral disorder" (Lishman, 1978), which includes not only the consequences of structural brain disease, the field of behavioural neurology, but also metabolic, toxic and other systemic conditions which disrupt cerebral function and may provoke psychopathology.

The recent growth of interest in three of these disciplines (behavioural neurology, neuropsychiatry and organic psychiatry), all of which overlap at their boundaries, reflects the long overdue interest in the neglected disorders which form the hinterland between neurology and psychiatry. Furthermore, it represents attempts by their practitioners to integrate the fields of psychiatry and neurology, which have become too divergent.

Reynolds notes of behavioural neurology: "it is a discipline that does not appear to exist in the UK". If the sighting of one black swan disproves the premise that all swans are white, then I submit that behavioural neurology does exist in the UK.

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Prognosis of Depression in Old Age

SIR: Murphy (*Journal*, February 1987, **150**, 268) is incorrect in assuming that our mortality rate is remarkably similar to that obtained in her study. The figure of 35% she quotes for our patients refers to the entire follow-up period, which was as long as 104 months for some patients. We have already calculated the four-year rate for our cohort as part of another study (*Journal*, in press). For the 97 ascertained (three were untraceable) the deaths at four years amount to 25 (18 women and 7 men), i.e. 26%. It is usual to assume a year-on-year rate of 5% for this age group, or 20% at four years – not much different from our findings and in marked contrast to the 37% quoted by Murphy for her study. Thus, the difference in death rates between the two cohorts of patients that was evident at one year seems to persist at four-year follow-up.

This is certainly not the only difference identified, but it is the one that most robustly withstands arguments about the validity and reliability of our data. Murphy raises doubts about these on account of our retrospective methodology, although survivors were interviewed and information from GPs and, in some instances, personal contacts was collected for others. In fact, she too used a retrospective method for her own assessment of the course of depressive symptomatology, and unless patients are interviewed by a researcher extremely frequently it is hard to see how it can be otherwise.

We have attempted some replication of Murphy's work and found concurrence over major life events and physical ill-health, but not with regard to the prognostic significance of severity of depression and delusions. However, we have also hopefully widened the area of research. Hence, Post's four-fold categories of outcome were used not only to replicate his work, but also because a dichotomous 'good' versus 'poor' approach conceals significant differences in the quality of mood during life – something we believe to be immensely important. Also, we have specified the range of treatments and after-care offered, since they can hardly be discounted in assessing outcome.

It is hoped that future research can synthesise the different emphases in these studies, so that at least we are drinking from the same pint pots!

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Present State Examination Change Rating Scale

SIR: The inter-rater reliability of the ratings is impressively high (*Journal*, February 1987, **150**, 201–207), but reassurance is necessary on several points.

Are not the intra-class correlation coefficient, with its significance tested by the '*F*' statistic, and the Pearson product-moment correlation coefficient not parametric statistics, and if so why have the authors chosen to use these in the absence of evidence that the underlying scale of measurement has interval qualities? The PSE rating scale of 0, 1 and 2 is at best ordinal, being a set of ranks separated by unequal intervals, so how plausible is it to assume that a symmetrical bell-shaped curve arises out of such a scale? Would not the conservative use of non-parametric statistics be preferable?

Do the intra-class correlation coefficient and the Pearson product-moment correlation coefficient allow for agreement due to chance: thus, has such agreement been subtracted from the observed coefficients?

Do the intra-class correlation coefficient and the Pearson product-moment correlation coefficient distinguish between agreement and association? Agreement is a special kind of association of interest in reliability studies, and it is possible for association to be very high while agreement is poor.

Would the authors like to say why they did not place screens between the raters? This strategy would have made the authors' claim that the raters were independent more credible, because they would have been seen to acknowledge the possibility that raters can make inferences about one another's rating behaviour by noting when pen moves to paper. Placing a screen between raters has been shown to result in a fall in the very high agreement about symptoms in joint interviews to the level for independent interviews (Robinson *et al*, 1982), which raises the intriguing possibility that this very high agreement reported here and elsewhere, for example in the International Pilot Study of Schizophrenia (World Health Organization, 1973), has at least two components: one a measure of agreement about symptoms by psychiatrists, the other a measure of sensitivity between psychiatrists.

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Folate, Vitamin B_{12} and Posture

SIR: Abou-Saleh & Chung-A-On (*Journal*, January 1987, **150**, 133) have reported a study of vitamin B_{12} and folate plasma levels.

We observed in 23 patients who had been lying down for at least 6 hours, that standing for 30–40 minutes increased vitamin B_{12} and folate plasma levels by more than 10%. Red cell folate levels decreased non-significantly by an average of approximately 2%.

The explanation of our observations is the following: as soon as the subject stands, fluid (up to 23% of plasma volume) leaves the circulation under the influence of hydrostatic forces, particularly in the lower limbs. As a result, there is an increase in concentrations of blood constituents such as red cells, proteins (Fawcett & Wynn, 1960; Hagan *et al*, 1978), and protein-bound substances such as calcium (Husdan *et al*, 1973), folate, and vitamin B_{12}, which do not readily pass through the capillary membrane. Hagan *et al* (1978) have shown that standing for 35 minutes increases venous haematocrit, haemoglobin, and plasma protein levels by 10%, 10%, and 20% respectively.

Our observations underline the need to control posture and other factors which may change plasma volume (Husdan *et al*, 1973; Abalan *et al*, 1987) in folate and vitamin B_{12} plasma level studies as in studies of other protein-bound vitamins.

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Transient Effect of Diazepam in Some Sub-acute Organic States

SIR: Aynsworth's report (*Journal*, January 1987, **150**, 110–112) of a patient with a catatonic state possibly related to viral encephalitis prompts us to comment on her finding in that patient of a brief return to apparent normality after intravenous diazepam. We note that she quotes previous reports in which intravenous barbiturate resulted in improvement in organic catatonic states.

Case report: A 19-year-old woman suffered severe hypoxic brain damage due to drug overdose, with a severe defect in short-term memory and attention span and a trans-cortical aphasia. At times she was mute or nearly mute. Grimacing and posturing was accompanied by disinhibited behaviour and apathy. For several months she refused to eat. Intravenous sodium amytal produced a striking temporary improvement in her awareness, attention, speech, and interpersonal relationships. Oral diazepam resulted in a recordable improvement in her behaviour and level of activity over several weeks, and in a longitudinal cross-over study with placebo over several months the improvement was deemed attributable to the diazepam. Intermediate memory (recording and recall of verbally and visually represented material) improved slightly with diazepam and amytal.

We do not understand the mechanism of this finding in our patient or in Aynsworth's patient. The fact that improvement occurs in catatonic states with barbiturate or diazepam infusion should not necessarily lead clinicians to suspect hysterical mechanisms. In the female patient we report, the benefits of diazepam gradually wore off over six months. Although environmental changes have resulted in further improvements in her condition she remains in sheltered accommodation.

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Similar Incidence Worldwide of Schizophrenia: Case not Proven

SIR: The article by Sartorius *et al* (*Psychological Medicine*, November 1986, **16**, 909–928) is a summation of a prodigious amount of work and contains invaluable data. The two important conclusions reached, however, must be examined closely in the light of the diagnostic criteria and population cohorts which were used.

Firstly, we consider the significance of the "central" (by which they mean 'nuclear', Schneiderian) schizophrenia syndrome that the authors state occurs with approximately equal frequency worldwide. To study incidence data, the authors include all individuals in a given catchment area who made a first lifetime contact with any helping agency, hospital, clinic, or religious or traditional healer over the one-year collection period of the study with a disorder that met either broad (ICD–9 or CATEGO S, P, O) or narrowly defined (CATEGO S+, Schneiderian, nuclear) criteria for schizophrenia. The problems with these definitions are well known: neither the ICD–9 definition nor the Present State Examination (PSE)-based CATEGO classification requires a minimum duration of symptoms for diagnosis of schizophrenia. This appears to have led to the considerable diversity of the schizophrenic samples collected in participating countries, as illustrated by the large differences in age-specific incidence rates in them. For example, in their Table 6 it is evident that the percentage of first-contact schizophrenic patients in the 15–24 age group in Cali (70.8%) is more than three times that in Moscow (21.8%). Among 25–34 year olds, Ibadan (42.3%) has twice the percentage of Cali (21.4%). For 35–44 year olds, the range is even wider: 3.6% of the sample of first contact schizophrenics in Rochester falls in this age-group, compared with 6.5% in Agra, 19.4% in Dublin, and 22.3% in Moscow. Finally, Moscow reports that 20.4% of their total sample of first-contact schizophrenic patients fell in the oldest age-group (45–54 years), while only 1.2% of first-contact schizophrenic patients in Agra and 3% of the Cali sample fell in this range – a nearly 20-fold difference.

With respect to the clinical syndrome, again the differences are as striking as the similarities: while only 26% of patients in developed countries had acute onset, 49% had such onsets in developing countries. A diagnosis of acute schizophrenic episode was made in 40% of patients in less developed countries, but only 10% in developed countries, and the catatonic subtype was 10 times more common in developing countries. Indeed, inspection of their Fig. 2 indicates that in the developing countries more than 30% of all patients in the survey had been ill less than one month and 50% for only two months! While the differences in clinical presentation, such as more violence and excitement in developing countries and more paranoia and depression from developed lands, might be attributed to cultural differences, the great differences in mode of onset and age distribution between centres indicates that the schizophrenia defined by the authors presents great differences between the places studied. Indeed, what would be designated by RDC or DSM–III criteria as *acute reactive, brief* or *schizophreniform* psychosis is characteristic of a majority of admissions to psychiatric facilities in the developing countries. If diagnosed as schizophrenia, this may lead to the false conclusion that the same disorder has an equal prevalence around the world. Clearly, inclusion of this large number of acute psychoses of less than two months' duration, predominantly from the developing countries, will also bias the statistics by indicating a more favourable course for schizophrenia in developing countries. Inclusion of alcohol and drug-related psychoses by the authors will further blur the data, and may account in part for the large differences in age of first-contact schizophrenic patients between the centres.

The incidence data, claimed to show a similarity in all populations studied, are flawed in two respects. Firstly, only 8 of the 13 areas studied had sufficient case-finding coverage and base population data to derive incidences; Ibadan, Cali, Agra, Prague, and Rochester were excluded. Exclusion of the first three means that the incidence data from developing countries derive solely from Chandigarh – a rather unique 'model city' in the Punjab and the site of one of India's major medical centres. With all of South America, Africa, and most of Asia excluded, the incidence data should not really be referred to the *worldwide* prevalence question. Secondly, the similarity of incidence worldwide which is claimed by the authors holds only for their S+ category, i.e. 'nuclear', Schneiderian schizophrenia; this category shows poor specificity, especially in early cases (Silverstein & Harrow, 1978), and very poor predictability for subsequent outcome diagnosis (Brockington *et al*, 1978). Furthermore, Schneider's first-rank symptoms are frequently found in mania, in addition to schizophrenia (Carpenter & Strauss, 1974). The S+ category is thus of doubtful validity for the diagnosis of schizophrenia. Yet it is only with this narrow, S+ definition that the author's claim of equal incidence and prevalence holds, for there is a large variation in incidence between centres with their broad (Catego S, P, O) definition of schizophrenia, with e.g. rural Chandigarh women having four times the incidence of women in Aarhus. This suggests either an epidemic-like pocket of schizophrenia in the rural Punjab, or more likely reflects the high incidence of culturally-based brief psychosis that would probably have been found in the other traditional societies, had they been included in the incidence survey.

The greater prevalence of acute reactive psychosis in developing countries has been long and widely recognised by psychiatrists working in these regions (Dembovitz, 1945; Carothers, 1953; German, 1972). During a three-month period in which JRS served as Consultant Psychiatrist at the School of Medicine of the University of Zimbabwe, it was found that more than half the patients admitted to the psychiatric ward of the Harare Hospital with a diagnosis of schizophrenia failed to meet either RDC or DSM-III criteria for duration of the illness, and that onset was often close to an emotionally traumatic event. Using DSM-III criteria, their diagnosis was acute brief or reactive psychosis (Stevens, 1987).

There is no doubt that typical schizophrenia, meeting RDC, DSM-III, Bleulerian, and Kraeplinian criteria, exists in all parts of the world where it has been looked for. In the absence of some duration criteria, however, the greater incidence of acute, reactive, culturally-based psychoses in developing countries is likely to make an important contribution to the conclusion by the authors that schizophrenia has a better prognosis in developing countries. For the three reasons mentioned – the questionable validity of the Catego S+ category for diagnosis of schizophrenia, the exclusion of all but one developing country in the incidence study, and the inclusion of brief (including culturally-related, drug, and alcohol) psychoses in their study – the authors' conclusion of worldwide similarity of schizophrenic incidence also seems premature.

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Sir: The recently released WHO multi-centre study of the incidence of schizophrenia was said by the authors to lend "support to the notion that the 'central' schizophrenic syndrome may be occurring with approximately equal probability in different populations" (Sartorius *et al*, 1986). The WHO study, however, does not shed much light on the comparative incidence of schizophrenia because all seven centres utilised in the incidence study were located in areas in which previous prevalence studies would suggest no more than a two-fold to three-fold difference; that is exactly what the WHO incidence study found.

The centres utilised in the WHO study and prevalence of schizophrenia per 1000 population reported in previous studies in those countries (Torrey, 1987) included: Nagasaki (2.1-2.3), Nottingham (2.1-3.4), Moscow (2.6-3.8), and Chandigarh (1.9-5.6 in different areas of India; no prevalence study had been carried out in the Chandigarh region). Honolulu was utilised as the American centre despite the fact that it is very atypical demographically and that previous first admission rates for schizophrenia show Hawaii to rank comparatively low among the states; by contrast New Haven, Baltimore, and St Louis were recently reported to have high prevalence rates of schizophrenia (Myers *et al*, 1984).

Aarhus was used as the Scandinavian centre; previous prevalence studies (2.7 per 1000) (Nielson, 1976) and incidence studies (Munk-Jorgensen, 1986) have shown rates for schizophrenia in Denmark to be comparatively low. By contrast, most studies in Norway, Sweden, and Finland have reported high rates, up to 17.0 per 1000 in the Book *et al* (1978) study of northern Sweden. The final centre used in the WHO study was the St Loman's case register in Dublin. In 1982 schizophrenia prevalence figures from this case register were only 1.7 per 1000 (Walsh, personal communication), less than one-third the 5.9 per 1000 rate for the County Roscommon case register in western Ireland and less than one-seventh the rate of 12.6 per 1000 reported by Torrey *et al* (1984) for a suspected high prevalence pocket in western Ireland.

In summary, the WHO incidence study of schizophrenia studied seven centres for which previous prevalence studies would lead one to expect no more than a two-to-three-fold difference in incidence. That is precisely what was found. Until a multi-centre incidence study is done utilising centres from suspected high incidence (e.g. northern Scandinavia, western Ireland) and low incidence (e.g. some tropical areas) countries it would seem unwise to regard the WHO incidence study as a reflection of worldwide incidence rates.

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Psychiatrists' views on Treatment of Depression

Sir: I note Armstrong & Andrew's paper on the treatment of depression by Australian psychiatrists (*Journal*, December 1986, **149**, 742–750). This is one of many papers supported by the Royal Australian & New Zealand College of Psychiatrists under a so-called Quality Assurance Project.

The implications of this work are actually quite astounding. The modal length of treatment for each of the case vignettes is over fifteen hours per patient. Simple arithmetic would suggest that every consultant psychiatrist would have reached total saturation point with the referral of one hundred patients per annum. Quality treatment for these one hundred fortunate people would totally absorb the Quality Assured Practitioner to the exclusion of all other professional activities. This work obviously has more to do with maximising psychiatrists' incomes than with making psychiatry more available to the masses.

In Australia, I would add, this Quality Assurance Project is being held up as a model of excellence to which the College hope all psychiatrists will ultimately aspire, or should I say, conform.

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'Afternoon Radiator-Sitting Syndrome': Hypothermia and Early Diagnosis of Self-Induced Water Intoxication

Sir: We report on a patient with schizophrenia and acute self-induced water intoxication (SIWI) who presented with symptoms of hypothermia. Further investigations revealed a clinical sign which may be useful in the early diagnosis of SIWI.

Case report: A 37-year-old man, who had been an in-patient for 17 years, presented at 1600 hours with complaints of feeling unwell accompanied by uncontrollable shivering, shaking, and chills. Oral temperature was 35.8°C. Over the next two hours he became increasingly confused and ataxic, and was incontinent of large volumes of dilute urine. Laboratory investigations were normal except for serum sodium which was markedly low at 121. Overnight he passed approximately eight litres of dilute urine (S.G. 1.001), and by 0800 the following morning was alert and symptom-free, with a temperature of 37°C and serum sodium of 136.

Close observation over the next two weeks revealed that he typically consumed 9–12 litres of water daily, and dropped his sodium from a morning value of 136 to a mid-afternoon value of 126–128, accompanied by recurrent, but less severe, symptoms of hypothermia. Regardless of ambient temperature, he wore several sweaters and jackets and spent much of the afternoon sitting beside, or on, the hot water radiators which supplied heat to the building. Interviews with nursing staff who knew the patient well revealed that this was his 'typical' behaviour pattern, which had been present for many years. With fluid restriction to 3–4 litres a day this pattern disappeared, and his afternoon sodium values returned to normal.

Seven additional patients with a similar pattern of afternoon radiator-sitting syndrome were identified in a single afternoon by a visual survey of five chronic psychiatric wards involving 210 patients. On further investigation, all seven showed polydipsia and recurrent afternoon hyponatraemia (sodium levels of 124–132). In each case, fluid restriction abolished the afternoon hyponatraemia and radiator-sitting behaviour. Four patients had no previous known history of hyponatremia or polydipsia. One later developed hyponatraemic seizures. Three had previous episodes of acute water intoxication with hyponatraemia and seizures. Chart reviews of these three revealed numerous descriptions of what appeared to be, in retrospect, afternoon hypothermia, dating as far back as 14 years prior to the onset of seizures and formal diagnosis of water intoxication.

The recurrent afternoon hypothermia and hyponatraemia associated with polydipsia in these patients may be related to volume overload with tapwater at room temperature, faster than it can be heated or cleared. Alternatively, the altered temperature regulation, increased thirst, impaired water homeostasis and circadian rhythmicity may all be secondary to increased hypothalamic dopamine activity and may represent a tardive hypothalamic syndrome.

Self-induced water intoxication typically goes unrecognised until the development of episodes of acute intoxication with severe neurological symptoms which may be life-threatening (Ferrier, 1985). Additionally, it may be related to tardive changes in hypothalamic dopamine function (Jones, 1984). Early diagnosis is therefore crucial. Screening for afternoon radiator-sitting syndrome appears to be a rapid, inexpensive, sensitive, and, thus far, specific way of detecting patients at risk for this disorder.

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A Search for Sub-Clinical Arteriosclerotic Dementia

SIR: McDonald (1983) searched clinical records of 1432 psychogeriatric admissions for dementia plus one or more strokes or dementia plus epilepsy and found only 17 patients who might meet the criteria for the diagnosis of arteriosclerotic dementia, a sub-class of multi-infarct dementia.

Warlow (1980) points out that the deterioration in cognitive ability in arteriosclerotic dementia presumed to be caused by a succession of minor vascular catastrophes tends to be "stepwise" but that not all the episodes are "necessarily obvious". That is to say, to account for cerebral-vascular pathology which would not have been predicted from the clinical history, a sub-clinical course of arteriosclerotic dementia must be postulated. We report a clinical search for minor cerebro-vascular episodes which do not amount to strokes in a female chronic psychogeriatric population in a mental hospital.

On a census day, all the psychogeriatric patients in Warlingham Park Hospital (from a total of 227) who had had more than five "turns" of any kind were identified. "Turns" included transient ischaemic attacks, Alvarez' Little Fits, drop attacks, faints, collapses, and tonic or clonic attacks. The patients thus identified were examined for the presence of severe dementia using the Kew Cognitive Map (McDonald, 1969).

Six months later, from the same population, a second group of patients was identified as being on any drug used in cardiovascular disease; this population was presumed to include those patients with diagnosed (and treated) hypertension. Again, the population was examined for the presence of dementia.

The first search revealed 15 patients, none of whom were found to be demented following a stepwise history as judged from a review of the history of the present illness in the case notes. That is to say, no sub-clinical phenomena or pseudo-stroke phenomena were occurring which could have led to a diagnosis of arteriosclerotic dementia being made in any of these cases.

The second search found 26 patients, of whom 7 had a pre-existing psychiatric or neuro-psychiatric condition dating from middle age or earlier (i.e. antedating arteriosclerotic dementia), for example depression, schizophrenia, Korsakoff's psychosis and head injury. Six of the patients in this group were demented, and had presented with an episodic stepwise history.

The high discordance between the pathological findings and the ante-mortem diagnosis of 'arteriosclerotic dementia' is well known. This study has searched for a clinical way of bridging this gap, but has failed to find that in the concept of sub-clinical strokes.

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CORRIGENDUM

Journal, May 1987, **150**, 662–673 (R. E. Kendell *et al*). The first line of the summary should read "Computer linkage of an obstetric register and a psychiatric case register made it possible to investigate the temporal relationship between childbirth and psychiatric contact in a population of 470 000 people over a 12-year period: 54 087 births resulted in 120 psychiatric admissions within 90 days of parturition.

A HUNDRED YEARS AGO

Mental disorder caused by jaborandi

Dr Wauzh of Liège, who has great belief in the efficacy of jaborandi as a stimulant of the mammary functions, mentions a case which has recently occurred in his practice, showing that inconveniences may occasionally arise from the administration of this drug. He prescribed ten drops of the fluid extract every four hours for a patient whose milk had ceased for a fortnight, with satisfactory results, the secretion being re-established. After a time, however, the patient began to suffer from extreme nervous excitement, accompanied by a fixed idea that she should murder all her family with a hatchet. On the jaborandi being stopped these alarming symptoms disappeared, and together with them the activity of the mammary glands.

(Jaborandi: The dried leaflets of a Brazilian plant *Pilocarpus pinnatifolus. N.O. Rutaceae*, having diuretic and sudorific properties – *OED*)

Reference

The Lancet, 24 December 1887, 1286.

Researched by Henry Rollin, Emeritus Consultant Psychiatrist, Horton Hospital, Surrey.

British Journal of Psychiatry (1987), **151,** 136–143

Book Reviews

Editor: Sidney Crown

Basic Family Therapy (2nd ed.). By PHILIP BARKER. London: Collins. 1986. Pp 334. £9.95.

Barker aims to provide an easily read introduction to family therapy. This second edition of his book is much more than an update – it is a re-write that tries to create a sense of harmony in this rapidly developing field.

To the uninitiated like myself, family therapists seem both crazy and cavalier. They make bizarre recommendations to families, justifying them as 'paradoxical' and therefore 'strategic'. They complicate the therapeutic relationship by 'live supervision', in which the supervisor watches the session from behind a one-way screen and telephones messages through to the therapist. Scorn is poured on the importance of unconscious communication. Transference and counter-transference are not acknowledged. I therefore started this book with considerable scepticism.

Barker has tried to write a book that is both a work of reference and a practical handbook. His style varies from précis of the main literature to a personal account of his own way of working. He reviews all the major approaches and techniques, trying to give the whole field a sense of unity and cohesion. *En route* he points out key references, thus providing a most useful guide to the literature. His review makes it quite clear that family therapists have a lot to offer us all and are providing accounts of how families develop and how they are structured and function, which are useful even if we don't practice family therapy as such.

Barker's own method of therapy is based on the McMaster model, but in true eclectic fashion he weaves in concepts and techniques from all the other schools. He gives a practical step-by-step account of how to assess, contract, treat, and end therapy with families. He describes in detail both 'systemic' and 'structural' ways of looking at families, and gives many examples of strategic interventions. The account is systematic and reasoned – too much so, perhaps.

He has achieved his objective of providing a "clear, easily read and readily understandable introduction to the subject". This book should certainly be in the library, as it gives a broad picture of the world of family therapy. It is much more than an introduction: it is a textbook, it is a guide to further reading, and it is a practitioners' handbook. Reading it has lessened but not eliminated my scepticism.

C. R. WHYTE, *Consultant Psychotherapist, Humberstone Grange Clinic, Leicester*

Behavioural Psychotherapy: Maudsley Pocket Book of Clinical Management. By I. M. MARKS. Bristol: Wright. 1986. Pp 133. £9.95 pb.

Despite considerable advances in the field of psychotherapy in the last decade, the evidence would suggest that behaviour therapy is still one of the most effective psychological treatments available, albeit for a relatively small number of well-circumscribed disorders. Although descriptions of techniques and guidelines for their administration are well documented in the literature, comparatively little has been written about the clinical management of cases undergoing this form of treatment. Marks has addressed himself to this issue, and the result is a detailed set of instructions regarding screening procedures, interview formats, and letter writing. The style of the book is consistent with the author's orientation within the behaviourist school. Marks is a powerful advocate of the traditional approach which focuses on the achievement of objectively defined targets by direct means rather than the facilitation of internal change through a combination of cognitive and behavioural interventions. The attraction of such an approach lies in its simplicity. Not surprisingly, therefore, this tightly structured volume sets out to reduce the many sources of ambiguity in the psychotherapeutic process by operationally defining every activity the clinician might engage in. Although such an exercise in reductionism can be useful in the treatment of the simple phobias and obsessional disorders illustrated here, clinicians working with more complex cases might find many of these guidelines excessively rigid.

This book has been written primarily for students on nurse therapy training courses and for other professionals working in specialised behaviour therapy units. For the majority of clinicians, however, its main value will be as a source book for diverse questionnaires and rating scales which can be usefully employed to measure response to treatment, be this behavioural or otherwise.

DOUGAL MACKAY, *District Psychologist, Barrow Hospital, Bristol*

Developmental Disorders: The Transitional Space in Mental Breakdown and Creative Integration. By PETER L. GIOVACCHINI. Northvale, NJ: Jason Aronson. 1986. Pp 385. $35.00.

Giovacchini, an American psychoanalyst who has written extensively on character disorders and on adolescence, has now produced an excellent book on the psychoanalytic understanding and treatment of such patients. The book contains three interwoven strands: a discussion of contemporary psychoanalytic concepts, an exposition of the author's theories on character disorder, creativity, and the use of transitional space, and detailed clinical material illustrating the theory.

The general discussion on psychoanalytic concepts is well written, and highlights some of the changes in American psychoanalytic practice over the past few decades. One small criticism is the author's failure to include contemporary Kleinian writers, particularly as, for example, his discussion on transference seems to be informed by the work of Betty Joseph.

The discussion on character disorders is complex, and not always easy to follow. Giovacchini emphasises the importance of Winnicott's notion of transitional space, and the failure of some patients to make creative use thereof. He also stresses the way some severely disturbed patients will try to recreate in the analytic setting the primitive infantile environment which they find most soothing. Herein lie many pitfalls both for the analyst and for the patient.

Giovacchini is at his best in the clinical discussions, which are subtly and sensitively written. He describes some extremely difficult patients and their families, highlighting crucial features of the treatment. He is particularly good in his discussion of the pressures to modify the classical method, and emphasises that such modifications should only be as a planned therapeutic intervention, rather than happening by default.

Overall the book is a well-written account of one practitioner's work with difficult patients, and I can recommend it, especially the clinical discussions, to those who are puzzled by the pressures that such patients place on us.

MARK BERELOWITZ, *Senior Registrar in Child Psychiatry, Maudsley Hospital, London*

Psychotherapy and the Selfless Patient (The Psychotherapy Patient Series). Edited by JEROME A. TRAVERS. New York: The Haworth Press. 1986. Pp 109. $19.95.

This little book from the USA presents a series of papers by psychotherapists of diverse clinical and theoretical backgrounds, including Gestalt, transactional analysis, bioenergetics, client-centred and psychoanalytic. The purpose of the collection is given as "to explore various genres of selflessness" along a spectrum from developmental deficit or failure to developmental ideal. Most of the papers present clinical vignettes or case studies with an accompanying theoretical discussion.

Two case studies are offered demonstrating selflessness as an achievement: "the outcome of a rather mature religious orientation", and "a loss of self that is freely chosen in order to more deeply experience himself and God". For the most part the authors explore selflessness as a developmental deficit, although a view of this as a strategy for survival rather than mere fault is emphasised.

In this, as in other aspects, the self-psychology of Kohut is an important influence and a number of concepts from this approach are introduced. The idea of the self-object, a person experienced by the child as part of the self, who carries out certain personality functions for him and the nature of these functions in the development of self are related to transference phenomena in the psychotherapy. Empathy, vicarious introspection, and use of the therapist's own subjective experience in understanding the patient are recommended.

Other theoretical perspectives include that of attachment theory, discussing the development of self from the base of secure or insecure attachment. Holding and retrieval are described as forms of basic parental attachment behaviour, and it is suggested that their symbolic equivalents could be needed and provided in psychotherapy.

Winnicott and Guntrop are referred to in discussion of the ideas of true and false self and the development of these with a view of the true inner self concealed within a compliant false outer self.

The idea of selflessness as a survival strategy includes examples such as mindlessness and decathexis or 'blanking out'; the threatened selflessness of the suicidal patient and control or mastery gained through this; and the selflessness of the selfless caretaker. Systems theory is mentioned, and there is a sociological view with an analysis of the 1984 United States presidential election.

The book is a light but not unstimulating read. I regret it may be stimulating as much because of its deficits as its good points. The form of eclecticism in the book is such that the clinical and theoretical material is presented relatively undigested, and there is little critical discussion or examination of how one perspective might relate to or influence another. For me there was a kind of flirtation, but failure to follow through. It could serve as an introduction and stimulus to further reading, although the Americain origin and the particular view of varieties of psychotherapy should be borne in mind.

KATE DUFTON, *Senior Registrar in Psychiatry, Claybury Hospital, Essex*

The Analytic Experience: Lectures from the Tavistock. By NEVILLE SYMINGTON. London: Free Association Books. 1986. Pp 347. £22.50 (hb), £8.95 (pb).

If this *Journal* invited reviewers to nominate their Books of the Year, *The Analytic Experience* would head my list. A new book always holds promise of treasures in store, but sometimes the gold is sparse, or the reader has to struggle with turgid prose and impenetrable jargon to discover the buried bounty. Cause for celebration indeed that psychoanalysis is so lovingly and lucidly exposed by Symington in a work that is a joy to read from beginning to end, which clarifies areas shrouded in mystique and confusion that have been alien territory to many who spend their professional life concerned with the minds of men.

For many years Symington was on the staff of the Tavistock Clinic, and his lectures to mental health professionals attending training courses there influenced the development of many young psychiatrists. This book is the result of these lectures, and should ensure that his influence will continue to reach a wide audience, although it must be a cause for regret that he has now emigrated to Sydney. A former Director of the Camden Psychotherapy Unit and a member of the London Institute of Psycho-Analysis, Symington's extensive experience informs his exposition of psychoanalysis as it affects both patient and analyst, and many clinical examples are used to illuminate the nature of the psychoanalytic process. Humanity and humour shine through this book, which amply demonstrates the author's rare gift for communication, rendering the complex simple without ever being simplistic.

The student looking for a basic introduction to Freud's theory and the later developments of psychoanalysis could not do better, but experienced therapists too can benefit from this simple restatement of the familiar and the obvious. Free Association Books deserve an accolade for producing this text, and at the modest paperback price it is a bargain not be missed. A must for the library, for psychotherapy reading lists, and for every clinician who is interested in the function of the mind and the potent therapeutic promise of the doctor/patient relationship.

PAMELA M. ASHURST, *Consultant Psychotherapist, Royal South Hants Hospital, Southampton*

Aspects of the Feminine. By C. G. JUNG. London: Routledge & Kegan Paul. 1986. Pp 179. £3.95 (pb).

Analytical Psychology: Its Theory and Practice. By C. G. JUNG. London: Routledge & Kegan Paul. 1986. Pp 224. £3.95 (pb).

Aspects of the Feminine is a collection of some of Jung's writings about women, female psychology, the image of the female as 'mother' and 'maiden' in the psychology of the male, and the image of the male and other aspects of the masculine in the psychology of the female.

Jung is most interesting and readable when freely exploring his own inner images and his ideas. Although a brilliant clinician and observer of human behaviour, he is not so scintillating when writing about the real external world. His chapter on women in Europe, written in 1927 and uncannily prophetic, is to my mind heavy and boring, although the chapter on 'The love problems of a student', given to a student organisation, runs it a close second.

Jung's theory of archetypes affords a wide and illuminating perspective on history and social movements, and it is this broad and humane view of human psychology and of history which would appeal to the readers of this *Journal.* For example, he saw the loosening of the marriage bond by modern women, and the movement towards spiritual and economic independence, as a result of women's need for true relationships finally free from the remnants of the hampering exclusiveness of mediaeval marriage.

This book includes a useful introduction to archetypal psychology, a full understanding of the 'anima' archetype in man, and a less comprehensive account of the 'animus' archetype in woman (it fell to Jung's wife to attempt the latter).

Analytical Psychology; Its Theory and Practice is a new edition of the five brilliant lectures given by Jung at the Institute of Medical Psychology (now the Tavistock Clinic) in 1935. Respected figures such as Bion, Crichton-Miller, Dicks, Emmanuel Miller, and J. R. Rees attended and made their contributions. Although edited, these lectures and ensuing discussions are basically verbatim transcripts and retain all the humour and mischief of the original events. As a summary of Jung's work and views, at least up to that time, and as a foundation for understanding Jung, they could hardly be surpassed, and anyone seeking an entrance to his work should perhaps start here.

He does not shrink from explaining his divergence from Freud in viewing much of the infantile sexual and incestuous material of dreams and fantasies as essentially symbolic and metaphorical, and his view of neurosis as potentially leading the patient in the right direction. Unfortunately he did not have time in these lectures to explain his concept of the self as a non-ego centre of the personality to which some mature persons need to relate, as perhaps formerly and even now some people relate to the archetypal images of the world's religions. For this the interested reader is directed to the later writings. Jung believed that man's salvation lies within himself, but at the same time found techniques of relating to those 'objectively' experienced impersonal or 'archetypal' images pertaining to the hereditary structure of man which are also known as God or the gods. This belief, that the centre of gravity lies within the individual and is no longer an outside object or person on whom he depends, seems to place Jung alongside the oriental religions in his outlook. But this book is not about religion except insofar as he regards the great religions as psychotherapeutic systems. It is a book by a working psychotherapist who is also deeply concerned with the fundamental nature and structure of the psyche.

J. W. T. REDFEARN, *Director of Training (Adult), Society of Analytical Psychology, London*

Life-Span Research on the Prediction of Psychopathology. Edited by L. Erlenmeyer-Kimling and Nancy E. Miller. Hillsdale, NJ: Lawrence Erlbaum Associates. 1986. Pp 310. £25.50.

This book, dedicated to the late Barbara Dohrenwend, arose from a conference she organised. We are not told when; nor are the eminent contributors introduced to the reader other than by name. The chapters are of two types: research reports, and surprisingly sharp critiques of their methodology. To these latter the researchers have no come-back. The focus is firmly on US psychiatry, with hardly a mention of similar work done elsewhere. (George Brown's studies are referred to, but without a reference.)

This is not an overview of longitudinal studies in psychiatry; nor does the book tackle the methodological issues of such studies in a systematic way. For these reasons, and also because the projects presented range over the whole life span and many, often quite specialised (occasionally even trivial), areas of psychiatry, no-one is likely to read this volume with sustained interest from cover to cover.

I especially enjoyed the chapter on the implications of the prenatal endocrine environment for later gender identity and gender role behaviour; and the long-term follow-up of the Midtown Manhatten study; as well as Lee Robins' and George Vaillant's responses to this and other projects. There were also two interesting chapters on predicting dementia in old age and on Huntington's disease.

This nicely produced book is for the library shelf rather than the individual study.

Sula Wolff, *Honorary Fellow, University of Edinburgh Department of Psychiatry*

Pathology of Eating: Psychology and Treatment. By Sara Gilbert. London: Routledge and Kegan Paul. 1986. Pp 246. £14.95.

Eating and weight are topics which seem to have fascinated researchers as much as the general public over recent years, but most of the many texts which have appeared deal only with one form of problem. By contrast, Gilbert sets out to provide a concise summary of research on the determinants and treatment of anorexia nervosa, bulimia, and obesity. Her book presents a good range of material, objectively described, with extensive and up-to-date references.

The first section is a readable and informative look at the conflicting social and cultural pressures which shape our eating. The second moves on to the pathology of eating, with brief chapters on the medical and biological causes of appetite and weight disturbance but good discussions of psychological approaches. Anorexia and obesity are both given balanced and thorough reviews, each emphasising the danger of an oversimplified view of their aetiology. Binge eating is discussed as an aspect of both of these problems, as well as in a separate chapter. This may reflect clinical fact, but occasionally gives a repetitive feel to the discussion. A fuller account of the problems in classifying eating disorders might therefore have been useful. The third section, on treatment, is most thorough with regard to obesity. The chapters on anorexia and bulimia cover the main therapeutic approaches well, but might have benefited from some additional clinical detail. There is a firm focus on research data, which limits the coverage of some current therapies, with behavioural methods being given greatest prominence.

At times, some indication of the author's working hypotheses would have been welcome to help integrate the evidence presented. Nevertheless, this remains an excellent overview of eating disorders and would be well worth reading, both for psychologists in training or in need of an update on this field and for non-psychologists who may wish to consult selected chapters to broaden their perspective on a particular problem.

Ken Gordon, *Top Grade Psychologist, Winchester Health Authority, Hants*

Depression in the Elderly: Conceptual Issues and Psychotherapeutic Intervention. By Walter S. Weinstein and Prabha Khanna. New York: Philosophical Library. 1986. Pp 193. $12.50.

This book will be of some value in the libraries of those who work clinically with the elderly depressed. However, its use will mainly be as a reference source for more detailed reading; the chapters are too brief to be of greater value either to the novice or the more experienced clinician, being mainly short reviews or summaries of other review articles.

The discussion of conceptual issues is particularly limited. I was concerned at the absence of references to the intrapsychic processes of normal mourning and its distinguishing features from complicated mourning and the morbid depressions. Without these understandings depression cannot be treated by psychotherapeutic methods. I was also disappointed at the omission of descriptions of some of the central intrapsychic developments necessary for a healthy psychological adaption to the latter phase of life, such as a change in attitude to time and death as described by Elliott Jacques in his classic paper on the mid-life crisis.

To those outside the specialty it may come as a surprise that so much has been written about psychotherapeutic interventions in the elderly (there are 25 pages of references at the end of the book). It may be even more of a surprise that the bulk of the outcome literature is optimistic about the worthwhileness of such intervention. Much of the improvement is no doubt due to a reversal of the relative neglect of the elderly by society and the caring professions – indeed, psychotherapy is used in a very broad sense in this book. Those unfamiliar with the differing therapies will not get any idea from the book of the differences in therapist skills required to get a 'reminiscence group' together from those required to conduct psychoanalytic psychotherapy.

In spite of its limitations, this book will do nothing to discourage those interested in working psychologically with the elderly depressed, and hopefully it will lead them to explore the literature further.

BRIAN MARTINDALE, *Consultant Psychiatrist in Psychotherapy, Paddington Centre for Psychotherapy, London*

Theatres of the Mind: Illusion and Truth on the Psychoanalytic Stage. By JOYCE MCDOUGALL. London: Free Association Books. 1986. Pp 301. £25.00 (hb), £9.95 (pb).

Joyce McDougall is one of the most important and creative of contemporary psychoanalytic theoreticians. Being based in France, she is able to draw on some of the French psychoanalytic thinking and render this in a form which is intelligible to her British and American colleagues. She develops here the metaphor of drama and of analysis as a stage in which characters and scenarios from the patient's mind can be enacted. Although dealing with various fundamental kinds of disturbances, it is in the areas of perversion and psychosomatic states where she makes her most original and stimulating contributions.

She refers to perversions as "neosexualities"; her idea is that the person who creates a perversion has reinvented human sexuality, creating a new "primal scene" – one in which all the rules and roles are specified exactly, leaving nothing to chance, and which has the function of keeping intolerable anxiety at bay. The basic anxiety concerns various metaphorical levels of 'castration', involving restriction, humiliation, or even fantasied threats to life itself. In the perverse scenario many kinds of substitutive acts of castration are carried out in a playful way, thus mastering the anxiety through illusion. For example, a perverse man needed to see traces of his partner's faecal matter to bring him to the heights of sexual excitement; as a child his mother had given him frequent enemas and also regularly humiliated him by sending him out in the park with his soiled underpants tied round his head. According to McDougall, these complex sexual creations have an immense hold over the perverse person's life because they function to maintain mental equilibrium and are attempts to deal with profound insults to the sense of self and personal agency and autonomy.

In McDougall's view, psychosomatic patients are also attempting to ward off overwhelming anxiety of a primitive and psychotic nature. She describes how these patients are astonishingly out of touch with feelings and desires. For example, a patient was puzzled by how he could know whether he desired a particular woman or not, although he had "noticed" that he gained an erection in her presence; the erection was as meaningless to him as any other physiological process. McDougall suggests that in their infancy and childhood these patients were not helped to recognise, acknowledge, and understand emotions, which therefore became frightening. These emotions, which were never given access to language, were not recognised as belonging to the self, and nor, at one level, was the body. Threatening emotions were therefore not allowed entry to the conscious mind, but instead were given expression in the body.

Like McDougall's previous book, *Plea for a Measure of Abnormality,* this is rich, evocative, and clinically useful.

PHIL MOLLON, *Regional Psychologist in Psychotherapy (Top Grade), Dryden Road Hospital, Gateshead*

Neighbours: The Work of Philip Abrams. Edited by MARTIN BULMER. Cambridge: Cambridge University Press. 1986. Pp 282. £9.95 (pb).

It is common to view neighbours or neighbourhoods as persons or places living or located nearby. However, they also are part of a process able to enhance the health and well-being of those who live within them. In times when economic changes worsen conditions for many persons, or when austerity policies reduce funds for needed services, neighbours take on particular importance.

Neighbours collects the unpublished work of the late Philip Abrams, the distinguished social scientist who directed pioneering research on neighbours who join families and friends in informal interactions that serve social functions; on neighbouring as a process of everyday relations which sometimes (but not always) appear altruistic and reciprocal in nature; and on neighbourhood care as an organised way to mobilise resources for indigenous social support and mutual responsibility.

This book, edited and integrated with excellent commentaries by Martin Bulmer, includes theoretical and structural perspectives on neighbouring, case studies of streets in various English locations, general propositions, and policy statements from empirically-based research. The overall conclusion is that neighbours can help reduce isolation, promote interaction, and serve psychosocial functions with epidemiological effects in improving health and well-being. The suggestion is that neighbours can have curative and preventive therapeutic possibilities for the elderly, infirm, and other members of society.

The book makes a major contribution to knowledge of neighbours in relation to social dimensions of mental health. It includes an impressive theoretical review which breaks new ground in psychosocial studies, empirical research on neighbouring in different locations, and provocative discussion of policy issues of informal and formal neighbourhood services. It combines qualitative and quantitative methodology, employs microscale social studies and multiunit aggregate analyses, and reports research on the scope and quality of practice.

Bulmer reports that Abrams admired American studies of neighbourhoods, but it is difficult to find more important work on the topic than the research reported here. This book should win an appreciative audience among readers in several social sciences, academic disciplines, and professional fields in addition to those in mental health.

Barry Checkoway, *Associate Professor of Social Work, University of Michigan, Ann Arbor, USA*

Marriage and Family Enrichment. Edited by Wallace Denton. New York: The Haworth Press. 1986. Pp 125. $29.95.

Marriage and Family Enrichment has grown out of the marriage guidance movements. Beginning in Europe and the USA early in the 1960s, a number of counsellors and therapists (including David and Vera Mace in London) sought to develop methods to help couples actively attend to the untapped, underdeveloped strengths in their marital relationships. The aim was to reach people *before* severe problems arise and threaten a breakdown or motivate an approach to a doctor, social worker, or counsellor with a 'sick' marriage.

A gradual accumulation of experience in this preventative, 'well marriage' based approach to relationships has made it possible for Denton and his co-contributors to put together a practical little book. Those who are not familiar with the notion of enrichment will find the book a good introduction to the history, philosophy and practice of this group-based approach. Of particular note are the chapters which systematically explore how leaders can be trained, couples or families selected to participate in the enrichment groups, and timetables ('ground plans') made for the group meetings. The limitations to this approach are also discussed. The chapters are clearly set out, with illustrative case examples.

This book would be of direct interest to those working in community psychiatry, or where professional resources are turned toward prevention and self-help. The enrichment approach could be viewed as a contribution to the growing interest in a holistic/healing approach to couples and families.

Peter Fullerton, *Institute of Marital Studies, London*

Clinical and Pharmacological Studies in Psychiatric Disorders. Edited by Graham D. Burrows, Trevor R. Norman and Lorraine Dennerstein. London: John Libbey. 1986. Pp 394. £26.00.

This book represents selected papers from the 14th CINP Congress, held in Florence in 1984. It is divided into eight sections, covering affective disorders (both pharmacological aspects and clinical studies); anxiety, panic disorders and stress; psycho-neuroendocrinology; schizophrenia; basic neuropharmacology; and Alzheimer's disease and psychogeriatrics. The emphasis is on new developments in biological psychiatry. There are papers on desipramine and central adrenoceptor function, circadian rhythms, biochemical correlates of L-deprenyl, antidepressants, and brain levels of thyroid-releasing hormone. There are a number of other basic neurochemical studies of central and peripheral biogenic amine receptors and theoretical discussions of classification, genetics, and diagnosis. The major emphasis, however, is on new pharmacological therapies. There are reports of new clinical trials of established medications, as well as new drugs, such as alprazolam, fluvoxamine, toloxatane, verapamil, sulpiride, and femotatine.

Almost all the papers are concise and highly technical, reporting a large amount of data. This will appeal to those readers who want to make up their own minds about whether conclusions are warranted from the data presented. Despite their complexity, the papers are well edited and readable. Some of them have extensive introductions reviewing the background for these studies, which readers new to the field will find useful.

Overall this is a well written, well edited and well presented book covering many of the most exciting areas of biological psychiatry. At £26.00 it represents very good value.

Michael A. Reveley, *Senior Lecturer and Consultant in Psychiatry, London Hospital Medical College, London*

Medical Mimics of Psychiatric Disorders. Edited by I. Extein and M. S. Gold. Washington, DC: American Psychiatric Press. 1986. Pp 198. $15.95.

This little book is part of a *Progress in psychiatry* series, each volume of which contains texts based on the papers of a selected American Psychiatric Association conference symposium. Its first two chapters, on "the psychiatrist as physician" and on neurological screening, promote careful physical examination supplemented by the comprehensive battery of investigations more fashionable in the US than here. Physical disorders are seen as diagnostic alternatives to psychiatric disease, and the psychosomatic dimension virtually ignored.

The section on seizure disorders, which takes up almost a third of the book, explores in detail the kindling model as an explanation for the behavioural manifestations of epilepsy and for the efficacy of ECT and anticonvulsants in affective disorder. Briefer chapters on hypothyroidism and on the HPA axis describe clearly, if unexcitingly, the abnormalities of neuroendocrine function found in depression, but with scant discussion of why these associations should occur.

Perhaps most interesting is the editors' contribution exploring the neurotransmitter receptor changes underlying the frequent emergence of depression following alcohol or opiate (particularly methadone) withdrawal. The final chapter, on drug-induced psychiatric disorders, is long on lists and woefully short on mechanisms, or even on critical discussion of the many individual case reports invoked.

This book does not live up to its title's considerable promise, as is perhaps inevitable in a collection of conference papers not conceived for publication as a comprehensive text. It is, however, attractively produced and well referenced, and merits a library browse, although I would not recommend it for individual purchase.

C. L. E. KATONA, *Senior Lecturer in Psychiatry, Middlesex Hospital Medical School, London*

Help Starts Here. The Maladjusted Child in the Ordinary School. By I. KOLVIN, R. F. GARSIDE, A. R. NICOL, A. MACMILLAN, F. WOLSTENHOLME and I. M. LEITCH. Andover: Associated Book Publishers. 1981. Pp 436. £9.95.

This is a recently produced soft-back version of the seminal work of the Newcastle Child Psychiatry Department on the evaluation of psychotherapy in childhood using a community-based sample. The work is already well-known to many child mental health professionals, both academic and clinicians. The advantage of this new edition is its price, which will make it available to many individuals who previously would have felt unable to purchase a single volume for themselves.

The work represents an important landmark in the evaluation of child psychotherapy. The book is well laid out and describes clearly and concisely the method and background to the project, the treatment approaches used, results, and conclusions, and includes a useful and relatively clear technical appendix.

The design of the project was sophisticated, employing multi-method screening and outcome measures and including a control group at risk for psychiatric disorder. The study compared four types of treatment (behaviour modification, nurturing a teacher-aide programme, parent counselling and teacher consultation, and group therapy) applied to children of two age groups: 7–8 and 11–12. Three aspects of the results should persuade researchers and clinicians in child mental health (and perhaps adult mental health as well) to read the book in its entirety. Firstly, treatment is clearly and significantly effective compared with no treatment. Secondly, briefer treatments had the better outcome. Thirdly, the most cost-effective (and therefore the most likely to find realistic application in general child mental health services) were the most efficient in terms of outcome.

A further intriguing issue is the suggestion that the personal qualities of the therapist need to be different in different settings, as indicated by the finding that extraversion and assertiveness were important and had positive correlations with good outcome from treatment, whereas empathy and warmth in therapists did not. The authors infer that in the hurly-burly of school life a greater degree of assertiveness in therapists is required than in the clinic setting, where empathy and warmth may be more necessary.

It could be argued that the study does not adequately reflect the two mainstay psychological treatments that are probably most widely used in child psychiatry clinics: family therapy and individual child psychotherapy. It may thus be difficult to make direct comparisons between this study and clinic-based studies; however, the principles by which this study was carried out and the general issues it raises lay the foundations for the evaluation and design of psychotherapy studies in clinic and community settings.

The book is well laid out and provides something for all types of readership. The research worker will be able to chew over the selection of the treatments, the children, design, methods, and the outcome measures employed to evaluate the change. The clinician will be able to see how the Newcastle team successfully employed well-established psychological treatments in the community and pioneered the way for mental health professionals to work in ordinary schools. The overriding impression is the book is essential reading for child psychiatrists and their colleagues and will become a source reference book for psychotherapy research. Given the continuous vociferous debates concerning the merits of psychotherapy in adults it might be useful if they turn back the covers of this book and consider its contents!

I. M. GOODYER, *Senior Lecturer in Child & Adolescent Psychiatry, University of Manchester*

Anxiety Disorders of Childhood. Edited by RACHEL GITTELMAN. Chichester: John Wiley & Sons. 1986. Pp 271. £20.00.

This book, edited by an eminent American child psychiatrist with a long-standing interest in affective disorders in childhood, focuses specifically on anxiety disorders in the young. It is laid out in twelve clear chapters, two contributed by Gittelman herself; other contributors include other leaders in the speciality such as Werry, Puig-Antich, and Shaffer. All chapters are well-researched, with extensive and up-to-date bibliographies.

The first is an interesting but fairly brief look at anxiety-like disorders in young non-human primates by Stephen Suomi. Next, three chapters are given over to developmental issues, epidemiology, and diagnosis and assessment. A common, almost universal pattern of different specific fears at different ages is traced, but it is emphasised that definitional and measurement problems abound and there are some conspicuously neglected areas, including longitudinal research into the natural history of fears, anxieties, and worries in non-clinical populations, and the impact of family factors on children's fears. No epidemiological data are available specifically regarding anxiety disorders in children, which limits this chapter to a review of the existing (confusing) data on symptom prevalence. Accurate assessment using structured interviews, behavioural, and subjective rating scales is bedevilled by the low reliability among different measures and measurers, and the inadequacy of both the ICD–9 and DSM–III in diagnostic classification is highlighted.

A chapter each is devoted to the association of anxiety disorders with obsessive-compulsive disorder and affective disorder: in the former case little overlap is found; in the latter it is high, but mainly in the sense that a majority of depressed or dysthymic children show anxiety symptoms. There is a lower incidence of affective symptoms in children with primary anxiety disorders. A brief chapter reviews learning theories of anxiety, and a longer one psychodynamic theory. The last three chapters consider treatment, but are restricted to three approaches only: pharmacotherapy, behaviour therapy, and individual psychoanalytic psychotherapy. It will come as no surprise to anyone to learn that drugs have little place. The advice on behaviour therapy is basic, clear, and helpful to a non-psychologist, and similarly that on psychoanalytic psychotherapy to a non-analyst, although couched in general terms rather than focused specifically on treatment anxiety symptoms. One of Gittelman's own contributions is a chapter on correlates and outcome, rather oddly situated in the middle of the book – it might have been more logically placed at the end.

Although clearly set out, easy to read, and a most useful reference source for any aspect of anxiety in childhood, I am not sure that this book succeeds in its aim of defining and clarifying this range of disorders. However, it certainly succeeds in pinpointing aspects in urgent need of further research, including the natural history of fears in non-clinic populations, prospective studies of outcome and, most importantly, the need to tease out separation anxiety from other presentations.

Overall, I would expect most child psychiatrists and many clinical psychologists to find this book helpful.

PATRICIA AINSWORTH, *Lecturer in Child and Adolescent Psychiatry, University of Manchester*

Talking to a Stranger: A Consumer's Guide to Therapy. By LINDSAY KNIGHT. London: Fontana. 1986. Pp 321. £2.95.

This paperback aims to inform the potential psychotherapy patient about the various psychotherapies, to enable them to choose a helpful therapist and the most appropriate therapy. The author is a journalist and mental health film producer, and this book is written from her personal experience of psychotherapy and from numerous discussions with therapists and therapy researchers. The book is well laid out, easy to read, and informative. Each chapter succeeds in conveying the experience and process of the various therapies from the patient's point of view, and provides useful information about mechanisms of change and realistic ideas of the results that can be achieved. There are chapters outlining the nature of psychotherapy, its indications and contra-indications, psychoanalysis, the various schools of dynamic psychotherapy, group therapy, counselling, and sex therapy. The last two chapters indicate how to choose a therapist, and provide a guide on the resources available in the UK. Potential patients may find the chapter on theoretical underpinnings of the various therapies off-putting, however, as it is jargon-ridden and tries to explain too much in too little space.

The main limitation of this book is its scope. Behaviour therapy and cognitive therapy receive mentions only in a brief glossary. Other therapies such as transactional analysis, personal construct therapy and Gestalt therapy receive no mention at all. In addition, the description of therapies suggests that many patients require years of therapy for useful results to be achieved. This is an unnecessarily disheartening message for potential patients, and suggests a lack of awareness of the range of brief focal therapies.

This is a book to recommend to potential patients.

NORMAN D. MACASKILL, *Consultant Psychiatrist with a Special Interest in Psychotherapy, Whiteley Wood Clinic, Sheffield*

Memory and Amnesia: An Introduction. By ALAN J. PARKIN. Oxford: Basil Blackwell. 1987. Pp 227. £22.50 (hb), £7.95 (pb).

This welcome introductory textbook about amnesia is written by a British experimental psychologist who is involved in investigating amnesic conditions. The first four chapters consider current psychological theories about the nature of memory. The remaining seven chapters consider amnesia under the following headings: clinical assessment, the amnesic syndrome, explaining amnesia, ageing and dementia, transient memory disorders, psychogenic disorders of memory, and remediation. The book rounds off with suggestions for further reading, a list of references cited, an author index, and a subject index. The book succeeds in its aim of introducing the contemporary field of work on amnesia. For people unfamiliar with this field, here is a useful entry; for workers in the field, the book is a helpful overview of the field as a whole.

IAN M. L. HUNTER, *Emeritus Professor, University of Keele*

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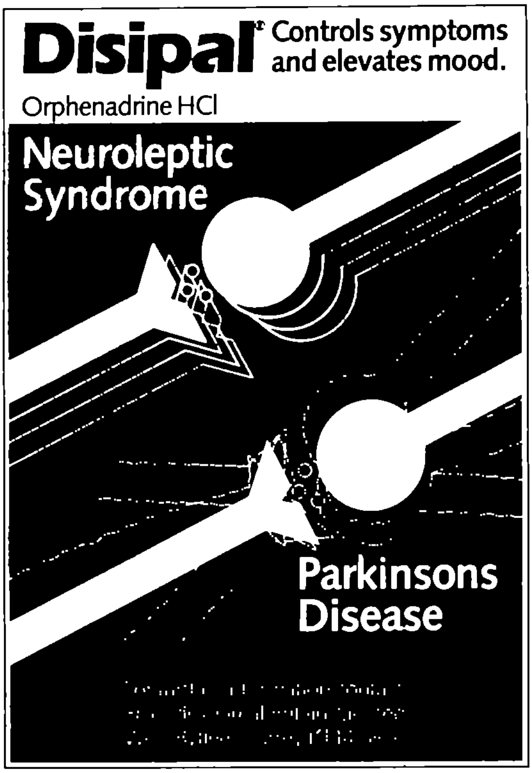

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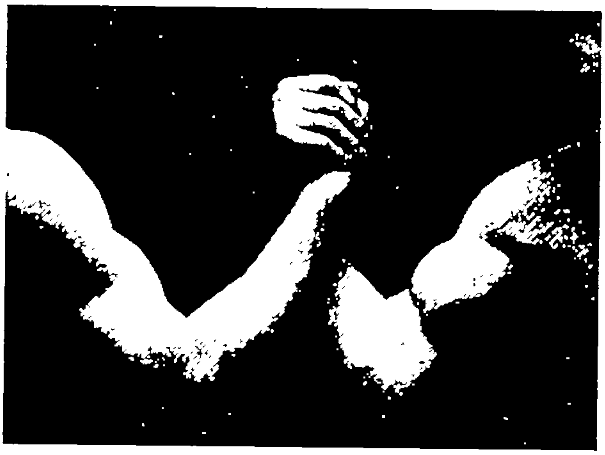

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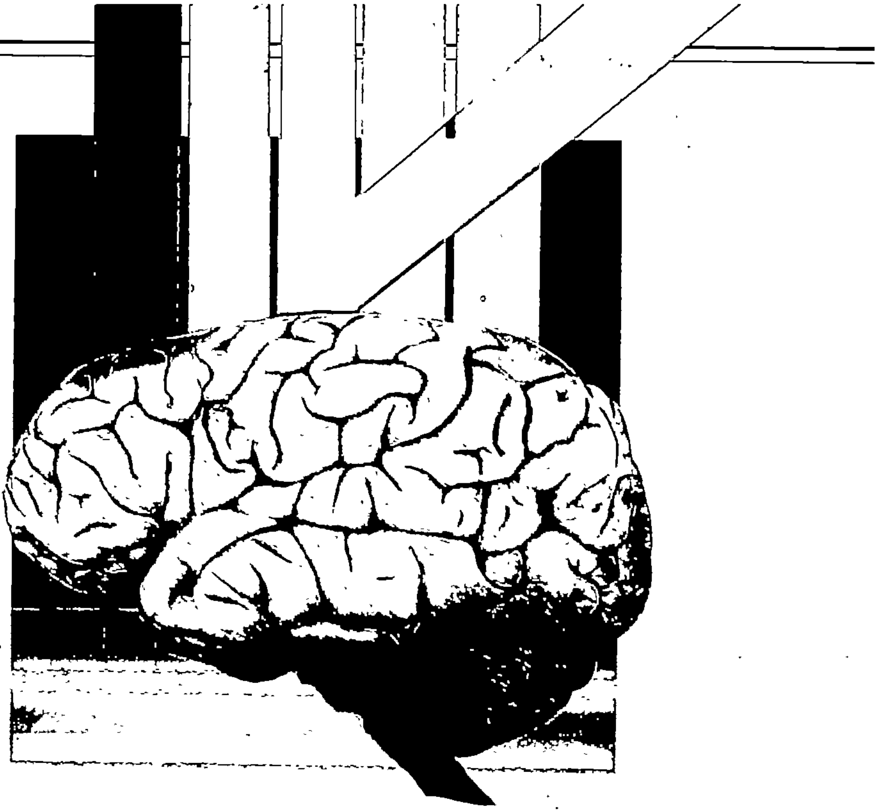

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1984;69: (Suppl 311) 7–30. 2. Edwards J G et al Br J Psychiatry 1980, 137. 522–529. 3. Munk-Anderson E Acta Psychiatr Scand 1984, 69 (Suppl 311) 31–41. 4 Schiff A A Br
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PETER F. LIDDLE

The relationships between symptoms in 40 schizophrenic patients, selected for persistence of symptoms, were examined. The symptoms segregated into three syndromes: psychomotor poverty (poverty of speech, lack of spontaneous movement and various aspects of blunting of affect); disorganisation (inappropriate affect, poverty of content of speech, and disturbances of the form of thought); and reality distortion (particular types of delusions and hallucinations). Both the psychomotor poverty and disorganisation syndromes were associated with social and occupational impairment; in particular, the psychomotor poverty syndrome was associated with impairment of personal relationships, and the disorganisation syndrome with poor self-care and impersistence at work.

The clinical diversity of schizophrenia has prompted many attempts to define subtypes of the illness. Several recent attempts have been based on the distinction between positive and negative symptoms (Crow, 1980*a,b*; Andreasen & Olsen, 1982; Lewine *et al*, 1983). Negative symptoms reflect deficiency of a mental function which is normally present, for example blunting of affect and poverty of speech. Positive symptoms, such as delusions and hallucinations, reflect aberrant mental activity.

Recent attempts to measure negative symptoms stem from the work of Venables (1957) and Wing (1961). They found that "withdrawal in chronic schizophrenic patients is a unidimensional variable which can be reliably measured. It is defined by such items as: underactivity, slowness, lack of conversation, lack of friends, avoidance of others, poor personal hygeine, carelessness about appearance, and lack of interests" (Venables & Wing, 1962). Electrophysiological evidence that withdrawal was associated with high arousal led Venables and Wing to suggest that withdrawal might be a protective mechanism adopted to cope with a decreased ability to filter sensory input. However, emphasis shifted to the role of social factors in the genesis of negative symptoms, after the demonstration by Wing & Brown (1970) that these symptoms are common in an unstimulating environment, and furthermore are less severe after an "increase in the richness of the social environment" (Wing, 1978).

Subsequently, the evidence that schizophrenic patients with negative symptoms are more likely to have abnormalities such as cerebral ventricular enlargement and intellectual impairment (Huber, 1957; Johnstone *et al*, 1978; Andreasen & Olsen, 1982) has regenerated interest in the role of organic factors. Crow (1980*a*) proposed two types of schizophrenia: type I is characterised by positive symptoms, usually in the setting of an acute illness, while type II is characterised by negative symptoms and indices of cerebral damage, and is usually chronic. Crow did not regard the two types as mutually exclusive, but as independent dimensions reflecting different underlying pathological processes (Crow, 1980*b*).

Andreasen & Olsen (1982) adopted a different view of the relationship between positive and negative symptoms, regarding them as characteristic of two different types of illness. In a sample consisting of consecutive hospital admissions satisfying modified DSM–III criteria for schizophrenia (American Psychiatric Association, 1980), they demonstrated a negative correlation between the occurrence of positive and negative symptoms. They were able to assign about one-third of their cases to each of the positive and negative categories. These two groups of patients differed in many respects, including course of illness, evidence of cerebral ventricular enlargement, and evidence of cognitive impairment.

It is possible that the negative correlation between positive and negative symptoms demonstrated by Andreasen & Olsen (1982) arises from a difficulty in eliciting positive symptoms in the presence of negative symptoms. Another possibility is that the pattern of correlations was influenced by the mixture of acute and chronic cases in their sample. Unless subjects are homogeneous with regard to chronicity, in the sense of having symptoms of comparable persistence over time, there will be a tendency for symptoms to segregate into groups on the basis of their tendency to persist. Although segregation of symptoms on the basis of persistence is of itself important, the question of whether persistent positive symptoms segregate from persistent negative symptoms in a separate issue. It is relevant to the further question of whether symptom type *per se*, rather than the persistence of symptoms, distinguishes between different types of schizophrenia. There are also unresolved issues concerning the assignment of symptoms to the negative symptom group. Andreasen (1982) included inappropriate affect in the negative group, with acknowledged misgivings, whereas Crow (1980*a*) regarded it as a positive symptom. Andreasen also included lack of volition and anhedonia in the group of negative symptoms, and assessed these unobservable attributes of the mental state by rating impairment of self-care, occupational performance, and social function. Since many different attributes of the mental state might in principle contribute to these observable impairments, the validity of this means of assessment remains to be established, and might well be limited to particular samples of patients. Finally, while Andreasen and Crow agree that disorders of the form of thought such as derailment and incoherence are positive symptoms, Lewine *et al* (1983) assign such symptoms to the negative symptom group.

This study attempted to delineate the segregation of symptoms in a sample of schizophrenic patients selected in a manner intended to discriminate in favour of cases with persistent symptoms. No prior assumptions about which symptoms should be regarded as positive or negative were made. The study also included an assessment of cognitive function and of neurological abnormalities, which will be reported separately.

Method

Selection of patients

The following criteria were adopted, with the object of selecting a sample of schizophrenic patients in a manner which discriminated in favour of those with persistent symptoms.

(a) The patient must have satisfied DSM–III criteria for the diagnosis of schizophrenia at some point in the course of the illness, and at the time of interview exhibit at least one symptom from section A or C of those criteria.

(b) The time since onset of illness must be 3–18 years. The lower limit of three years was chosen because Huber *et al* (1975) found that the pattern of illness is generally established by this stage. Cases with an extremely long duration of illness were excluded so as to decrease the likelihood that any observed segregation of symptoms would merely reflect the effects of prolonged illness; a 15-year range of illness duration was the minimum permitting the recruitment of an adequate number of cases from the population available.

(c) The patients must be under regular psychiatric care, but have had no increase in neuroleptic medication or readmission to hospital within the preceeding six months.

(d) Age must be less than 55 years, so as to minimise the influence of the effects of aging on cognitive function.

From all of the patients receiving in-patient, day-patient, or regular out-patient care from one of the seven clinical teams at Warneford Hospital, Oxford, Littlemore Hospital, Oxford, and Fair Mile Hospital, Wallingford, or receiving day or out-patient care at the Eldon Road Day Hospital, Reading, 47 patients satisfying the above criteria were identified. Of these, 7 did not reply or refused to participate in the study. The remaining 40 comprised 12 in-patients, 12 day-patients and 16 out-patients; there were 31 males and 9 females. The mean duration of illness was 10.5 years, and the mean age was 35 years (range 21–54 years).

Assessment of schizophrenic symptoms

Symptoms were rated according to section II of the Comprehensive Assessment of Symptoms and History (CASH) (Andreasen, 1983), and sections 13–20 of the Present State Examination (PSE) (Wing *et al*, 1974). Two separate rating scales were employed because the scope of the definition of each symptom, and the range of symptoms assessed, influence the detectable pattern of correlations between symptoms, and it is therefore valuable to be able to compare the results obtained using different rating scales.

CASH was used because it is designed to provide a comprehensive rating of schizophrenic symptoms. It incorporates the Scale of the Assessment of Negative Symptoms (SANS), which consists of five sub-scales: affective flattening, alogia, lack of volition, anhedonia, and attention impairment. In addition, CASH includes comprehensive ratings of disorders of the form of thought, and of delusions and hallucinations. Most of the items in CASH are rated on a scale of 0–5, reflecting grades of severity.

A number of SANS items demand a detailed knowledge of the patient's lifestyle. Andreasen (1983) recommends that where possible this information be obtained from a third party, such as nursing staff. In this study, with in-patients in the minority, this was not always possible, However, several interviews with each patient were required to allow the administration of the cognitive test battery in a manner that avoided prolonged periods of testing, and these interviews were structured so as to allow a substantial time for informal conversation, including standard questions about the patients' activities and relationships.

The PSE was employed because it has been standardised and because its glossary provides precise definitions of the items to be rated. To avoid prolonged formal examination of the patients, the full PSE was not administered. Questions from sections 13–15 were used to elicit evidence of delusions and hallucinations, and scores were assigned to all syndromes concerned with delusions or hallucinations in the PSE syndrome checklist. Sections 18–20 of the PSE, which involve observations of behaviour, affect, and speech, were also completed.

Data analysis

The symptom ratings were subjected to factor analysis using the program FACTOR from the Statistical Package for the Social Sciences (Nie *et al*, 1975). Initial factors were extracted by the method of principal factors, and subjected to oblique rotation. CASH and PSE ratings were analysed separately.

In the case of CASH rating single items, rather than sub-scale scores, were employed as units of analysis, to avoid having to make any initial assumptions about the relationships between items. Since major disparity in the frequency of occurrence of symptoms necessarily restricts the upper bound of the range of the correlation between items, only those items rated as definitely present in more than 10% of the patient sample were included. One consequence of this limitation was the exclusion of some specific types of delusions and hallucinations, most notably Schneiderian first-rank symptoms, because these items were individually quite rare in this sample.

Since it was not assumed that the items of an individual sub-scale could be taken as measures of the single construct embodied in that sub-scale, a problem arose in the case of the symptoms lack of volition, anhedonia, and attentional impairment. As these symptoms are represented by SANS sub-scales consisting of items measuring self-care, occupation, and social function, which might be regarded as measures of performance in daily life rather than symptoms, the relationship between these items and symptoms was examined separately by determining the Pearson correlation coefficients between these items and symptom factor scores.

In the case of the PSE ratings, the units of analysis representing sections 18–20 consisted of single items. These items record ratings of behaviour, affect, and speech observed at interview. The relatively fine distinctions between individual items in sections 13–15 of the PSE, which record delusions and hallucinations, made it necessary to employ syndrome scores embracing these items as units of analysis. In general, PSE syndromes contain a small number of symptoms which are judged on clinical grounds to be similar. These use of PSE syndrome scores as units of analysis has been justified statistically using data from the US-UK Diagnostic Project and the International Pilot Study of Schizophrenia (Wing *et al*, 1974) which confirmed that PSE symptoms have a satisfactory degree of statistical association with the syndromes to which they have been allocated.

In comparing the results of the factor analysis of the two sets of symptom ratings, it is necessary to bear in mind that the prior existence of statistically justified syndromes in the case of the PSE permits analysis of the relationships between delusions and hallucinations at a different level from that for the CASH ratings. Also, differences in the scope of the definitions of items in different rating scales is inevitable, and is the reason why two rating scales were employed in the first place.

Results

Factor analysis of CASH ratings

The factor analysis of CASH ratings revealed three factors, each having high loadings in a separate group of symptoms (Table I). The first factor loads heavily in poverty of speech, decreased spontaneous movement, and four items which might be regarded as measures of blunting of affect: unchanging facial expression, paucity of expressive gesture, affective non-responsivity and lack of vocal inflection. This group of six symptoms is designated the psychomotor poverty syndrome.

The second factor has heavy loadings in inappropriate affect, poverty of content of speech, and four items which are recognised disturbances of the form of thought: tangentiality, derailment, pressure of speech, and distractibility. This group of six symptoms is designated the disorganisation syndrome.

The third factor has heavy loadings in auditory hallucinations of voices speaking to the patient, delusions of persecution and delusions of reference. This group of symptoms is designated the reality distortion syndrome.

In this sample of patients, the three syndromes are not strongly correlated. There is a weak negative correlation ($r = -0.22$) between the factors associated with the psychomotor poverty syndrome and the disorganisation syndrome, and the other correlations are near zero. The syndromes are thus not mutually exclusive. For example, the probability that a patient has symptoms of the psychomotor poverty syndrome is independent of whether he has symptoms of the reality distortion syndrome.

Factor analysis of PSE ratings

The factor analysis of PSE ratings (Table II) reveals a pattern of segregation of symptoms similar to that obtained for the CASH ratings. The first factor loads heavily in the incongruous affect, poverty of content of speech, incoherence of speech, distractibility, and self-neglect. These symptoms are similar in character to the symptoms of the disorganisation syndrome. The second factor loads heavily in restricted quantity of speech, slowness, and blunted affect, symptoms which are similar in character to those of the psychomotor poverty syndrome.

There is evidence of a sub-division with regard to delusions and hallucinations. A third factor has heavy loadings in the PSE nuclear syndrome (which consists mainly of Schneiderian first rank symptoms) and in sexual and fantastic delusions. This group of symptoms is designated the disintegrative reality distortion syndrome. A fourth factor has substantial loadings in delusions of persecution, delusions of reference, and grandiose delusions; this group is designated the integrative reality distortion syndrome. However, delusions of reference and persecution also have a moderate dependence on the third factor, and hallucinations of voices speaking to the patient depend, albeit weakly, on both factors. Furthermore, the third and fourth factors have a moderate positive correlation ($r = 0.29$) in this sample of patients. There is therefore some justification for considering delusions and hallucinations as a group of related symptoms, irrespective of content.

TABLE I
Factor loadings obtained by factor analysis of CASH symptom ratings

Symptoms	*Factor 1*	*Factor 2*	*Factor 3*
Psychomotor poverty syndrome (high loadings on factor 1)			
Poverty of speech	0.80	−0.01	−0.03
Decreased spontaneous movement	0.95	−0.04	−0.03
Unchanging facial expression	0.85	−0.01	0.05
Paucity of expressive gesture	0.97	0.02	−0.04
Affective non-responsivity	0.82	0.02	−0.00
Lack of vocal inflection	0.90	−0.20	−0.05
Disorganisation syndrome (high loadings on factor 2)			
Inappropriate affect	0.19	0.84	0.09
Poverty of content of speech	−0.08	0.57	0.01
Tangentiality	−0.05	0.94	0.03
Derailment	−0.05	0.94	0.04
Pressure of speech	−0.10	0.61	0.08
Distractibility	−0.00	0.81	0.01
Reality distortion syndrome (high loading on factor 3)			
Voices speak to patient	0.04	−0.07	0.67
Delusions of persecution	−0.19	0.06	0.51
Delusions of reference	0.13	0.04	0.84
Somatic delusions	−0.03	−0.03	0.03

Self-care, occupation and social function

The correlations between the items of the lack of volition, anhedonia and attentional impairment sub-scales of SANS, which are ratings of self-care, occupation and social function, and the factor scores derived from the factor analysis of CASH ratings are shown in Table III. If the factor scores are regarded as measures of the corresponding syndromes, it is apparent that in this sample of patients both the psychomotor poverty and disorganisation syndromes are associated with impairment of self-care, occupation, and social function. However, the syndromes differ in the pattern of their association with these impairments.

The most widespread and severe disturbance is associated with the disorganisation syndrome. In particular, self-care and persistence at work are poor. The psychomotor poverty syndrome is associated with relatively greater impairment of recreational interests and ability to make friendships. There were no significant correlations between factor scores and age, duration of illness, or duration of hospital in-patient care.

Associations between syndromes and medication

The reality distortion syndrome was correlated to current dose of neuroleptic drugs ($r=0.31$, $P<0.10$), current use of anticholinergic drugs ($r=0.53$, $P<0.01$), and current use of benzodiazepines ($r=0.35$, $P<0.05$). The disorganisation syndrome was related to total duration of neuroleptic treatment only ($r=0.31$, $P<0.10$), and the psychomotor poverty syndrome is not associated with any of the variables related to medication.

Discussion

The segregation of symptoms in this sample of patients provides some support for the previously postulated positive-negative dichotomy. Firstly, the symptoms belonging to the Psychomotor Poverty syndrome (blunting of affect, poverty of speech and decreased spontaneous movement) are similar in character to the symptoms that are common to the

TABLE II

Factor loadings derived by factor analysis of PSE ratings

Symptom	*Factor 1*	*Factor 2*	*Factor 3*	*Factor 4*
Disorganisation syndrome (high loading on factor 1)				
Incoherence of speech	0.93	−0.14	0.01	−0.00
Poverty of content of speech	0.68	−0.06	0.20	−0.34
Distractibility	0.74	−0.16	0.03	−0.01
Incongruous affect	0.72	−0.00	0.05	−0.00
Self-neglect	0.80	0.24	−0.16	0.30
Psychomotor poverty syndrome (high loading on factor 2)				
Restricted quantity of speech	0.01	0.84	0.06	−0.01
Slowness	−0.04	0.95	−0.01	−0.05
Blunted affect	−0.08	0.79	−0.06	−0.08
Disintegrative reality distortion syndrome (high loading on factor 3)				
PSE nuclear syndrome[1]	0.05	−0.09	0.54	0.10
Sexual & fantastic delusions	−0.00	−0.04	0.93	−0.17
Voices to the patient	0.04	0.17	0.27	0.23
Integrative reality distortion syndrome (high loading on factor 4)				
Delusions of reference	−0.03	0.04	0.39	0.49
Delusions of persecution	−0.04	−0.17	0.28	0.33
Grandiose delusions	0.04	−0.10	−0.05	0.64

1. Consists of passivity experiences, alienation of thought, primary delusions and third person auditory hallucinations.

TABLE III

Correlations between factor scores obtained by factor analysis of CASH symptom ratings and measures of self-care, work performance and social function from the lack of volition, anhedonia and attentional impairment sub-scales of SANS

	Factor 1 (psychomotor poverty)	*Factor 2 (disorganisation)*	*Factor 3 (reality distortion)*
Poor grooming and hygiene	0.13	0.56****	0.15
Impersistence at work	0.06	0.38**	0.18
Physical anergia	0.52***	−0.28	0.16
Recreational interests and activities	0.29*	0.23	−0.07
Sexual interest and activity	0.21	−0.21	−0.14
Ability for intimacy	0.03	0.31*	−0.04
Relationships with friends and peers	0.35**	0.19	0.06
Social inattentiveness	0.09	0.39**	0.01

*P <0.10; **P <0.05; ***P <0.01; ****P <0.001.

negative symptom groups defined by Wing & Brown (1970), Crow (1980*a,b*), Andreason (1982) and Lewine *et al* (1983). Secondly, in accord with previous studies, this study demonstrates that delusions and hallucinations belong to a group which can be separated from the psychomotor poverty group of symptoms.

The evidence provided by the factor analysis of PSE ratings that there is a division between types of delusions and hallucinations is slender, but gives partial support to the view that first rank symptoms should be distinguished from other types of delusions and hallucinations. Nonetheless, it is important to bear in mind that the factors associated with the disintegrative reality distortion syndrome and the integrative reality distortion syndrome were positively correlated in this sample, suggesting that all hallucinations and delusions constitute a related group of symptoms.

The major difference between the findings of this study and other recent investigations of positive and negative symptoms is the identification of the disorganisation syndrome as a separate syndrome. It consists of symptoms which other investigators have variously assigned to either the positive or negative symptom groups. Disorders of the form of thought constitute a major part of this syndrome. Bleuler (1911) considered it likely that disturbances of association are primary symptoms of schizophrenia, and subsequently many clinicians have accorded them a central place among schizophrenic symptoms. Harrow *et al* (1983) attempted to assess whether disordered form of thought identifies a subgroup of schizophrenic patients with poor outcome. They found that thought disorder persisting after discharge from hospital identified a subgroup with poor outcome, but some patients without thought disorder also had a poor outcome. They concluded that "severe thought disorder is one of several major features of schizophrenia". The present study supports their conclusion.

The relationships between the three syndromes has important implications for predicted relationships between symptoms in different patient samples, and also for hypotheses concerning pathological processes in schizophrenia. In this sample the psychomotor poverty and reality distortion syndromes are not mutually exclusive; some patients have symptoms from both syndromes. This suggests that in a patient sample including a substantial number of cases without any schizophrenic symptoms, correlations between symptoms from these two syndromes would be positive. On the other hand, in a patient sample selected in a manner that favours cases having a single pure syndrome, the correlations between symptoms from the psychomotor poverty and the reality distortion syndromes would be expected to be negative.

For example, the findings of this study predict a negative correlation between the psychomotor poverty syndrome and the reality distortion syndrome in a sample such as that studied by Andreasen & Olsen (1982). Their sample included some acute cases, likely to have reality distortion symptoms only (or perhaps disorganisation symptoms as well), and other cases likely to have psychomotor poverty symptoms only, because DSM-III criteria were modified to accept severe poverty of speech in place of positive symptoms in the mandatory symptoms section.

Furthermore, Pogue-Geile & Harrow (1984) recently studied schizophrenic symptoms recorded 18 months after the patients' discharge from hospital. It is likely that their sample of patients was similar to the patients assessed in this study with regard to persistence of symptoms. In contrast to Andreasen & Olsen, they found a negligible correlation between positive and negative symptoms, consistent with the findings of the current study.

The observation that a patient can have symptoms from more than one syndrome suggests that the syndromes do not represent distinct types of schizophrenia, but instead reflect discrete pathological processes occurring within a single disease. This suggests that there is a fundamental abnormality in schizophrenia, which in any individual case might be associated with one or more of three distinct pathological processes, depending on the patient's constitution and current environment. In principle, the putative pathological processes might be either a cause or a consequence of the fundamental abnormality.

The existence of patients who currently have symptoms of only the psychomotor poverty syndrome, despite selection criteria which demand symptoms from either the reality distortion or disorganisation syndromes at some stage in the illness, demonstrates that the syndromes detectable at any one time can change during the course of the illness. The absence of significant correlation between syndrome scores and duration of illness makes it unlikely that the syndromes follow each other in a specific sequence.

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The Efficacy of Electroconvulsive Therapy in the Treatment of Schizophrenia

A Comparative Study

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The efficacy of ECT was investigated in a double-blind trial. Twenty-two patients with schizophrenia received trifluoperazine and were randomly allocated to receive eight real or eight simulated ECTs. In the first eight weeks, the group receiving real ECTs showed significantly more improvement as measured on the Brief Psychiatric Rating Scale. However, the groups showed no significant differences from the twelfth week onwards. The superiority of real ECT was not confirmed at the end of six months.

Although electroconvulsive therapy (ECT) is often employed in the treatment of schizophrenia, particularly in combination with neuroleptic drugs, its efficacy has rarely been evaluated by controlled investigations. It is generally believed that ECT is beneficial in ameliorating catatonic symptoms and excitement. However, by itself ECT does not seem to lead to sustained improvement, and relapses frequently occur (Kalinowsky *et al*, 1969). Childers (1964) and Smith *et al* (1967), in two controlled studies, observed that the combination of ECT with antipsychotic medication accelerated the process of recovery in schizophrenics. Kelly & Sargant (1965) and Roth *et al* (1966) showed that when a combination of ECT and drugs was used in the treatment of chronic schizophrenic patients, the improvement was immediate but transitory. In a study of chronic schizophrenic patients, Gambill & Wilson (1966) felt that the ECT–drug combination was only marginally better than placebo.

Many studies on the efficacy of ECT in conjunction with neuroleptic medication have attempted to assess whether the combination is more beneficial than either treatment alone. Ford & Jameson (1955), Kinross-Wright (1955) and Rhode & Sargant (1961) found an ECT-neuroleptic drug combination to be superior to ECT or drug therapy alone. These studies also indicated that patients receiving an ECT-drug combination were discharged earlier from the hospitals. However, Berg *et al* (1959) and Lesse (1959) were unable to demonstrate the superiority of an ECT-neuroleptic drug combination and cautioned that such combined treatment at times had serious side-effects.

May & Tuma (1965) compared five modalities of treatment, in a carefully selected sample of schizophrenic patients, and observed that ECT alone occupied an intermediate position between drugs and psychotherapy alone. May (1968) stated that ECT could not be considered a serious rival to pharmacotherapy or psychotherapy in schizophrenia. In a five-year follow-up study, May *et al* (1976) reported that ECT-treated patients spent shorter periods of time in hospitals after the initial period of treatment in comparison with medicated patients. Rahman (1968), El-Islam *et al* (1970), and Weinstein & Fischer (1971) found ECT in conjunction with drugs to be definitely superior to each treatment alone. In two critical reviews, Turek (1973) and Abrams (1975) commented that most studies were poorly designed, contained heterogeneous groups of patients, and had innumerable treatment modalities and variable lengths of follow-up; they found the data relating to the superiority of ECT-drug combinations unacceptable, and advocated more rigorous research. Salzman (1980) was of the opinion that the literature regarding the therapeutic efficacy of ECT in schizophrenia did not meet contemporary research criteria, and felt that this subject needed more research. Pippard & Ellam (1981), in a report to the Royal College of Psychiatrist, conceded that the status of combined neuroleptic drug-ECT treatment in schizophrenia was far from settled, and identified it as an area for further research.

Since these reviews, Taylor & Fleminger (1980) and Brandon *et al* (1985) have reported much better-designed studies describing the effectiveness of ECT-drug combinations in schizophrenia. Both studies compared ECT-drug combinations with simulated ECT-drug combinations, and showed that those receiving the former improved significantly more in the initial weeks but that this advantage was totally lost with the passage of time.

It appears, therefore, that combined ECT and neuroleptic therapy increases the pace of improvement. The literature also indicates that the more rapid improvement curtails duration of hospitalisation and that subsequent hospital admissions also tend to be of shorter duration.

Notwithstanding the moral and emotional issues involved in the administration of ECT, the possibility that ECT-drug combinations might be cost-effective in terms of duration of hospital stay and maintenance drug therapy offers certain advantages in the management of schizophrenic patients in a country such as India, where resources to cope with a problem of the magnitude of schizophrenia are sparse and under continuous strain. The present study was undertaken to assess the comparative efficacy of an ECT-trifluoperazine combination with a combination of trifluoperazine and simulated ECT. The study was double blind, and had a six-month follow-up period.

Method

Patients attending the out-patient department or admitted to the psychiatric ward were screened for selection purposes. Those satisfying the following criteria were taken up for the study:

(a) fulfilling Research Diagnostic Criteria (Spitzer *et al*, 1978) for a definite diagnosis of schizophrenia
(b) not received ECT before
(c) age at onset of illness less than 40 years
(d) duration of illness not greater than two years.

Using the Present State Examination (Wing *et al*, 1974) one of us interviewed all the patients and randomly allocated them to either ECT or simulated ECT. An informed consent was requested from all patients and their relatives for inclusion in the study. This had two parts: consent for receiving general anaesthesia and muscle relaxants; and information and explanation regarding the purpose and design of the study. It was explained to the patients and their relatives that only half of the patients in the study were likely to receive ECT, although neither the patients nor the other investigator, who evaluated the patients before, during and after the course of treatment, knew to which treatment group a particular patient belonged.

All the patients received a course of eight ECTs or simulated ECTs with intravenous thiopentone sodium (150–300 mg) and scoline (25–30 mg). All the patients were premedicated with atropine. The treatments were administered twice weekly. Trifluoperazine (20 mg/day in divided doses) was given to all the patients, and in the first 16 weeks no change in drug dosage was permitted. In addition, benzhexal hydrochloride, which is an antiparkinsonian agent, and nitrazepam were given as and when necessary. To facilitate drug compliance and follow-up, the drugs were given free of charge to all poor patients.

Using the 18 item version of the Brief Psychiatric Rating Scale (BPRS) (Overall & Gorham, 1962; Overall, 1981) and a clinical global impression scale (CGIS), KRA blindly assessed all the patients before the start of the treatment and at the end of 2,4,6,8,12,16,20, and 26 weeks. The CGIS was based on the clinical impressions of KRA, who rated each patient for the degree of mental illness and for the total improvement since admission, both on a scale of 0–7. The data was analysed by the chi-squared test for non-parametric variables and by the *t*-test for comparison of means. All the *t*-tests were one-tailed. The study commenced on 1 November 1983, and six month follow-up was completed for all cases by 31 December 1984.

Results

The total number of patients at the start of the trial was 28, of which only 22 completed the treatment and six month follow-up; the analysis and results pertain only to these 22 patients. Of the six patients who did not complete the trial, three were in the ECT group and three in the simulated ECT group. One female patient in the simulated ECT group was withdrawn from the study by the clinician in charge because of a deterioration in her condition, and the other five completed the treatment but did not attend regularly for follow-up assessments. Only three patients (one in the simulated ECT and two in the ECT group) were admitted to hospital; the remaining 25 received treatment on an out-patient basis. No patient was given any additional ECT in the study period. For the first 16 weeks drug dosage was held constant. The dose of trifluoperazine was then adjusted according to the needs of the patient, but no patient was given more than 20 mg/day even after 16 weeks. The patients were not given any other medication except nitrazepam and benzhexal hydrochloride.

The two groups did not differ significantly on age, sex, duration of illness, paranoid/non-paranoid diagnosis, or initial BPRS and severity of illness (CGIS) scores. The response to treatment, as indicated by the BPRS scores, is shown in Table I. The ECT group showed more rapid improvement than the simulated ECT group, but by the 12th week the scores for both the groups were similar and did not show any significant difference thereafter till the 26th week.

TABLE I
Inter-group comparison of BPRS scores

Week	*ECT group (n=11)*		*Simulated ECT group (n=11)*			
	Mean	*s.d.*	*Mean*	*s.d.*	t^1	*P*
0	22.36	4.80	22.09	6.07	0.110	NS
2	13.45	6.99	19.55	7.33	1.904	0.05
4	10.54	4.33	17.09	7.79	2.324	0.05
6	8.64	3.11	15.91	8.23	2.613	0.01
8	8.36	4.03	15.0	9.26	2.079	0.05
12	6.64	3.58	12.64	8.32	2.095	0.05
16	6.73	3.66	10.36	7.70	1.346	NS
20	6.36	2.83	9.55	8.97	1.073	NS
26	5.36	3.58	10.36	9.02	1.629	NS

1. One-tailed test.

Mean scores for severity of illness and the degree of improvement as assessed on CGIS for both groups are shown in Figs 1 and 2.

The rate of improvement within the group was examined more closely by comparing the change in scores over successive two week intervals. The ECT group showed continuous improvement at a significant level until the 12th week (Table II). The simulated ECT group also showed significant changes in scores at 0–2 weeks and 2–4 weeks. However, in this group the improvement was not continuous but stepwise.

Discussion

Our study demonstrated that in two groups of patients comparable for age, sex, and duration of illness, the group receiving ECT-trifluoperazine showed significantly greater and more rapid improvement than the simulated ECT group. The drugs given to both the groups were strictly controlled. The group receiving simulated ECT also started to manifest significant improvement from the second week. However, the rate of improvement in the two groups was different. The ECT group displayed continuous improvement until the 12th week; the simulated ECT group showed stepwise improvement over the same period. Despite the faster and more sustained rate of improvement of the ECT group, the two groups did not show any significant difference from the 12th week onwards. At the 26th week the ECT group appears to have fared better. Otherwise, our findings are in agreement with those of Taylor & Fleminger (1980) and Brandon *et al* (1985).

Our study is somewhat limited in measures of outcome. Except for the BPRS we have not employed any other measures, particularly measures to assess depression. However, on the BPRS our patients did not score highly on depressed mood, retardation or hopelessness. Therefore, it could be argued that the improvement found was not specifically linked to improvement in depression, but was associated

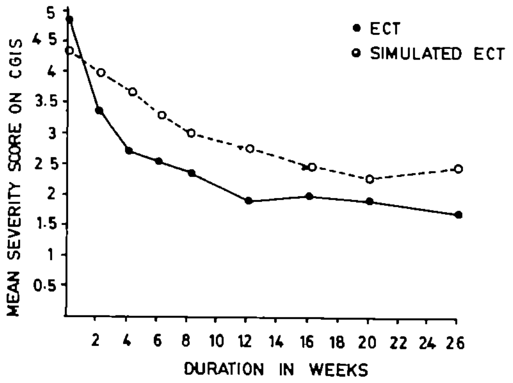

FIG. 1. Inter-group comparison: mean severity rating score on CGIS.

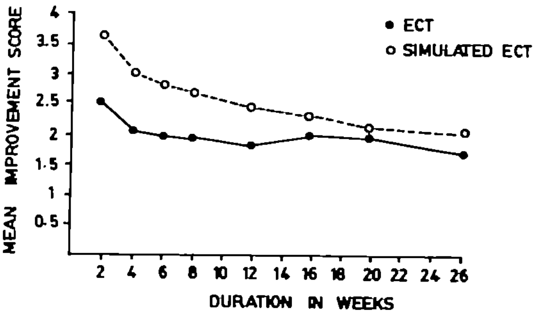

FIG. 2. Inter-group comparison: mean improvement score on CGIS.

TABLE II
Rate of improvement in BPRS scores within the groups

Weeks	*ECT Group*				*Simulated ECT group*			
	Mean difference	*Standard error of difference*	t^1	*P*	*Mean difference*	*Standard error of difference*	t^1	*P*
0–2	8.91	1.75	5.10	0.01	2.54	0.97	2.62	0.01
2–4	2.90	1.21	2.40	0.05	2.46	0.93	2.63	0.01
4–6	1.90	0.869	2.18	0.05	1.18	1.24	0.95	NS
6–8	0.27	1.08	0.25	NS	0.91	0.86	1.04	NS
8–12	1.72	0.83	2.07	0.05	2.36	0.66	3.54	0.01
12–16	0.09	0.73	0.112	NS	2.27	0.99	2.28	0.05
16–20	0.36	0.47	0.76	NS	0.81	0.65	1.23	NS
20–26	1.0	0.40	2.5	0.05	0.81	0.69	1.15	NS

1. One-tailed test.

with improvements in psychotic symptoms of a schizophrenic nature.

It is commonly accepted that ECT has no role in chronic schizophrenia, and that patients who are young and have been ill for less than a year are likely to benefit most from ECT (Salzman, 1980; Kendell, 1981; Taylor, 1981). The group studied by us was younger than average and had not been ill for more than two years. As such, our patients would have improved without ECT. However, the differential rates of improvement of the two groups lend support to the view that ECT leads to more rapid recovery.

Although our study is comparatively small, in our national context and interest we have demonstrated that ECT is probably cost-effective, as it speeds up the rate of improvement in schizophrenic patients. Because of the small size of the sample it was not possible to study the relationship between response to ECT and clinical symptoms. To further define and delineate the role of ECT in the therapy of schizophrenic patients, more studies involving larger number of patients and longer follow-up periods are needed. Until then, some of the questions pertaining to the efficacy of ECT in the treatment of schizophrenia will remain unanswered.

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Expressed Emotion and Schizophrenia in North India

I. Cross-Cultural Transfer of Ratings of Relatives' Expressed Emotion

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A bilingual rater was trained in English in a technique of assessing relatives emotional attitudes to patients, and was then required to rate material in Hindi without any further experience. This strategy revealed that the rating of critical comments, hostility and positive remarks could be transferred from English to Hindi without distortion. There were problems with the remaining two scales, over-involvement and warmth, but these were due to technical issues connected with rating and not to cross-cultural distortion.

Measurement of expressed emotion

In order to study the emotional aspects of relationships between psychiatric patients and their relatives, a semi-structured interview, the Camberwell Family Interview (CFI), was developed in the 1960s (Brown & Rutter, 1966; Rutter & Brown, 1966). Originally this was administered to relatives at the time the patient was admitted to hospital.

The interview is tape-recorded, then rated from the recording. Ratings are made on five principal scales: *criticism, hostility, over-involvement, warmth* and *positive remarks. Criticism* is measured by the total number of critical comments made in the course of the interview. A critical comment is carefully defined in a manual used in conjunction with the CFI. *Hostility* is defined as either a generalisation of criticism (1), rejection of the patient as a person (2), or a combination of generalisation and rejection (3). *Over-involvement* can be detected and rated either from the respondent's behaviour during the interview (as with the other scales), or from reported behaviour outside the interview. It comprises several different elements, any one of which can give rise to a high rating. They are: exaggerated emotional responses, unusually self-sacrificing and devoted behaviour, over-protection that is inappropriate considering the patients' age, and an inability to maintain a boundary between the subject's existence and the patient's. Over-involvement is rated on a global scale extending from 0 to 5. *Warmth* refers not to a general quality in the respondent's personality, but to an expressed emotion specifically about the patient. It too is rated on a global scale which runs from 0 to 5. A *positive remark* is a statement which expresses praise, approval or appreciation of the behaviour or personality of the patient. As with criticism, the number of positive remarks made throughout the interview is counted.

On all five scales, raters are required to pay attention not only to the content of relatives comments but also to vocal aspects of their speech, including rate, volume and tone. Despite the niceties of judgement demanded by this latter task, high levels of inter-rater reliability (about 0.8) are normally achieved after attendance at a training course of three to four weeks.

Implications of high EE

The value of this assessment has been established in a series of studies, reviewed by Kuipers (1979). Relatives were characterised as high on expressed emotion (EE) if they scored above certain levels for criticism or over-involvement, or if they expressed any hostility towards the patient. It was found that schizophrenic patients whose illness remitted and who were discharged from hospital to live with high-EE relatives were much more likely to relapse during a nine-month follow-up than those who returned to low-EE relatives (Brown *et al*, 1972; Vaughn & Leff, 1976a). These findings, originally derived from studies of families in London, have been replicated in a study in California (Vaughn *et al*, 1984); and evidence for a causal association between high EE and schizophrenic relapse has been provided by a controlled trial of intervention in high-EE families (Leff *et al*, 1982). In one of the earlier studies the degree of criticism expressed by relatives was also found to predict the outcome of neurotic depression (Vaughn & Leff, 1976a), although the threshold for high and low criticism was much lower than that for schizophrenia. The finding has recently been replicated by Hooley *et al* (1986).

The importance of relatives' EE in psychiatric disorders is now beyond doubt in English-speaking cultures on both sides of the Atlantic. Considerable interest in this measure has also been expressed in other European cultures, and research workers from the Federal Republic of Germany, Holland, Austria and Belgium have been trained in this technique.

The WHO project on outcome of schizophrenia

In 1976 the World Health Organization initiated a multi-centre collaborative research programme on the outcome of schizophrenia, in which the EE measure has been incorporated. The project, on *Determinants of Outcome of Severe Mental Disorder*, is a prospective epidemiological, clinical and social study of schizophrenia and related conditions in different cultures, coordinated by the World Health Organization (Geneva). The study is financed jointly by WHO, NIMH, and twelve field research centres (FRCs) in Aarhus (Denmark), Agra and Chandigarh (India), Cali (Colombia), Dublin (Ireland), Honolulu and Rochester (USA), Ibadan (Nigeria), Moscow (USSR), Nagasaki (Japan), Nottingham (UK) and Prague (Czechoslovakia). All of the FRCs are participating in a 'core' epidemiological and clinical study and sub-groups of centres are carrying out a number of special sub-studies designed to test specific hypotheses about the course of schizophrenia, its social sequelae, and its associations with other diseases.

The 'core' study has set out to identify, over a 24-month period, all the psychotic patients (aged 15–54) making a first-lifetime contact with any 'helping agency' located in or near each of the twelve geographically defined catchment areas in the project. The clinical and diagnostic assessment of these individuals should permit an estimate of the incidence of schizophrenia and related syndromes. The one-year and two-year follow-up assessments of the patients should help to evaluate previous findings of differences in the prognosis of schizophrenia in the developing and developed countries (WHO, 1979).

In addition to the 'core' study, sub-groups of field research centres are undertaking certain special investigations. The one with which this paper is concerned focuses on the effects of patterns of emotional expression and communication in the family on the short-term course of schizophrenic illnesses.

The WHO study of EE and outcome

The centres chosen for a study of EE and outcome of schizophrenia were Aarhus in Denmark and Chandigarh in North India. Danish culture is similar to that in England, both being industrialised Western countries with a well-developed welfare system, whereas India provides a marked contrast. Furthermore, the two-year follow-up of the *International Pilot Study of Schizophrenia* (WHO, 1979) revealed that schizophrenic patients from certain developing countries, including India, had a better prognosis than those from centres in Europe and North America. This raised the possibility that more tolerant family attitudes to illness and handicap might explain or contribute to the better outcome in developing countries. A study of EE in Chandigarh could test this hypothesis.

In training candidates from other European countries to rate EE, we had assumed that the languages and cultures were similar enough for the technique to be transferable from English; but we considered that the cultural and linguistic differences between London and Chandigarh were too great for us to be comfortable with this assumption. The main language spoken in Chandigarh is Hindi, the tonal qualities of which are quite distinct from those of English. Since tone is a crucial element in rating EE, it was obviously essential to devise some method of assessing the transfer of the technique from English to Hindi.

Method

At first sight it may seem that routine tests of inter-rater reliability are sufficient to determine whether the assessment of EE can be transferred from one culture to another. However, a moment's consideration shows that this is not so. Suppose that two bilingual researchers are trained to an acceptable level of reliability in rating EE in English. They then return home and start rating in their native language. They check the reliability of their own ratings in their own language and find it is acceptably high. Unfortunately this does not prove the point. In the process of adapting the interviewing and rating techniques to their own cultural setting, it is inevitable that they will discuss between themselves the difficulties encountered. This could readily lead to a steady drift away from the techniques as taught. Because of their continued dialogue, the two raters could attain a high level of reliability in their own language whenever tested. However, this would by no means guarantee that what they were doing in their own language was the same as they had been taught to do in English. One way to check on this is to train another bilingual subject to rate EE reliably in English and then require him, before he has any experience of rating EE in the second language, to rate tapes of interviews made by the other raters in their native language. Since the 'naive subject', as we shall call him, has had no opportunity to discuss the technique with the other raters, he has no option but to transfer the rating rules that he has learned in English to the second language as best he may. If he then reaches a high level of reliability with the other raters, it demonstrates that what they are doing in their native language must be very close to what was taught in English.

This was the approach we took to test the transfer to the interviewing and rating techniques for EE from English to Hindi. The local researchers (DKM and HB) were trained in English initially, one in London and one in Chandigarh, and achieved acceptable reliability levels on all scales compared with the London centre. The naive bilingual subject chosen was AG, who had no previous acquaintance with the techniques in either language. He was trained in English in the London centre to a satisfactory level of inter-rater reliability on the various scales. He was then required to rate tapes of interviews conducted in the Chandigarh centre in Hindi by the local researchers. It was originally planned that the whole exercise would be carried out on taped interviews sent from Chandigarh. Unfortunately some of the tapes were lost in transit, only six arriving safely in London. These were rated by AG as planned. As it happened, AG decided to visit Chandigarh at this time, and advantage was taken of this opportunity to sit in on four live interviews conducted by the local researchers and to rate them. AG fully understood the necessity to refrain from discussing interviewing or rating techniques with the local researchers until the exercise was completed, and he strictly observed this principle.

Results

Immediately following the training course, AG rated a standard set of tapes used to test reliability. He was found to have reached high levels of reliability on criticism, hostility and over-involvement, but not on warmth and positive remarks. Further attention was given to training AG and in early 1982, just before he visited Chandigarh, AG was asked to rate ten additional English tapes as a final check on his reliability. The product moment correlation coefficients between his ratings and those of the London centre researchers were as follows: critical comments 0.86, hostility 0.90, over-involvement 0.91, warmth 0.20, positive remarks 0.40. As before, his ratings of criticism, hostility and over-involvement were completely satisfactory, whereas those of warmth and positive remarks did not reach even a minimum standard of acceptability. This was not such a drawback as it might seem, since earlier studies had suggested that only the first three scales were associated with the outcome of schizophrenia, and only these three had been used to categorise relatives as high- or low-EE. However, we considered it important to measure these elements of EE in the Chandigarh relatives, since it was feasible that their relationship with outcome might differ considerably from that found in the Anglo-American studies.

The problem AG experienced in becoming reliable on the warmth and positive remarks ratings was unexpected, as trainees are usually able to master all the EE scales by the end of the course. Moreover this problem failed to yield to a further period of training, but because of AG's imminent departure for Chandigarh, we were not able to try other approaches.

The raw scores on the Hindi interviews rated by AG and the two Chandigarh raters (DKM and HB) are displayed in Table I. The data from the six taped interviews which were sent to England have been combined with those from the four interviews at which AG was present. An inspection of this table reveals that the three raters differ little in ratings of critical comments, hostility and positive remarks; whereas for over-involvement and warmth differences are more marked.

For critical comments the intraclass correlation coefficient (ICC) for the three raters is 0.87, a satisfactory value. There are some differences between the raters but these are not consistent and are small compared to differences between the subjects. A positive score for hostility was recorded on only four of the thirty assessments. However, there was complete agreement between the three raters on the presence or absence of hostility in nine of the ten cases. There was considerably more disagreement on emotional over-involvement. The intraclass correlation coefficient (0.21) is not statistically significant. We conclude that the three raters were rating differently, and a visual inspection of the results shows that AG consistently rated higher than either DKM or HB, who differed little from each other. The ICC for warmth is only 0.49, which is barely significant. Once again, AG rated differently from DKM and HB, giving a lower rating on all but one subject. The agreement between the three raters is satisfactory for positive remarks, the ICC being 0.86.

The current criteria for assigning relatives to a high-EE category are: (1) six or more critical comments, or (2) any positive rating on hostility, or (3) a score of three or more on over-involvement. When these criteria are applied to the material in Table I, there is complete agreement between the three raters on the assignment of two relatives to a high-EE category and seven relatives to a low-EE category. In one case (number 7) there is a disagreement, with the two Chandigarh raters scoring the subject low on critical comments while the naive rater scored above the cut-off point for a high-EE rating.

Discussion

It is evident from these results that the rating of critical comments can be transferred satisfactorily from English to Hindi. This is reassuring, since critical comments contribute most to the assessment of EE. In Vaughn & Leff's (1976a) study, 18 out of 21 families (86%) were assigned to a high-EE group on the basis of six or more critical comments.

We cannot be so certain about the transferability of ratings of hostility since so few subjects in the Chandigarh reliability exercise were given a positive rating on this scale. However, the findings suggest that this assessment is also likely to be transferable across the linguistic frontiers without significant distortion.

The position regarding over-involvement is less

TABLE I
Raw scores of individual raters

Subject	*Critical comments*			*Hostility*			*Over-involvement*			*Warmth*			*Positive remarks*		
	DKM	*HB*	*AG*	*DKM*	*HB*	*AG*	*DKM*	*HB*	*AG*	*DKM*	*HB*	*AG*	*DKM*	*HB*	*AG*
1	2	2	2	0	0	0	0	0	1	3	3	2	2	1	2
2	12	14	14	1	2	2	0	0	0	1	1	0	0	0	0
3	0	0	1	0	0	0	1	1	1	3	3	3	4	4	4
4	2	2	2	0	0	0	0	0	1	3	3	2	1	1	1
5	4	4	4	0	0	0	0	0	0	3	3	2	0	0	0
6	1	2	2	0	0	0	0	0	0	2	2	1	1	1	1
7	3	1	8	0	0	0	0	0	0	0	1	1	0	1	1
8	0	1	4	0	0	0	0	2	2	3	2	1	2	2	2
9	1	0	1	0	0	0	0	0	2	3	4	1	1	1	2
10	12	15	9	0	1	0	0	0	1	2	2	1	0	1	2
Mean	3.7	4.1	4.7	0.1	0.3	0.2	0.1	0.3	0.8	2.3	2.4	1.4	1.1	1.2	1.5
Intraclass correlation coefficient		*0.87*			*0.79*			*0.21*			*0.49*			*0.86*	

satisfactory. AG noted some degree of over-involvement in a higher proportion of subjects than did DKM, and HB was much closer to her Chandigarh colleague in this respect than to AG. Since AG showed no tendency to over-rate on this scale in English, we can conclude that the Chandigarh researchers were likely to be under-rating in Hindi. However, this is not a consequence of cultural differences, as far as we can judge from AG's observation of the live interviews in Chandigarh and discussions with the researchers there.

He reported that judgements of over-involvement were almost exclusively based on overt expression *during the interview* of over-concern or excessive anxiety about the patient's condition. Relatively little attention was paid to reports of behaviour by the subject that reflected over-involved attitudes. This clearly represents a shift of emphasis subsequent to training: during training raters are instructed to give equal weight to the expression of emotional over-involvement and reports of behaviour that suggest it. This drift away from the original convention apparently affected both raters and is likely to have led to under-estimates of over-involvement in the Chandigarh sample of relatives.

The possibility of a drift away from the rating conventions inculcated during a training period has to be guarded against with any rating scale. A number of precautions can be taken. It is very helpful to have a detailed manual of rating instructions available, which trainees can take away with them: this is the case with EE assessment. If there are two trainees in one centre, they should be instructed to carry out inter-rater reliability checks at regular intervals. Finally, trainees should send a proportion of their taped interviews, say one in ten, to the training centre for monitoring by the trainers. This creates extra work for the training centre but is essential for quality control of rating techniques. Of course in the case of Chandigarh it was not possible for the trainers to assess the reliability of ratings of interviews conducted in Hindi. Only the technique of using a 'naive' bilingual rater can reveal the kind of drift that we detected in respect of the over-involvement scale.

Our assessment of the reliability of the transfer of warmth ratings from English to Hindi was clouded by the difficulty AG experienced in mastering this scale in English. He consistently tended to under-rate warmth when assessing English tapes, and the same phenomenon is evident in his Hindi ratings, since both

Chandigarh raters scored higher than he did for the subjects in this study. AG's relatively low reliability with the Chandigarh raters on the warmth scale is almost certainly due to his difficulty with these ratings in both languages. Whether there is an additional problem in transferring this assessment from English to Hindi cannot be determined from these data.

The results for the 'positive remarks' scale are peculiar, since AG attained a reliability on this scale of no more than 0.40 with the English raters, but reached highly satisfactory levels of agreement with the Chandigarh workers in terms of the intra-class correlation coefficient. How is it possible for him to have achieved so close a unanimity with the Chandigarh raters when he appeared to have difficulties with this scale in English? We can only speculate that he grasped the principle behind the rating of positive remarks but was unable to operationalise it satisfactorily in English. However, when he applied it in Hindi without any prior experience of using it in this language he employed it in a very similar way to the other Hindi-speaking raters.

The findings of this study leave us reasonably satisfied that the EE components of critical comments, hostility and positive remarks can be transferred intact from English to Hindi. In the case of over-involvement it was discovered that a drift had occurred away from the rating conventions established during the training. A drift of this kind could just as easily have occurred with researchers rating in English, and does not indicate a specific problem in transferring the rating procedure across languages. However, the presence of this rating bias prevented us from determining whether an additional cross-cultural distortion existed. The difficulties the bilingual rater experienced in achieving reliability on the warmth scale were equally evident in English and Hindi: they therefore represent an idosyncratic problem and not a cross-cultural one.

We conclude that the strategy of using a 'naive' bilingual rater is an effective method of evaluating the cross-cultural transfer of a rating technique. However it is clear that this strategy is viable only if the naive rater is able to become highly reliable on all·the scales to be compared across cultures. This exercise was undertaken to assess the validity of comparing the effects of relatives' EE on the course of schizophrenia in Chandigarh with previous work in English-speaking cultures. We have complete confidence in the ratings of critical comments in Hindi and reasonable confidence in the hostility scores, but some doubts about the ratings of over-involvement. These are the three components which have contributed to the assessment of high-EE relatives in the past. Insofar as critical comments have made the major contribution to the detection of high EE, we consider that a direct comparison between the findings of the Chandigarh study of EE and the work in Britain and North America is meaningful.

II. Distribution of Expressed Emotion Components among Relatives of Schizophrenic Patients in Aarhus and Chandigarh

N. N. WIG, D. K. MENON, H. BEDI, J. LEFF, L. KUIPERS, A. GHOSH, R. DAY, A. KORTEN, G. ERNBERG, N. SARTORIUS and A. JABLENSKY

We measured the components of expressed emotion among two samples of relatives of first-contact patients from Aarhus (Denmark) and Chandigarh (India). The Danes were very similar in most respects to samples of British relatives, whereas the Indian relatives expressed significantly fewer critical comments, fewer positive remarks, and less over-involvement. Within the Chandigarh sample, city-dwellers were significantly more expressive than villagers of all EE components except over-involvement.

As stated in the first paper of this series of three, a study of the relationship between relatives' expressed emotion (EE) and outcome of schizophrenia was undertaken in Aarhus in Denmark and Chandigarh in North India. It was assumed that the techniques of interviewing and rating were readily transferable from English to Danish, these being closely related languages. The same assumption could not be made about English and Hindi, the predominant language in Chandigarh.

The issue was studied by using a bilingual rater. It emerged that the EE components of critical comments, hostility and positive remarks could be transferred reasonably intact from English to Hindi. It was considered likely that over-involvement was under-rated by the Chandigarh researchers, but an adjustment can be made for this bias by lowering the cut-off point for high over-involvement (Wig *et al*, 1986a). These findings allow us to make a direct comparison of the distribution of EE components between samples of relatives from Aarhus, Chandigarh and London.

Method

Research workers in the Danish and Indian centres visited London for training in the interviewing and rating techniques. Satisfactory inter-rater reliability was achieved on all EE scales by the end of the training. In both centres, as part of the standard procedure for trainees, a further set of standard tapes was rated by all researchers after they had returned home, as a check on their rating technique. These proved satisfactory for each individual. In addition, the two London researchers made separate visits to Chandigarh to train an additional researcher and to study the application of the technique under local conditions.

Selection of samples

In the studies conducted in London (Brown *et al*, 1972; Vaughn & Leff, 1976a), patients with a diagnosis of schizophrenia were included on admission to hospital, regardless of length of history. The patients in Aarhus and Chandigarh on the other hand form part of an incidence study co-ordinated by WHO and limited to those making their first contact with psychiatric facilities. In the British samples, however, no differences were found in the distribution of EE components between first-admission patients and the rest, so that it is not unreasonable to compare the Aarhus and Chandigarh samples with the complete British sample.

The Danish and Indian samples comprised all those patients aged 15–54 who contacted a psychiatric service for the first time within a specified two-year period and who could be classified as either schizophrenic or suffering from paranoid or other psychotic states (excluding typical affective psychosis). Each patient was interviewed with the Present State Examination (PSE) and the data obtained were processed with CATEGO program (Wing *et al*, 1974). In addition to a CATEGO class, each patient was assigned a diagnosis by the psychiatrists at the field research centre on the basis of information collected with standardised history and social data schedules in addition to the PSE: we shall refer to this as the patient's 'centre diagnosis'.

Any patient who lived with at least one key relative with whom they maintained daily face-to-face contact was eligible for inclusion in the EE sub-study. Relatives were asked to take part in the Camberwell Family Interview (CFI) (Brown & Rutter, 1966; Vaughn & Leff, 1976b).

The great majority of British patients not living on their own were either in parental or marital households. In the latter case the spouse was interviewed; in the former, both parents were interviewed if available. In Chandigarh many patients (37%) lived in extended families, which gave the researchers a choice of relatives to interview. They were guided in their choice by considering who spent most time with the patient, as determined from a preliminary discussion with the patient. In a number of families the patient's mother-in-law, sister-in-law, or son-in-law was interviewed, in addition to a parent or spouse. In Aarhus, as in London, the spouse was the interviewee in marital households; but in parental households only one parent was interviewed, due the pressure of other research work. In the majority of instances (75%) the mother was chosen as the respondent. In the earlier British Studies the convention was adopted that where two relatives in one household were assessed for EE, the higher rating on each index was used to categorise the family as high or low in EE. The absence of data from a second parent in the Aarhus group means that this sample is likely to under-represent the higher levels of EE and is not entirely comparable with the other two samples. Nevertheless, comparison is worthwhile as long as this reservation is kept in mind.

Results

Danish sample

In Aarhus, 28 patients included in the EE study were given a centre diagnosis of schizophrenia; of these 25 were assigned to CATEGO class SOP, which corresponds to a diagnosis of schizophrenia (SOP encompasses classes S+, S?, P+, P? and O+), one was assigned to class O?, and the other two were classified as D+, equivalent to depressive psychosis. Both the depressed patients were diagnosed by the centre psychiatrists as depressive psychotic at the two-year follow-up. Nevertheless, for the purposes of comparability with the Chandigarh data they have been retained in the initial Aarhus sample. Thus EE data are available for 28 Aarhus relatives.

Indian sample

Of the 78 patients in Chandigarh given a centre diagnosis of schizophrenia, 50 also received a CATEGO class of schizophrenia. Of these, 31 were assigned to class S+, indicating that they exhibited at least one Schneiderian first rank symptom. The CATEGO class does not take account of the history, which is of considerable importance in those patients who present with an acute psychotic disturbance which settles very rapidly, often before a full PSE can be carried out. Furthermore, the distribution of EE components was found not to differ between relatives of centre-diagnosed schizophrenics with and without a CATEGO classification of SOP. For these reasons, all 78 patients with a centre diagnosis of schizophrenia have been included in this analysis. 104 of the relatives with whom these patients lived were rated for EE, 67 being parents, 29 spouses and 8 other kin.

Patients in the Indian sample were drawn from two different environments; the city of Chandigarh, which is a

TABLE I

Distribution of EE components among relatives from London, Aarhus and Chandigarh

	London	*Aarhus*	*Chandigarh*
Mean number of *critical comments*	8.4[1]	4.5	1.9
Proportion of relatives showing *hostility*	18%[1]	21%	16%
Proportion of parents showing *over-involvement*	36%[1]	n.a.[3]	4%
Mean score on *warmth*	2.3[1]	2.5	2.0
Mean number of *positive remarks*	2.6[2]	3.1	0.8
Proportion of relatives classed as *high-EE*	54%[1]	54%	23%
Proportion of hostile relatives with low criticism	0%	0%	29%
Correlation between warmth and critical comments	−0.44	−0.57	0.10
Number of patients assessed	37	28	78
Number of relatives assessed	46	28	104

1. Data from Vaughn & Leff (1976a)
2. Data from Leff *et al* (1982)
3. Danish data not comparable with Indian and English (see text)

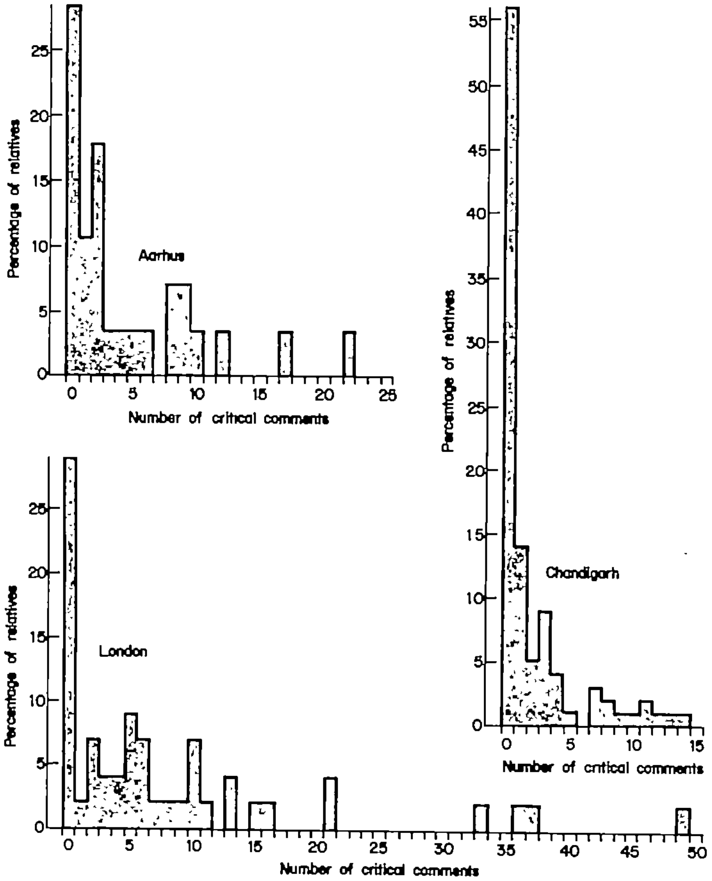

FIG. 1 Distribution of critical comments among relatives of schizophrenic patients in London (Vaughn & Leff, 1976), Aarhus and Chandigarh.

modern urban centre designed by the Swiss architect Le Corbusier, and a rural area of villages in Ambala district. The city dwellers include a high proportion of the professional classes, whereas the villagers are mostly peasant farmers. A more traditional way of life is maintained in the villages, an index of this being the proportion of patients living in extended families—20% of urban dwellers compared with 75% of villagers ($P<0.001$). (Comparisons were tested for significance by the t statistic, χ^2 or Fisher's exact test where appropriate.)

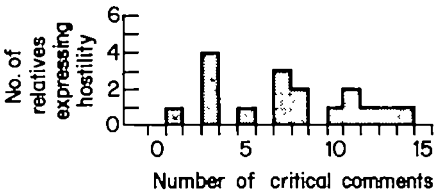

FIG. 2 The relationship between expressed hostility and critical comments among relatives of schizophrenic patients in Chandigarh.

Distributions of EE components

The data on EE components are set out in summary form in Table I.

Critical Comments

The distribution of critical comments among Aarhus and Chandigarh relatives is shown in Figure 1 with comparable data for British relatives, from the study of Vaughn and Leff (1976a). It can be seen that 56% of Chandigarh relatives made no critical comments at all, compared with 29% of Aarhus relatives and 28% of British relatives ($P<0.001$). Furthermore, while 16% of British relatives scored 15 or more, no Chandigarh relative made more than 14 critical remarks. Only two of the 28 Aarhus relatives scored more than 15 on criticism, but the sample was small and the omission of a second parent introduces a bias against more extreme responses. Nevertheless, the shape of the Aarhus distribution of critical comments closely resembles that for British relatives and the proportion rated high on this component is very similar (36% compared with 43%).

It is clear that Chandigarh relatives are far less expressive of criticism than British or Danish relatives, but there are also interesting differences *within* the Indian sample. The mean number of critical comments made by urban relatives was 2.42, compared with only 0.58 for rural relatives ($P<0.01$). These means contrast sharply with the figure of 8.4 for the British sample (Vaughn & Leff, 1976a).

The mean criticism score for the Danish patients was 4.5, intermediate between the Indian and British means. This figure may have been artificially depressed by the absence of respondents who were extreme on this measure or it may indicate a genuine cultural difference, the Danes being less inhibited in expressing criticism than the North Indians, but not as expressive as the Londoners.

Hostility

In previous British and American studies of EE, hostility was a relatively uncommon attitude which almost always occurred in conjunction with excessive criticism. In our Chandigarh sample hostility was expressed by 16% of relatives. Of the 17 hostile relatives, 16 were city-dwellers with only one from the rural area; a significant difference ($P<0.01$). The proportion of Chandigarh relatives voicing hostility (16%) compares with 21% for Danish relatives and 18% for British relatives.

All the hostile Danish relatives scored above the threshold of six critical comments for high criticism, whereas 29% of Chandigarh relatives making hostile remarks scored below this level of criticism. However, when the distribution of critical comments in hostile relatives was examined, it was found that only one relative made fewer than three critical remarks: in Figure 2 hostility can be seen to cluster at three critical comments, which is the modal value. This shows that hostility occurs at much lower levels of criticism in Chandigarh relatives than in Danish or British relatives, which could be taken to indicate that the threshold for high criticism should be set lower for Chandigarh relatives, possibly at three critical comments instead of six.

Warmth

The Chandigarh relatives were found to have similar scores for warmth to the Europeans, the average rating being 2.04 compared with 2.54 for the Danes and 2.32 for relatives from Vaughn & Leff's study. As with critical comments, however, there was an interesting difference between urban and rural samples. The mean level of warmth for urban relatives was 2.34, compared with 1.23 for rural relatives ($P<0.001$).

Relationship between warmth and critical comments

In sorting through the data it became apparent that a surprising number of Chandigarh relatives were high on both critical comments *and* warmth, a combination that was not commonly encountered among Anglo-American families. In order to check this impression, the correlation coefficient between the scores on warmth and critical comments was calculated for the English, Danish and Chandigarh samples. Three English samples were subjected to this analysis: the relatives from the naturalistic study of schizophrenia by Vaughn & Leff (1976a); the relatives from the intervention study in families of schizophrenics by Leff *et al*, 1982); and a group of parents of patients with anorexia nervosa (Szmukler, personal communication). The correlation coefficients for the English groups were −0.44, −0.45, and −0.46 respectively, and the figure for the Danish data was −0.57: these correlations are all significant ($P<0.01$) and are remarkably consistent across studies and diagnostic groups. By contrast, the equivalent correlation coefficient for the Chandigarh relatives was 0.10, which is significantly different from the figures for the English and Danish relatives (critical ratio = 2.86, $P<0.01$).

These findings indicate that warmth and criticism bear a different relationship to each other among British and

Danish relatives than among Chandigarh relatives. In the Indian sample warmth is as likely to be associated with high criticism as with low criticism, whereas in the English and Danish samples warmth is much more likely to accompany low criticism.

Over-involvement

In the Anglo-American studies, over-involvement was found much more commonly in parents than in spouses, and significantly more often in mothers than in fathers. In the recent British studies, a score of 3 or above is taken to indicate high over-involvement: this level was reached by 36% of Vaughn & Leff's parents of schizophrenics. Of the 67 parents in the Chandigarh sample, none scored more than 3 on this scale, and only three parents (4%) scored 3. This is of course a highly significant difference from the comparable figure for the English parents ($P<0.001$).

None of the 29 spouses originally interviewed scored above 2 on over-involvement, but one patient left the parental home during the course of the one-year follow-up and went to live with her husband, who scored 3 on over-involvement. Thus only 1 out of 30 spouses was rated as high on over-involvement.

As we have seen in Paper I, it is likely that the Chandigarh raters were underestimating the degree of over-involvement present in the patients' families. However, even if a score of 2 rather than 3 is taken to indicate over-involvement, only an additional seven relatives, all parents, would be included. This would bring the total of over-involved parents to 15%, which is still significantly lower ($P<0.05$) than the comparable proportion among English parents. So even when some adjustment for the possible under-rating of this component is made, Chandigarh relatives are significantly less expressive of over-involvement than English relatives.

There is a problem in making comparisons with the Danish material, since only one parent was interviewed in each household, and mothers were over-represented. Of the nine mothers assessed, five (56%) scored 3 or more on over-involvement, which compares with the British figure of 44%. It is noteworthy than no Danish mother scored high on both over-involvement *and* criticism, whereas this combination occurred in 25% of British mothers.

Positive remarks

In a recent English series (Leff *et al*, 1982) the average number of positive remarks made by relatives of schizophrenic patients was 2.55, while for our Danish relatives the mean was 3.14. In the total Chandigarh sample the comparable figure was 0.79, significantly lower ($P<0.001$). Once again, dividing the Chandigarh relatives into urban and rural dwellers produced an interesting difference, the mean for urban relatives being 0.96 and for villagers 0.39 ($P<0.02$).

The EE index

In current British research, families are assigned to the high-EE category if they score 6 or more on critical comments, *or* show any degree of hostility, *or* are rated at 3 or above on over-involvement. The proportion of relatives in our samples rated as high-EE is shown in Table I. The proportions in the London and Aarhus samples are identical (54%), while only 23% of Chandigarh relatives were classed as high-EE. Within the Chandigarh sample, 30% of urban families and only 8% of rural families qualified as high-EE.

Discussion

On virtually every characteristic of EE the Danish relatives were very similar to British relatives both in terms of mean score and in the inter-relationship of the components. Hostility occurred only in the presence of high criticism, and warmth was significantly and inversely related to criticism. The absence of high criticism among over-involved Danish mothers is a notable difference from the British group, but cannot be given much weight in view of the small size of the sample. The same reservation applies to the relatively low mean score for critical comments: the accumulation of a larger and more representative sample is essential before concluding that Danish relatives of schizophrenic patients are less critical than their English counterparts. Despite the lower mean criticism score, the proportion of Danish relatives rated as high on EE, 54%, is identical to that in Vaughn & Leff's sample. This is because of the lack of overlap between high criticism and over-involvement among the Danes, so that every over-involved Danish relative increased the number in the high-EE group. As a consequence, one would anticipate a relapse rate among the Aarhus first-admission sample comparable with that in London. Unfortunately, the small size of the initial Aarhus sample for the EE study was further reduced: some patients' initial illness failed to improve during the 12 months following the initial examiniation, and others did not return home but were placed in sheltered accommodation. The attrition of the sample made it impractical to analyse the relationship between EE and outcome for the Aarhus cases.

The relatives of the Chandigarh schizophrenic patients were found to be significantly less expressive of critical comments, over-involvement and positive remarks than Danish or English relatives. However, the degree of warmth and of hostility expressed did not differ signifcantly between the cultural groups. The differential effect of culture on the various components of EE is puzzling and needs further exploration. It must be emphasised that these ratings were made from an interview with the relative, and a basic assumption of the technique is that the relative's responses to questions about the patient reflect current behaviour between the individuals. To the extent that the relative's EE has been consistenly found to be related to the outcome of the patient's psychiatric condition, this assumption appears to be justified. More direct evidence has also been provided by recent parallel studies of relatives' EE and observations of interaction between relatives and patients (Miklowitz *et al*, 1984). This work was conducted with American subjects and is now being replicated with a British sample. Unfortunately the cultural distance between North American, European, and North Indian relatives is so wide that it cannot be assumed that the same relationship will hold in Chandigarh between responses to an interviewer and current behaviour. The follow-up of the patients' clinical state will throw some light on this issue, but observations do need to be made of direct interaction between relatives and patients in India.

Within the Chandigarh sample, rural relatives were rated significantly lower than urban relatives on critical comments, hostility, warmth and positive remarks. In fact over-involvement was the only EE component for which this effect could not be demonstrated, but this may well be a consequence of the rarity of this attitude in Chandigarh, where it was rated in only four individuals. Of the urban families, 30% were classified as high-EE, compared with only 8% of rural families ($P<0.01$).

A number of possible explanations can be put forward for this urban/rural difference in the expression of emotion. It is almost certainly bound up with differences in life-style, any of which might be responsible. For instance, the rural dwellers were much more likely to live in extended families than the city-dwellers, whose households were more often nuclear. It is conceivable that the emotions generated by family relationships become dispersed throughout the network of an extended family, instead of being concentrated in the much smaller group of a nuclear family. This hypothesis was tested by examining the distribution of high-EE families between nuclear and extended households: in fact 21% of nuclear families and 25% of extended families were classified as high-EE, a non-significant difference. Another possible explanation is based on evidence that in many non-Western cultures emotional distress tends to be more readlily expressed in somatic rather than psychological terms (Leff, 1981). However, this would be expected to occur predominantly with the negative emotions, such as criticism and hostility, but in this study the positive emotions of warmth and positive remarks were equally affected. It has also been argued that the expectations of rural relatives are lower and their tolerance higher than those of urban relatives (Waxler, 1979). Once again, though, this would explain the muted expression of negative emotions by the villagers but not of positive emotions. Finally, it has been postulated that the increased emphasis on the individual in Western culture has led to a greater value being placed on introspection and on the untrammelled expression of emotion (Leff, 1981). This influence would affect the whole range of positive and negative emotions. However, the data required to test this hypothesis were not collected in this study.

Before considering the implications of these results for the course of schizophrenia in Chandigarh, we need to discuss an unexpected and surprising finding from this study. Whereas critical comments and warmth were significantly negatively correlated among the Aarhus relatives and in several British studies, their correlation for Chandigarh relatives was positive, although very small and non-significant. This indicates that Chandigarh relatives commonly express high criticism and high warmth at the same time, whereas this combination is rare among British relatives. A European might well regard warmth and criticism as incompatible emotions, whereas North Indians appear to have no difficulty in expressing both about the same patient during the hour of the interview. It may be that this is another example of a broad difference between developing and developed countries, whereby concepts that are considered mutually exclusive in one area are able to co-exist harmoniously in another. Whatever the explanation, the combination of high warmth and high criticism expressed by some Chandigarh relatives has potentially important implications for the patients concerned, since warmth was found to be protective in the British studies (Brown *et al*, 1972; Vaughn & Leff, 1976a), whereas criticism had the opposite effect. When the follow-up data are analysed, we shall explore the possibility that high warmth may neutralise the deleterious effect of high criticism.

As discussed above, it is not possible at this stage to decide whether the lower levels of EE rated in Chandigarh relatives compared with Danish and British relatives actually reflect a lower emotional tone in interactions with patients. If they do, then this should be detectable in terms of a lower relapse rate in this sample of Chandigarh schizophrenic patients than in comparable groups of British patients. Furthermore, the striking urban-rural differences in EE exhibited by the Chandigarh relatives should also be reflected in differential relapse rates for the patients. The test of these hypotheses lies in the one-year follow-up data, which are presented in the third paper of this series.

III. Influence of Relatives' Expressed Emotion on the Course of Schizophrenia in Chandigarh

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We conducted a one-year follow-up of patients who had made a first contact with psychiatric services in Chandigarh, North India, and had been assigned a diagnosis of schizophrenia. The expressed emotion (EE) of the patients' relatives was assessed early on. We found the same associations between the individual components of EE and relapse of schizophrenia as in previous Anglo-American studies, but only the association between hostility and relapse was statistically significant. Applying the same criteria as in the Anglo-American studies for 'high EE', we found a significant relationship between high EE and relapse. This relationship was not explained by other factors often associated with higher relapse rates. We conclude that the significantly better outcome of Chandigarh first-contact patients compared with a London sample is largely due to the significantly lower proportion of high-EE relatives in the North Indian sample.

The two-year follow-up of the *International Pilot Study of Schizophrenia* (WHO, 1979) revealed that the outcome for schizophrenic patients in the centres at Agra and Ibadan was substantially better than in the centres situated in Europe and North America. This could not be attributed to divergent diagnostic practices, since there was close agreement on the diagnosis of schizophrenia between psychiatrists at the Indian and Nigerian centres and those in the developed centres. Another possible explanation was that the better outcome in developing countries might be attributable to more tolerant family attitudes to illness and handicap. An opportunity to test this hypothesis was presented by the initiation of the WHO *Determinants of Outcome* project. The design of the 'core' study of first-lifetime contact, and of the sub-study of the relationship between relatives' expressed emotion (EE) and outcome of first-contact schizophrenia, have already been described in the two preceding papers (Wig *et al*, 1986a; Wig *et al*, 1986b). In this paper, we present data on the relationship between relatives' EE and outcome in Chandigarh.

Before investigating possible links between relatives' EE and relapse of schizophrenia in India, it was essential to determine whether the techniques of assessing EE could be transferred intact across linguistic and cultural frontiers. A preliminary study was conducted using a 'naive' rater who was bilingual in English and Hindi. He was trained to rate EE in English and then without further experience was required to rate interviews conducted in Hindi by the Chandigarh interviewers. The results of this study (Wig *et al*, 1986a) indicated that two major components of the EE index, critical comments and hostility, could be transferred from English to Hindi with little or no distortion. In the case of the third major component, over-involvement, it appeared that the Hindi-speaking raters were under-estimating the scores on this scale. However, an adjustment can be made to compensate for this possible source of bias by lowering the cut-off point for 'high over-involvement'.

Method

All 207 patients entering the 'core' component of the *Determinants of Outcome* study in Chandigarh were considered for the study of relatives' EE. Patients were drawn from two different environments, the city of Chandigarh and an adjacent block of rural villages in the Ambala district. A high proportion (66%) of the city dwellers are literate compared with 20% of the villagers, most of whom are peasant farmers. A traditional Indian way of life characterises the villages: 75% of patients came from households occupied by extended families, while in the city the corresponding proportion was only 20% ($P<0.001$).

Patients included in the 'core' study had to be living with at least one key relative with whom they maintained daily face-to-face contact to be eligible for inclusion in the EE sub-study: there were 103 such patients. Relatives were asked to take part in the Camberwell Family Interview (CFI) (Brown & Rutter, 1966; Vaughn & Leff, 1976b). The relatives of 15 patients eligible for inclusion in the EE sub-study refused to be interviewed, all but one of these being city-dwellers. Interviews were finally collected for 93 patients, 78 of whom had a centre diagnosis of schizophrenia.

The interview procedure in the city closely resembled that in the developed centres, but interviews in the villages often began as public events, with women and children crowding round, attracted by the novelty. However, it was usually possible to establish some degree of privacy. About three-quarters of the interviews were conducted in patients' homes and the remainder in a clinic. No problems were encountered in making tape-recordings, and 7% of the interviews were rated independently by the two Chandigarh raters. Their inter-rater reliability remained at a satisfactory high level.

The patients were generally treated on an out-patient basis, very few being admitted: 6% of the 'core' study sample were in-patients, compared with 12% of the 70 schizophrenics included in our follow-up and EE study. No patient was an in-patient for more than one month. Almost every patient was seen monthly over the course of the following year. At each visit a condensed form of the Present State Examination (PSE) (Wing *et al*, 1974) was administered, the clinician concentrating on symptoms rated as present during the index episode. Thus it was readily apparent when psychotic symptoms disappeared or, in case of relapse, when they reappeared. These detailed and meticulous records were scrutinised by an independent researcher (JL) who was blind to the EE ratings of the relatives, and who decided whether a relapse had occurred within nine months of first contact with the service, or within one year. A comprehensive follow-up at one year was built into the design of the *Determinants of Outcome* project, but it was necessary to use a nine-month follow-up as well to ensure comparability with the previous EE studies, in which patients were assessed for relapse after a period of nine months living with key relatives. However, the previous studies considered only in-patients, and their average length of stay in hospital was 11 weeks: the mean duration of follow-up from date of admission was thus close to one year, including the time spent in hospital. In some respects, therefore, the one-year follow-up of the Chandigarh patients is more appropriate than the nine-month follow-up for comparison with the data from developed countries.

Results

Diagnosis

Two diagnostic systems were employed; the CATEGO program was used to process the PSE symptoms (Wing *et al*, 1974), and a clinical diagnosis was made by the centre psychiatrists, taking into account everything that was known about the patient. The advantage of the CATEGO system is that it is highly standardised. However, it is entirely dependent on signs and symptoms elicited by use of the PSE. Some of the patients in this sample presented with an acute transient psychosis and were very difficult to interview with the PSE during their acute disturbance. When they were seen later, very few symptoms were still present. Such cases are assessed as 'doubtfully psychotic' or 'non-psychotic' when the data are processed by CATEGO, although the clinicians who saw these patients often made a firm 'centre diagnosis' of schizophrenia. Because each diagnostic system has its own advantages and disadvantages, they are used in parallel in the analyses presented here.

A total of 93 patients were included in the EE study. Of these, 78 were given a centre diagnosis of schizophrenia (Table I, first three columns). The CATEGO classes usually

TABLE I

Distribution of patients included in the EE study at initial evaluation, divided according to CATEGO class, centre diagnosis, inclusion/exclusion at follow-up, and relatives' rating on EE for those followed up

CATEGO class	*Centre diagnosis of schizophrenia*			*Other centre diagnosis*			*Totals*
	Included in follow-up		*Excluded from follow-up*	*Included in follow-up*		*Excluded from follow-up*	
	High EE	*Low EE*		*High EE*	*Low EE*		
S+ S? P+ P? O+	11	33	6(+1*)^1	1	4	2^4	*57(+1*)*
O?	3	13	1^2	0	1	0	*18*
Other	2	8	1^3	—	—	6^5	*17*
Totals	*16*	*54*	*8(+1*)*	*1*	*5*	*8*	*92(+1*)*

*1 patient's ratings were lost

1. 5 patients had persistent florid symptoms, and 1 patient changed household
2. persistent florid symptoms
3. died
4. 1 pateint had persistent florid symptoms, 1 patient had 6-month follow-up only
5. excluded for diagnostic reasons

included under the rubric 'schizophrenia' comprise S+, S?, P+, P? and O+. These classes will be referred to here as 'CATEGO SOP'. Between them they accounted for 57 of the patients in this sample (Table I, first row): 50 of these also had a centre diagnosis of schizophrenia. Another class, O?, is considered to apply to doubtful schizophrenia and has not been included in past studies of EE in England or North America, where it is rarely encountered. However, it was applied by CATEGO to 18 of the patients in this sample, 17 of whom were diagnosed as schizophrenic by the centre psychiatrists. Most of these cases presented as acute transient psychoses. Class O? accounted for more than half the cases with a centre diagnosis of schizophrenia who were not classified as CATEGO SOP. The number of patients included in each diagnostic category and the numbers excluded from follow-up for various reasons are shown in Table I.

Relapse

In the British studies, relapse was defined as Type I, the reappearance of characteristic schizophrenic symptoms after at least one month's absence, or Type II, the exacerbation of persistent schizophrenic symptoms. The judgement involved is much more difficult and subjective for a Type II relapse than for a Type I. In the study very few patients (9%) were found to have persistent schizophrenic symptoms following the key episode, and they have been excluded from the follow-up because of the difficulty of making a judgement about relapse. In the other cases, relapse was defined as the recurrence of delusions or hallucinations or the appearance of markedly disturbed behaviour after at least one month's freedom from these symptoms.

Of the 49 cases given a CATEGO SOP class and included in the EE follow-up, seven had relapsed within nine months and a further two within the year, giving a one-year relapse rate of 18%.

Of the 70 centre-diagnosed cases included, eight had relapsed within nine months and a further two within one year, giving a one-year relapse rate of 14%. It will be appreciated that a relapse rate as low as this decreases the likelihood of demonstrating significant associations of any factor with relapse. Because there are a few more relapses at one year than at nine months, and for the reasons given above concerning the duration of in-patient stay, the relapse data presented here are those for the one-year follow-up only.

Relationship between EE components and relapse

In the first-ever study of EE (Brown *et al*, 1972) the relationship between the individual components of EE and relapse were examined before constructing an index of EE. We will proceed in the same way here, since there is no *a priori* reason why the method of constructing the composite EE index in England should apply to the Chandigarh sample. We adopted the convention used in the Anglo-American studies that where two relatives in one household are assessed for EE, the higher rating on each index is used to analyse the relationship with relapse.

Critical comments and relapse

During the follow-up period one patient, who had been living with the most highly critical relatives in the sample, married and went to live with his wife who was completely uncritical. He remained well for at least a year. Taking this patient's change of household into account, and applying the British threshold for high criticism, the data are shown in Table II. It is only at the one-year follow-up and using the centre diagnosis that there is any suggestion of a relationship between high criticism and relapse, but the difference between the two relapse rates does not even approach statistical significance.

The relationship between criticism and hostility shown by Chandigarh relatives suggested that a cut-off point of three critical comments for 'high criticism' might be more appropriate (Wig *et al*, 1986b). Consequently the data were

TABLE II

Relationship between critical comments and relapse during one-year follow-up of schizophrenic patients. Two different thresholds for 'highly critical' (3 or 6 critical comments) were considered

Diagnostic group	*Number of critical comments*	*Number of patients relapsed*	*Number of patients well*	*% relapse*	
CATEGO SOP ($n=49$)	6 or more	1	6	14%	NS
	<6	8	34	19%	
Centre diagnosis ($n=70$)	6 or more	2	7	22%	NS
	<6	8	53	13%	
CATEGO SOP ($n=49$)	3 or more	3	10	23%	NS
	<3	6	30	17%	
Centre diagnosis ($n=70$)	3 or more	4	14	22%	NS
	<3	6	46	12%	

also analysed using this lower threshold (see Table II). This produced only a marginally greater difference in relapse rates between high- and low-criticism groups at the one-year follow-up, and none of the differences approach statistical significance.

Hostility and relapse

In the British studies any degree of hostility led to the relative being assigned to the high-EE group. Using this convention, the results are as shown in Table III. A significant difference in relapse rates appears within the group of centre diagnosed schizophrenics at the one-year follow-up.

Warmth and relapse

Warmth was found to indicate a good prognosis in the British studies but was not incorporated in the EE index because when it was linked with over-involvement a bad outcome ensued. As we have seen, over-involvement is rare amongst the Chandigarh relatives. The relationship between warmth and relapse is shown in Table IV.

The relapse rate in families where a relative scores 4 or more (on a 5-point scale) is half the rate in low-warmth families, but this difference is not statistically significant.

In the preceding paper it was found that relatives exhibiting both high warmth and high criticism were relatively common in the Chandigarh sample, in sharp contrast to the Danish and British samples (Wig *et al*, 1986b). Since warmth was linked with a good outcome in the British work, this combination raised the possibility that warmth might counteract the deleterious effects of criticism in Chandigarh relatives. Unfortunately there were too few patients in high-criticism homes to explore this possibility statistically. There were nine patients in high-criticism homes, two of whom relapsed. It is worth recording that while the two patients who relapsed did have relatives who were low on warmth, five of the seven patients who remained well also had low-warmth relatives. There is no suggestion from these figures that warmth might protect patients against the effects of criticism.

Over-involvement and relapse

The number of relatives scoring at least 3 on over-involvement was too few to conduct a meaningful analysis. Of the four patients with families rated high on this component, one relapsed. The patient who relapsed had a spouse with a warmth rating of 1, whereas the other three patients came from families with warmth rated as 5, 4 and 4. This suggests that, as in the British studies, warmth and over-involvement may be closely associated.

TABLE III

Relationship between hostility and relapse during a one-year follow-up of schizophrenics

Diagnostic group	*Hostility*	*Number of patients relapsed*	*Number of patients well*	*% relapse*	*Statistical significance*
CATEGO SOP ($n=49$)	Present	3	5	38%	NS
	Absent	6	35	15%	
Centre diagnosis ($n=70$)	Present	4	7	36%	exact $P=0.04$
	Absent	6	53	10%	

TABLE IV

Relationship between warmth and relapse during a one-year follow-up of schizophrenics

Diagnostic group	*Rating on warmth scale (1–5)*	*Number of patients relapsed*	*Number of patients well*	*% relapse*	
CATEGO SOP ($n=49$)	4 or more	1	10	9%	NS
	<4	8	30	21%	
Centre diagnosis ($n=70$)	4 or more	1	14	7%	NS
	<4	9	46	16%	

The possibility that over-involvement has been consistently under-rated in this study was raised in the first paper of this series (Wig *et al*, 1986a). To compensate for this potential bias it seemed sensible to lower the cut-off point for over-involvement to 2 and then re-examine its relationship to outcome; but when this was done no alteration was produced in the relationship between over-involvement and relapse.

High EE and relapse

In the current British studies, families are assigned to a high-EE category if they score 6 or more on critical remarks, or show any degree of hostility, or are rated at 3 or above on over-involvement. These same criteria have been applied to a sample of Californian relatives, and despite a significantly different distribution of critical comments from London relatives, the resulting measures of EE have proved to be as strongly linked with outcome of schizophrenia (Vaughn *et al*, 1984). Among the Chandigarh sample, only hostility was found to be significantly linked with relapse. However, there was also a tendency for excessive critical comments and over-involvement to be associated with relapse. There was no indication from the data that the criterion for high criticism should be lowered, or that warmth should be incorporated in the index as a modifying factor. Therefore the criteria used in London to assign families to high- or low-EE categories were applied unchanged to the Chandigarh data.

The relationship between high/low EE and outcome is presented in Table V. It can be seen that the relapse rates in high-EE and low-EE groups are very similar, both for cases classified as CATEGO SOP and for centre-diagnosed schizophrenics. However, it is only in the latter group that the difference in relapse rates between high-EE and low-EE homes reaches an acceptable level of statistical significance, mostly because of the larger number of patients involved.

Our results for the centre-diagnosed schizophrenics replicate the relationship between relatives' EE and outcome of schizophrenia found in the Anglo-American studies, in which face-to-face contact between relative and patient and maintenance neuroleptics were both found to modify the outcome in high-EE homes. Unfortunately the one-year relapse rate in the Chandigarh sample is too small to investigate the possible role of these factors.

Comparability of London and Chandigarh samples

There are two major differences between the centre-diagnosed schizophrenics from Chandigarh and the patients included in the London studies: the Chandigarh patients were all first contacts with the psychiatric services; and they included cases classified by CATEGO as O?, which were excluded from the London samples. To make a valid comparison between relapse rates, it is necessary to take account of these differences.

It is well documented that first-admission patients have a better outcome than patients on subsequent admissions. Therefore it is necessary to compare the Chandigarh first-contact patients with a sample of first-admitted patients from London. The studies by Brown *et al* (1972) and Vaughn & Leff (1976a) used identical techniques of clinical assessment and EE rating, so that their data can be pooled. Of their 128 patients, 77 were first admissions and are used here for comparison with the Chandigarh sample.

The nine-month relapse rate for the London first-admission patients was 29%. The comparable rate for Chandigarh CATEGO SOP cases was not significantly different, at 18%. However, the rate for the centre-diagnosed patients was 14%, significantly lower than that for the London cases ($P<0.05$). This difference could be attributable to the inclusion of CATEGO class O? patients in the centre-diagnosed sample, to the lower proportion of high-EE homes, or to both factors.

Of the 77 London patients, 36 (47%) lived with high-EE relatives. The proportion of high-EE homes in the Chandigarh centre-diagnosed sample was 23%, significantly lower ($\chi^2=9.16$, $P<0.005$). Since living in a high-EE home was significantly associated with a poorer outcome in both samples, this difference could account for the better outcome of the Chandigarh patients.

Of the 78 patients diagnosed by the Chandiagarh psychiatrists as suffering from schizophrenia, 17 (22%) were assigned by CATEGO to Class O?. Among the 24 schizophrenics from rural areas Class O? accounted for 9 (38%), among the 54 urban schizophrenics it accounted for

TABLE V
Relationship between EE and relapse during a one-year follow-up of schizophrenics

Diagnostic group	*EE level*	*Number of patients relapsed*	*Number of patients well*	*% relapse*	*Statistical significance*
CATEGO SOP ($n=49$)	High EE	4	8	33%	exact $P=0.105$
	Low EE	5	32	14%	
Clinical diagnosis ($n=70$)	High EE	5	11	31%	exact $P=0.035$
	Low EE	5	49	9%	

8 (15%): these proportions are significantly different ($P<0.05$). No patient in Class O? relapsed, compared with 19% of the remainder, but this difference is not significant. This may, of course, be an artifact of the small sample-size, but the data do not allow us to conclude that the inclusion of Class O? patients in the Chandigarh sample explains the better outcome compared with the London sample.

Influence of other factors on relapse

In all the previous studies of EE and relapse, male sex and unmarried status have been found to be associated with a poor outcome. However, after partialling out the variance due to these factors, we found that the relationship between EE and relapse remained significant.

In the Chandigarh sample the relapse rate of married patients (7%) was considerably lower than that of single patients (21%) but not significantly so. Similarly the relapse rate of women (8%) was less than half that of men (22%), but this difference was not statistically significant. It was noteworthy, however, that all seven males who relapsed were single. Considering males alone, relapse was found to be significantly higher among single men than among those who were ever married (exact $P=0.023$).

The other factor in this sample that might be expected to be associated with relapse is urban or rural domicile, since high-EE homes found significantly more often in the city than in the country ($P<0.01$) (Wig *et al*, 1986b). The urban relapse rate was 19% compared with a rural relapse rate of 9%, a difference which does not reach statistical significance although it is in the expected direction.

Thus there are a number of factors apart from EE which are marginally or significantly associated with poor outcome; male sex, single status and residence in the city. In order to determine whether any of these factors might account for the relationship between EE and relapse, a series of log-linear analyses was performed using the SAS–FUNCAT program. The model employed supposes that the logit of the probability of a relapse is a function of EE and one other variable. The variables tested with this model were: sex of patient; marital status; urban/rural domicile; diagnostic group CATEGO O?/other classes. The results of these analyses are shown in Table VI, from which it can be seen that in each case only EE is significantly associated with relapse.

Table VI

Results of testing the contribution of various factors to relapse. Each log-linear model includes EE and one other factor

	Significance
EE	$P=0.03$
Sex	$P=0.10$
EE	$P=0.05$
Urban domicile	$P=0.78$
EE	$P=0.04$
Marital status	$P=0.09$
EE	$P=0.03$
CATEGO Class O?	$P=0.24$

Considerable attention has been given in the *Outcome* study to Schneiderian first-rank symptoms and their discriminatory and predictive possibilities. Patients who manifest one or more of eight such specified symptoms rated on the PSE are assigned a CATEGO class of S+. Comparing the 27 patients in the EE study who had CATEGO class S+ with the other 53 patients, we found no significant difference between the levels of EE in the two groups: 33% of S+ patients and 16% of non-S+ patients had high-EE households. On the other hand, the relapse rate of the S+ patients (30%) was significantly higher than the 5% rate of the non-S+ patients ($P<0.006$). We also found that the association of EE with relapse was no longer statistically significant if the association with CATEGO class S+ was taken into consideration: log-linear analysis gave $P=0.02$ for CATEGO class S+ and $P=0.11$ for EE.

Relationship between life events, EE and schizophrenic episodes

In the studies carried out in London it has proved possible to study the relationship between life events and EE in the precipitation of relapse (Leff *et al*, 1983). Life events were assessed by the technique of Brown & Birley (1968). It was found that among patients who suffered an episode of schizophrenia when not taking maintenance neuroleptics, *either* independent life events *or* high EE appeared to be responsible. Independent life events exerted their effect during the three weeks or so before the onset of schizophrenic symptoms. In a study conducted by Leff & Vaughn (1980), 9 out of 16 (56%) patients who were off medication and living with low-EE relatives experienced an independent life event in the three weeks before relapse. The corresponding proportion for those in high-EE homes was one out of 15 (7%), a highly significant difference (Exact $P=0.004$).

It is possible to conduct a similar analysis on the Chandigarh sample of first-contact patients in order to study the relationship between life events, expressed emotion and onset of schizophrenic disorder. One of the sub-studies being carried out as part of the *Determinants of Outcome* project concerns the prevalence of life events prior to the sudden onset of an episode of illness (Day *et al*, in press). The technique of measuring life events is basically that of Brown & Birley (1968) with certain modifications to aid its cross-cultural application. Hence the data on life events from the Chandigarh centre are comparable with those from the London studies. Unfortunately not all the patients in the EE sub-study were eligible for inclusion in the life-events sub-study, but the overlap is sufficient to allow a statistical analysis.

In all, 44 patients in the EE sub-study had a life-events interview completed. Seven of these were on neuroleptic drugs when first seen: this group is too small for analysis. It is worth noting, however, that none of these patients lived in high-EE homes and that four experienced life events in the 3 weeks preceding onset. Of the remaining 37, seven were living in high-EE homes and 30 in low-EE homes. The life-events data for these patients are shown in Table VII, with the London figures for comparison. It can be seen that the

TABLE VII
Relationship between EE and life events in the three weeks before onset for patients off medication

	Patients with life event	*Patients without life event*	*Proportion with event*	*Statistical significance*
Chandigarh				
High-EE home	1	6	14.3%	$P=0.05$
Low-EE home	17	13	56.7%	
London				
High-EE home	1	14	6.7%	$P=0.004$
Low-EE home	9	7	56.3%	

proportions in each group in the Chandigarh sample are similar to the proportions in the London sample, and that the effect of events in Chandigarh just reaches significance.

Discussion

This study has supplemented the findings of the *International Pilot Study of Schizophrenia* (WHO, 1979) by showing that a cohort of first-contact patients in Chandigarh had a significantly better outcome after one year than a comparable sample in London. This result is most unlikely to be due to selection factors since strenuous efforts were made to include all instances of a first contact with the psychiatric services for schizophrenic illness within the Chandigarh centre's catchment area.

The significant association between high-EE attitudes and relapse of schizophrenia established in studies in London and California has been replicated in Chandigarh. It is remarkable that the same cut-off points on the EE scales that best predicted relapse and non-relapse in the Anglo-American studies also applied to the Chandigarh sample. This predictive validity across disparate cultures is particularly surprising in view of the markedly different distributions of EE components in the different cultural groups. It is of considerable interest, however, that hostility contributed most of the predictive validity of the EE scale in the Chandigarh sample, and that it was the only predictive component that occurred with similar frequency among North American, British, Danish and North Indian relatives. This suggests that the expression of anger in the form of hostility is relatively unmodified by cultural factors and has a similar effect on schizophrenic patients regardless of their cultural background.

Two other factors were found to be significantly associated with relapse in these North Indian patients. The first was the presence of Schneiderian first-rank symptoms, corresponding to CATEGO class S+. This was not expected, since previous studies have not found Schneideran symptoms to be of value in predicting outcome (e.g. Radhakrishnan *et al*, 1983). Furthermore, in the study of Vaughn & Leff (1976a) the relapse rate of S+ cases (27%) was not significantly different from that of other CATEGO schizophrenics (36%). Elucidation of this link between CATEGO class S+ and outcome must await further analysis; it may be an artifact of the small numbers of patients involved. The only other factor we found to have a significant association with relapse in the Chandigarh sample was single status among male patients.

Log-linear analyses demonstrated that, except for the presence of Schneiderian symptoms, none of the intervening variables studied could account for the significant association between EE and relapse. This replicates the findings of the earlier Anglo-American studies. It was somewhat surprising that no significant difference emerged between urban and rural relapse rates, since there were striking differences in the distribution of EE components between city dwellers and villagers. It is possible that a difference might be revealed with the accumulation of a larger number of relapses over the period of the two-year follow-up, which remains to be analysed.

The significant association between EE and relapse found in this Chandigarh first-contact sample, and the significantly lower proportion of high-EE households than in a comparable London sample, indicate that relatives' EE could definitely be a major factor in explaining the better outcome of the North Indian patients. This raises interesting questions about cultural influences on the emotional attitudes that are represented by the EE index.

The starting point for further studies could well be the major difference in distribution of EE components between urban and rural relatives. The possible insights afforded by this line of enquiry could contribute to therapeutic endeavours to alter the emotional environment in high-EE homes (Leff *et al*, 1982).

The overlapping data from this sub-study and the life-events sub-study have indicated a similar relationship between life events and relatives' EE to that found in the study of Vaughn & Leff (1976a): independent life events appear to play an aetiological role in the onset of schizophrenia for a substantial proportion of unmedicated patients living with low-EE relatives in Chandigarh.

The data presented in this series of three papers illustrate the feasibility and importance of conducting psychosocial research in a variety of cultural settings. Given the necessary coordinating and training facilities, work of this nature can elucidate which social factors in the pathogenesis of psychiatric conditions transcend culture and which are moulded by the cultural milieu.

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Expressed Emotion in Individual and Family Settings: A Comparative Study

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Expressed emotion ratings were performed, in two separate settings, on the parents of 20 children suffering from anorexia nervosa. Ratings were made on conventional individual interviews and on videotapes of a family interview. Comparison of ratings in the two settings showed a very high correlation for 'critical comments'. Findings on the other scales were harder to interpret, but significant correlations as well as differences were noted. Expressed emotion rated in family interviews provides new possibilities for the study of families.

The measure 'expressed emotion' (EE) has proved reliable and valid in predicting relapse in both schizophrenia and depression (Brown *et al*, 1972; Vaughn & Leff, 1976*a*; Vaughn *et al*, 1982) and also for early termination of psychotherapy, particularly family therapy, in anorexia nervosa (Szmukler *et al*, 1985). It is a measure of relatives' attitudes to a patient, and is estimated on the basis of a standardised interview with each relative. Of the five scales rated, 'critical comments' (CC) and 'emotional over-involvement' (EOI) are the most powerful predictors. Family interventions which have resulted in a reduction of relatives' EE have also reduced the relapse rate in patients with schizophrenia (Leff *et al*, 1982).

It is assumed that EE, thus assessed, has some relationship to interactions in the family, but this has been little studied. Valone *et al* (1983) found a significant relationship between parental criticism on individual interview (rated in the conventional EE manner, but not on the standard Camberwell Family Interview) and critical statements made during direct interactions between parents and their disturbed, non-psychotic adolescent offspring. Critical statements, 'benign' or 'harsh' in quality, were rated from transcripts of videotaped dyadic interviews (parent and offspring) and triadic interviews (both parents and offspring), according to a previously devised method for assessing 'affective style' (Doane *et al*, 1981). High-EE parents made significantly more critical statements in direct interaction with their child than low-EE parents. No significant difference was found between high-EE and low-EE parents in the number of 'neutral instrusive statements' (a possible analogue of emotional overinvolvement) in direct interactions. In fact, only one of the 52 parents in the study was classified as high-EE on the basis of EOI alone.

Valone *et al* (1983) also examined the combined effect of both parents' EE on 'affective style' in direct interaction. 'Dual' high-EE parents made, in total, more critical statements than 'mixed' parents (one with high EE, the other with low), who in turn made more than 'dual' low-EE parents. It was noted, however, that in the 'mixed' parental groups the low-EE parent, when present, seemed to exert an ameliorating effect on the number of 'harsh' criticisms made, but that the number of 'benign' criticisms was the same as for dual high-EE families.

Miklowitz *et al* (1984) compared EE ratings of key relatives of patients with schizophrenia estimated from the Camberwell Family Interview (CFI) (Brown & Rutter, 1966; Vaughn & Leff, 1976*b*) with measures of 'affective style'. These were made from transcripts of audiotaped family discussions involving the patient and his parents. A significant association was found between parental high EE and a negative affective style', derived by summing critical and negative intrusive statements. There was also a relationship between the type of high EE of the parent and the type of negative 'affective style'; those with high CC made more 'critical statements', while those with high EOI made more 'negative intrusive statements'.

These studies thus support a correspondence between EE measured in an individual interview and analogous parental behaviour in direct interaction with the disturbed offspring.

The aim of the research presented here was to assess whether parental EE, estimated conventionally in an individual interview, would show a relationship to EE derived using identical scales but estimated in a family interview with all members of the family present. The families in this study all contained a patient with anorexia nervosa.

Method

Subjects

The subjects were 20 intact families, each with a young member suffering from anorexia nervosa. The patients, three of whom were males, were all admitted to the Maudsley Hospital. The mean age of the patients was 19.4 years (s.d. 3.7), the mean age of onset of their illness 16.0 years (s.d. 3.7), and the mean duration of the illness 42 months (s.d. 29). At the time of admission the patients weighed a mean of 65% (s.d. 10%) of the standard weight for their age and height. Three patients had developed bulimia nervosa as a sequel to anorexia nervosa. Eleven of the families were of social class I or II, and a further seven from social class III (Registrar-General's classification, based on father's occupation). Nine parents had a history of psychiatric disorder – usually neurotic depression.

Individual ratings of EE

During the patient's stay in hospital both parents were interviewed separately, using the abbreviated version of the CFI. All five scales of EE were rated from audiotapes of these interviews. These scales comprised 'critical comments' about the patient (a simple count), 'hostility' (0–3), 'emotional overinvolvement' based on expressions by the parent of overprotectiveness, overconcern and exaggerated emotional responses (0–5), 'warmth' (0–5), and 'positive comments' (a simple count). The ratings are based not only on content but also on the vocal aspects of speech, such as tone and pitch.

Family ratings of EE

All of the families participated in a 'picnic lunch' at the end of the hospital stay or shortly afterwards, which was videotaped. All members of the family were invited, and in nine cases one or more siblings were present in addition to the parents and patient. These sessions were not designed for the purposes of this study, but for another research project: examining patterns of family interaction through direct observation. Nor were they intended as therapy sessions, although they were conducted by a therapist.

A fairly loose structure was imposed. In the first half of the session the therapist was asked to discuss in a neutral way the family's normal eating behaviour (e.g. who prepares the food, when do they normally eat, who is present, food preferences). The patient's eating disorder was also discussed, with the therapist adopting a simple fact-finding approach. In the second half of the session pressure was exerted by the therapist on the parents to encourage the patient to eat. The intention was to examine the way in which the family responded to this stress-inducing intervention. The sessions lasted one hour. Sessions did not always run according to plan, particularly as different therapists exerted differing amounts of pressure in the second phase, but in every case, the family found the situation a strain.

TABLE I

Comparison of family and individual EE ratings

	Individual EE score		*EE score in family setting*		*Correlation of ratings in the two settings*[1]	*Significance*[2] *of difference* of mean scores ($P<$)
	Mean	*s.d.*	*Mean*	*s.d.*		
All parents ($n=40$)						
CC	3.75	4.57	3.38	3.71	0.80***	NS
EOI	0.60	1.15	1.12	1.34	0.28*	0.02
Warmth	2.60	1.45	1.10	1.32	0.47**	0.001
PC	1.15	1.08	0.40	0.70	0.05	0.001
Mothers ($n=20$)						
CC	5.45	5.58	4.85	4.12	0.82***	NS
EOI	1.05	1.43	1.50	1.43	0.25	NS
Warmth	2.65	1.53	1.15	1.23	0.69**	0.001
PC	1.10	1.07	0.40	0.75	−0.10	0.001
Fathers ($n=20$)						
CC	2.05	2.38	1.90	2.57	0.59**	NS
EOI	0.15	0.49	0.75	1.16	0.20	0.01
Warmth	2.55	1.39	1.05	1.43	0.28	0.001
PC	1.20	1.10	0.40	0.68	0.21	0.001

1. Spearman rank correlation: *$P<0.05$; **$P<0.01$; ***$P<0.001$

2. Wilcoxon signed rank test

The videotapes of the sessions formed the material for our EE ratings in the family setting. The decision to use them for this purpose was not taken until after they had been made, and the therapists were not aware of the parents' individual EE ratings. The EE ratings were made by RB, who was blind to the individual ratings. Ratings were made for each parent on all five scales, but only concerning attitudes expressed about or to the patient.

Results

Table I summarises the main findings for four of the scales rated. The fifth scale, 'hostility', was excluded from the analysis because so few parents scored on this, in either situation, that a comparison was not meaningful.

Critical comments

A strong correlation was found between ratings of CC in the two settings for both mothers and fathers. The degree of association of the two sets of ratings is best seen in a scattergram (Fig. 1). The correlation of +0.8 is as high as the correlations found between trained raters, rating the same audiotape in the conventional manner. The overall level of criticism in the two situations did not differ, but mothers were significantly more critical than fathers in both situations. Using the commonly adopted cut-off point of 6+ for a classification as 'high' EE results in an identical classification of 18 of the 20 mothers in the two settings (Fig. 1). With the same cut-off point, only three fathers are classified differently in the two settings, but this is somewhat misleading, as few fathers scored 6 or more in either situation. The application of a lower cut-off point for fathers in this population is discussed elsewhere (Szmukler *et al*, 1985).

Emotional over-involvement

Overall there was a significant though modest correlation between the ratings of EOI in the two settings. Fathers obtained significantly higher ratings in the family interview. A similar trend was found for mothers, but the difference was not as clear, and did not reach statistical significance. Emotional overinvolvement also showed different associations with other measures when the two settings were compared. A significant correlation ($r=0.48$) was found between EOI and CC in the family setting, but not in the individual setting. Fathers' and mothers' EOI ratings showed a strong correlation with each other in the family setting ($r=0.60$), but not in the individual settings ($r=0.12$).

Warmth

A high correlation was found for mothers' ratings of warmth; fathers' ratings correlated only modestly, and did not reach statistical significance. Both fathers and mothers, however, were rated as being significantly warmer in the individual interviews. In both settings, mothers' and fathers' ratings were strongly correlated with each other (family interview $r=0.59$; individual interview $r=0.51$).

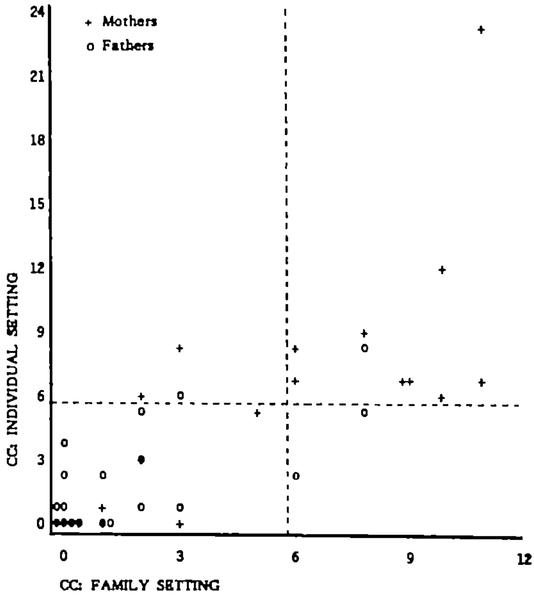

FIG. 1 Critical comments by mothers and fathers in the two settings.

Positive comments

The ratings for this scale did not correlate significantly between the two settings, but again both fathers and mothers made significantly fewer positive comments (PC) in the family session than in the individual interview. Ratings of mothers' and fathers' positive comments were strongly correlated with each other in the individual setting ($r=0.56$), but not in the family setting ($r=0.13$).

Discussion

In evaluating the results of this study, a number of general points should be borne in mind. The family interviews on which EE ratings were made were not structured to the same degree as the CFI. The conductors of these sessions were following a general plan, but it was followed to varying degrees. In contrast to the individual interviews, the parents were put under considerable pressure during the latter part of the family sessions. Although these sessions lasted about the same time as the individual interviews the parents spoke less, since they shared the floor with each other, with the patient, and often with the patient's siblings as well.

The method of rating EE in the family sessions, although based on the usual principles and definitions, required new circumstances to be taken into account. Direct comments to, as well as about the patient were rated (although the former were uncommon) and, as the ratings were made from videotapes rather than audiotapes, visual cues were also available. However, the rater felt that such cues did not substantially influence her ratings, and the content of speech and tone of voice still provided the principal basis for the ratings. Although high reliability is achieved between trained raters of EE on individual interviews on audiotapes, it is not known whether comparable results can be achieved on family interviews on videotape.

Our sample was restricted to families with an anoretic youngster, and as it is possible that such families have certain characteristics in common (Minuchin *et al*, 1978) which may not appear to the same extent in other families, our results cannot automatically be generalised to other groups.

In the light of these points, the correlation for CC between the two settings is remarkably high. Perhaps more remarkable still is that the absolute number of CC was as high in the family setting as in the individual one. These findings strongly support the reliability of this rating, and demonstrate that CC noted in individual interviews also occur in the course of family interactions without any attenuation of frequency.

In contrast to the clarity of the results concerning CC, those for EOI are more difficult to interpret. Correlations were modest, and there were significant differences in the levels rated in the two conditions, these being higher (especially for fathers) in the family setting. Two major lines of explanation can be invoked for this. The first is that the inter-rater reliability was low, possibly because of the novel application of EE ratings in a family setting. If this is not the case, then the results suggest that EOI changes across the two situations studied here. The fact that EOI was positively correlated with CC in the family setting but not the individual setting might suggest that different components were being rated, but it is also possible that in the family situation EOI had a relationship with CC which was somehow a product of that situation. For these issues to be resolved, further studies are required; the first point to be examined must be the reliability of EOI ratings in a family setting.

Mothers' warmth was significantly correlated for the two settings, but it was also significantly less in the family setting. Fathers' warmth was not correlated, but again was significantly less in the family setting. Time might have been a factor here, since there might have been fewer opportunities for individuals to express positive feelings in the family interview. We do not believe, however, that this is the main reason for the reduction of warmth. Discussions between the authors, after the experiment, of the ratings for individual parents across the two settings suggested that they were reliable, and that the family setting itself seemed to inhibit warmth. This is also supported by the finding that 'positive comments', another component of positive feelings about the patient, were also significantly fewer in the family interviews. If warmth is less evident in the family setting, it may be asked whether this represents a general characteristic of all families or one specific to families with an anorectic youngster, reflecting a particular family dynamic. This question merits further study.

The results of our study lend support to those of Valone *et al* (1983) and of Miklowitz *et al* (1984), showing a significant correspondence between criticism in the individual EE interview and in family sessions. Whereas the former studies relied on transcripts of family sessions, thus excluding the contribution of 'tone', ours has included this, and has shown that very high correlations can be obtained. The robustness of the rating of criticism is extraordinarily good, judged by the standards of psychological measures. It stands up well even when estimated in quite dissimilar settings, and whether measured directly or on the basis of transcripts of interviews. If its predictive abilities in other disorders are as good as for relapse in schizophrenia or dropping out of therapy in anorexia nervosa, then we have a valuable measure – even more so if it is amenable to change through directed therapeutic interventions.

The value of the other scales of EE is less well defined, but when applied in a family setting may prove fruitful in the elaboration of 'hypotheses' about how particular families or groups of families function. Expressed emotion may thus provide a much needed reliable basis for describing patterns of family interaction.

At present EE is seldom used in clinical practice, in spite of its obvious potential. This is due in part at least to the time-consuming nature of arranging separate individual interviews and to the less apparent relevance of such interviews for those engaged in family work. Ratings based on observation of family sessions, videotaped or live, may overcome some of these obstacles.

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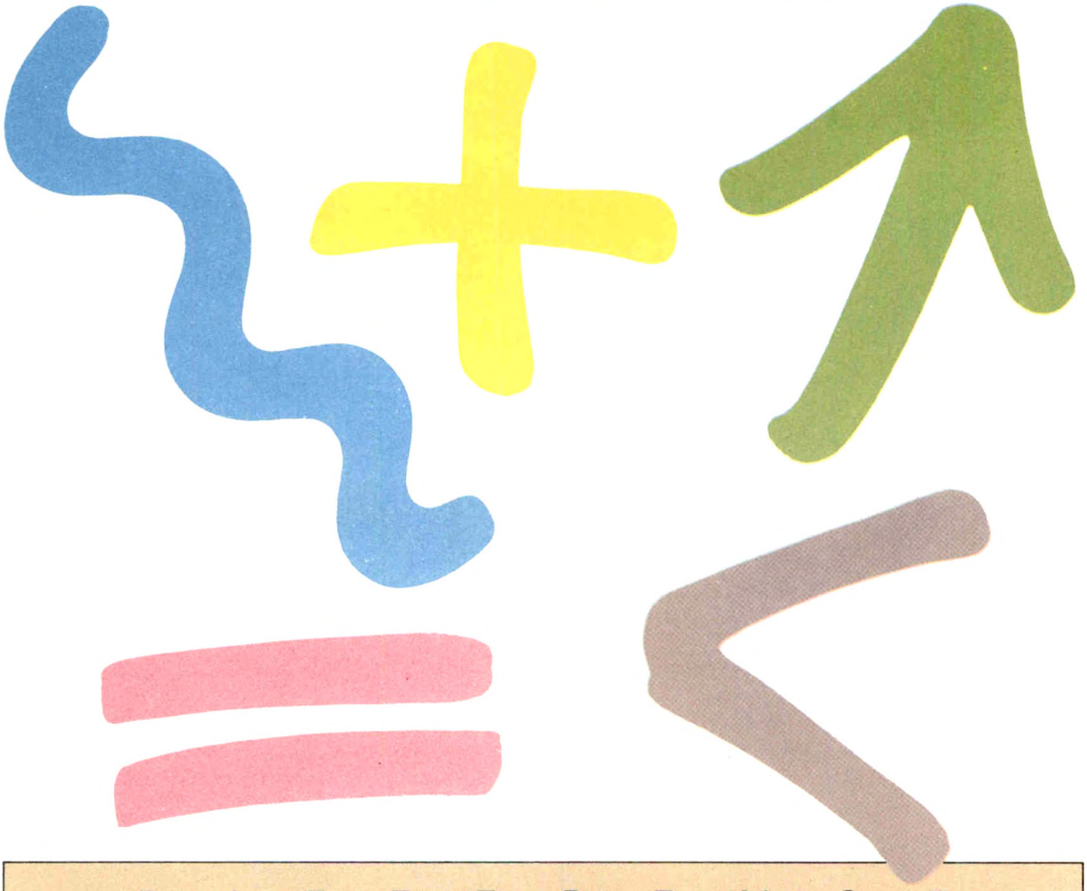

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The Impact of Recent Undesirable Life Events on Psychiatric Disorders in Childhood and Adolescence

I. M. GOODYER, I. KOLVIN and SONIA GATZANIS

The timing and number of recent stressful life events occurring in the year before onset of emotional or behavioural disorder was examined in a consecutive sample of children. Overall, events increase the relative risk of psychiatric disorder by 3–6 times. Events occur throughout the 12 months, but tend to cluster in the 16 weeks nearest onset of symptoms. The number of events influences the onset of disorder: cases with multiple events are more likely to have an event within 16 weeks of onset; cases with single events are more likely to have the event 36–52 weeks before onset. Cases whose onset occurs within 4 weeks of an event may have experienced single or multiple events. The results support the concept of additivity of recent stressful events in some cases of emotional and behavioural disorders in childhood.

Recent research indicates that recent undesirable life events which are independent of illness onset and carry a severe degree of negative impact (undesirability) may have a causal association with the onset of psychiatric disorder in children and adolescents (Goodyer *et al*, 1985*a*). Studies on adults, using general population controls, consistently indicate important effects, with the risk of psychiatric illness being increased by 2–7 times in the six months after an event (Paykel, 1978; Cooke & Hole, 1983). In addition Brown & Harris (1978), in their major study on depression in women, showed that severe events lead quickly to depression – most often within 9 weeks, and for the great majority within 6 months. However, these authors suggested that severe events may have even longer-term effects.

Brown & Harris (1978) also examined the effects of experiencing single or multiple undesirable life events in the year prior to onset, and concluded that multiple unrelated events had an additive effect, thereby increasing the risk of becoming depressed. However, they pointed out that only one in five of depressed women experienced two or more such unrelated events.

Since our data (Goodyer *et al*, 1985*a*) was derived from a case-control study and can be expressed in 2 × 2 contingency tables, we felt that: (a) use of relative risk (odds) was an appropriate and convenient way of estimating the effect that events exert on psychiatric illness in childhood and adolescence (see Appendix 1); (b) the dating of when life events occurred prior to onset would allow us to examine whether events have predominantly short or long-term effects over the 12-month period; and (c) comparing children who had experienced single and multiple events would allow us to examine the concept of additivity.

Method

Full details of the method are available elsewhere (Goodyer *et al*, 1985*a*). The study used a case-control design, and compared a consecutive sample of children attending a child psychiatry clinic with a group of community subjects drawn from the same area.

Subjects

The psychiatric sample consisted of 157 school-aged children and adolescents. The cases were classified into four mutually exclusive clinical groups according to the predominant presenting signs and symptoms obtained from the clinicians' routine assessments:

(a) conduct disorders: presenting with predominant symptoms of antisocial behaviour such as stealing, cruelty, destructiveness, or bullying ($n = 44$)
(b) mild emotional disorders: presenting with predominant symptoms of mild anxiety. This group includes transient states of anxiety, fearfulness and misery, adolescent rebellion, and parasuicide in the absence of other psychiatric disturbance ($n = 55$)
(c) severe emotional disorders: presenting with predominant symptoms of depression and serious neurotic symptoms. This group includes depression, phobic and obsessional states, school refusal, and suicide attempts ($n = 32$)
(d) somatic disorders: presenting with predominant physical symptoms. This group includes vague aches and pains, monosymptomatic hysteria, complex medical problems, (e.g. diabetes or asthma), and

secondary encopresis and enuresis occurring in the absence of any other predominant clinical features ($n = 26$).

The name, sex, date of birth, and school was recorded for each case and a table of random numbers used to select 50% of this sample. These subjects were attending 36 schools from three educational areas (City of Newcastle, North Tyneside, and Northumberland).

Following agreement with each of the Directors of Education a pool of controls was obtained by asking head teachers to provide the names and addresses of three children of the same age and sex and attending the same school for each child referred to the unit. A letter was sent to the first name listed in each group requesting an interview. Of the 78 approached, 72 (92%) agreed to the interview. Four 'reserves' were obtained for the 6 refusals by writing to the second or third names listed; the control sample thus consisted of 76 children. For the analyses presented in this paper the community controls were used as a single group, so that the base rate of events in the controls remained constant in any comparisons with the clinical groups.

The control group was compared with the psychiatric group as a whole and with the four clinical subgroups for age (using Student's *t*-test), sex, and social class (using chi-squared). There were no significant differences for any of these analyses.

Collection of life events

A schedule for recent life events was developed, and administered as a semi-structured interview to mothers or mother surrogates of subjects. For psychiatric cases the onset of illness was carefully established. Only events that occurred in the 12 months before illness in cases, or before the date of interview for controls, were included. Events received two ratings according to criteria described by Paykel (1983): independence and objective negative impact, both rated on a five-point scale. Only events considered independent or probably independent of illness and carrying a moderate to severe impact were used in the analysis.

Results were expressed as the proportion of cases experiencing events over monthly periods prior to onset of disorder. We have previously reported that the numbers of specific types of events are rather low (Goodyer *et al*, 1985*a*); the results in this paper therefore include all events carrying a moderate to severe degree of negative impact. Where appropriate the chi-squared test has been used to look for differences between cases and controls and between clinical groups. Relative risk has been estimated where appropriate, according to the formula described by McMahon and Pugh (1970) and used by Paykel (1978) (see Appendix 1).

Results

Characteristics of the clinic sample

Of the 157 patients, 68 (43%) were girls and 89 (57%) were boys. The age range was 4.7–16.25 years (mean = 11.4, s.d. = 2.8 years). The influence of age and sex on the association between recent life events and psychiatric disorder are considered elsewhere (Goodyer *et al*, 1986).

Recall of events

One method of testing the reliability of reporting events is to examine the fall-off in the mean number of events per month

TABLE I

The monthly distribution of cases by (a) event nearest to onset of disorder, (b) cases with only one event, and (c) cases with more than one event

	Number of months from onset											
	12	*11*	*10*	*9*	*8*	*7*	*6*	*5*	*4*	*3*	*2*	*1*
Event nearest onset ($n = 96$):%	9.3	7.3	5.2	7.3	8.3	4.2	4.2	5.2	11.5	5.2	10.4	21.9
		29.1				21.9					49	
Cases with only one event ($n = 54$):%	14.8	9.3	7.4	13.0	9.3	5.5	5.5	3.7	5.5	1.85	7.4	16.7**
		44.5***				24†					31.5*	
[1]Cases with 2 or more events ($n = 42$):%	2.4	4.8	2.4	0	7.1	2.4	2.4	7.2	19	9.5	14.3	28.5**
		9.6***				19.1†					71.3*	

1. Arranged by event nearest to onset of disorder.
*$\chi^2 = 15.08$, d.f. = 1, $P < 0.001$
**$\chi^2 = 2.109$, d.f. = 1, NS
***$\chi^2 = 13.944$, d.f. = 1, $P < 0.001$
†$\chi^2 = 0.349$, d.f. = 1, NS

recalled by subjects as times further in the past are considered. In the general population such events should be randomly distributed in time. If there is a large fall-off (greater than 5% per month) there may have been a serious under-reporting of events. Taking all controls ($n = 76$) as a single group, the fall-off per month in our sample was less than 1% – an acceptably low rate and in line with adult studies using interview techniques (Paykel, 1983). We conclude that it is unlikely there has been serious under-reporting of moderate to severe events.

Presence of events

Of the 157 patients, 96 (61%) experienced one or more moderate to severe events in the 12 months before the onset of illness; 54 (34%) had only one event, compared with 13 (17%) of the controls ($\chi^2 = 7.472$, d.f. = 1, $P < 0.01$), and 42 (27%) had two or more events, compared with 4 (5%) controls ($\chi^2 = 14.923$, d.f. = 1, $P < 0.001$).

If severe events make an important contribution to disorder there should be a rise in risk. Events were experienced by 27, 35, 17, and 17 patients in the groups with conduct, mild emotional, severe emotional, and somatic disorders respectively. This gives relative risk factors for these groups of 5.5, 6.1, 3.7, and 6.5 respectively.

Timing and number of events

The speed with which stressful events exert their effects on the onset of disorder cannot be established from a 2 × 2 contingency table, as all such events over the 12 months are included. In order to examine whether or not events exert long or short-term effects, the distribution of the events nearest onset for each case was established. However, if a patient had more than one event there was no way of deciding whether symptoms were brought on by a single-event. The distribution of cases experiencing only one event was therefore examined. Finally, if a greater proportion of cases experiencing multiple events have their final event nearer onset than cases with only one event, it may be that there is some additive effect of events. Table I shows the three distributions.

Onset of symptoms within 16 weeks of an event was experienced by 49% of all cases. The proportion of single-event cases experiencing an onset within 16 weeks was 31%, compared with 71% of multiple-event cases ($\chi^2 = 15.08$, d.f. = 1, $P < 0.001$); there is thus a significant association between multiple events and onset of disorder within 16 weeks of the final event.

An event occurring within 4 weeks of onset of disorder was denoted as having a 'rapid' effect. This was experienced by 21.9% of all patients. 16.7% of single-event cases experienced a rapid effect, compared with 28% of multiple-event cases ($\chi^2 = 2.109$, d.f. = 1, NS). Patients with an onset within 4 weeks of their last event, therefore, may equally have experienced single or multiple events in the previous 12 months.

A 'long-term' effect was defined as onset of symptoms occurring 36–52 weeks after the last event. This was experienced by 29.1% of all cases, accounted for by 44.5% of single-event cases and 9.6% of multiple-event cases ($\chi^2 = 13.944$, d.f. = 1, $P < 0.001$). Patients experiencing a long-term effect from events are therefore more likely to have experienced one event only.

Examination of events occurring 17–35 weeks before onset shows that 22.9% of cases experience onset of symptoms after this period; these are accounted for by 24% of single-event cases and 19.1% of multiple-event cases ($\chi^2 = 0.349$, d.f. = 1, NS). Thus there is no significant association with this period and number of events.

The timing of events for all community subjects was also examined. Numbers are small: only 22% of controls experienced one or more moderate to severe events. Of these 23.5% were multiple events and 76.5% single events. Thus, division into time periods by single and multiple events is impractical. If all controls are taken together, 8% had events within 16 weeks, 6% between 17 and 35 weeks, and 8% between 36 and 52 weeks. Comparison with all cases for each time period resulted in significant differences. Thus an excess of cases experiencing one or more events occurs throughout the 12 months.

Events therefore exert long-term (over 36–52 weeks), short-term (over 16 weeks), and rapid (over 4 weeks) effects on the onset of psychiatric disorder. Short-term effects are more likely in multiple-event cases, whereas long-term effects are more likely in single-event cases. Events exerting rapid effects can occur in either single or multiple-event cases.

TABLE II

The proportion of subjects experiencing one or more moderate to severe events at 4-monthly periods preceding onset of illness for cases or day of interview for controls, expressed as a percentage of cases with at least one event

		No. of weeks before onset					
		52–35		*35–17*		*16–1*	
	n	*n*	*%*	*n*	*%*	*n*	*%*
Conduct	44	5	11	12	27	10	23
Mild mood	55	10	18	10	18	15	27
Severe mood	32	7	22	4	13	6	19
Somatic	26	7	27	5	19	5	19
Controls	76	6	8	5	6	6	8

Specificity of events and diagnosis

The results so far consider the impact of events on all psychiatric cases. However, we have previously reported the association between events and four classes of psychiatric disorder: conduct, mild mood, severe mood, and somatic (Goodyer *et al*, 1985). We therefore divided the psychiatric sample into these four groups, to examine two questions:

(a) is the proximity of events nearest onset different between clinical groups?

TABLE III

The proportion of subjects experiencing 'single' or 'multiple' moderate to severe events in the 12 months prior to onset of illness for cases or day of interview for controls

		Single event		*Multiple event*	
	n	*n*	%	*n*	%
Conduct	44	11	25	16	36
Mild mood	55	20	36	15	28
Severe mood	32	12	37	5	16
Somatic	26	12	46	5	19
Controls	76	13	17	4	5

(b) is there any specificity between multiple events and any clinical group?

Table II examines short or long-term effects for undesirable events for each clinical group. The numbers of cases are too small to present the results as in Table I, and refer to all cases in each group experiencing one or more events. The results show that events exert short and long-term effects for all four diagnostic groups. Only mild mood disorders show any increase in cases experiencing events in the 16 weeks closest to onset, where 27% have an event nearest onset compared with 18% in the previous time periods.

Furthermore, 22% of severe mood and 27% of somatic cases occur with events occurring 35–52 weeks from onset. Although this is an increase in this time period for both disorders the differences are not significant.

The relative risk was calculated for each time period (comparing cases against controls) and the odds were the same magnitude for each time period within each clinical group as compared with events over all the 12 months.

Table III shows subjects divided into single and multiple event groups. The proportion of cases experiencing single events are highest for somatic disorders (46%) and lowest for conduct disorder (25%), with mild and severe mood showing similar rates (36% and 37%). The proportion of cases experiencing multiple events are highest for conduct disorders (36%) and lowest for severe mood (16%) and somatic disorders (19%) with mild mood in between (28%). All four clinical groups show significantly higher rates of both single and multiple events compared with the community controls. However, there are no significant differences in rates between the clinical groups. Thus there appears to be no specificity between multiple events and type of disorder.

Discussion

The results suggest that life events carrying a moderate to severe degree of negative impact (undesirability) exert effects of some importance. Thus the risk (odds) of developing a psychiatric disorder presenting with conduct/emotional symptoms is increased by a factor of 3–6 in the 12 months after exposure to an event. The lowest risk appears to be for severe mood disorders; however, the proportion of cases with severe mood disorder is only 10% less than for other cases. It may be that events exert a smaller degree of risk for severe mood disorders compared with other clinical groups, but this statement requires confirmation.

The distribution of events in the 12 months prior to onset is of interest for two reasons. Firstly, events occur throughout the 12 months, indicating that moderate to severely undesirable events exert long-term, short-term, and rapid effects on the onset of psychiatric disorder. Secondly, there are different associations between events and disorder according to the timing and the number of events. Thus nearly three-quarters of multiple-event cases experienced an event in the short-term (i.e. 1 to 16 weeks before onset of symptoms) compared with only one third of single-event cases. Clearly, not only multiple-event cases experience a short-term effect; however, the findings suggest that previous *recent* stresses increase the likelihood of experiencing an event in the 16 weeks before onset of illness. This suggests that for some cases (19% in this study) an additive effect is occurring between recent events over the 12-month period.

When considering rapid effects of events, the number of recent events appears unimportant. Thus in acute crises (i.e. symptoms occurring immediately and up to four weeks after an event) symptoms are partially or totally dependent on the effects of a single event but occur in multiple-event cases.

Interestingly, single-event cases occur throughout the 12 months period, with almost half of the cases experiencing their event 36–52 weeks before onset, i.e. a long-term effect. Furthermore, long-term effects are more likely to be associated with experiencing a single event and few multiple-event cases (less than 10%) experience their event nearest onset in this time period.

These results suggest that the mechanisms by which events exert their effects are likely to be different and depend to some extent on their number and timing. Possible mechanisms can be considered according to the three differences in timing already described. Thus, for the long-term effects of single events:

(a) the effects may be a result of buffering factors antedating the event and influencing its impact. Such buffering factors may occur in the child's social supports or family relationships (Rutter, 1985)

(b) the nature of the event may be such that protective factors are activated subsequent to the event, e.g. serious illness, family loss, or death may mobilise support for the child
(c) the salience of the event may be important. Thus, an event may not be fully understood at the time of occurrence and its personal meaning and impact may not become explicit until later.

These potential mechanisms merit further study.

As indicated, the short-term effects of multiple-events suggest an additive effect. Whether this additivity is a non-specific quantitative effect or contains qualitative factors is not clear. In studies on adult depressed women Brown & Harris (1978) found evidence for additivity only for unrelated events (i.e. events of different type and nature). Are multiple events experienced by children related or unrelated, and how does this effect the appraisal of such events? Brown & Harris (1978) considered four ways in which events might 'add': 'mechanical additivity', which may occur independently of the meaning of events, implying a threshold of stress above which psychiatric disorder is likely; 'general appraisal', where, although events are perceived as 'unrelated', the subject feels unable to cope with any further personal stressors; 'specific appraisal', where events have particular meaning and implications for the subject which indicates that one event may be related to another; and 'symbolic appraisal', where the first event influences the second through individual personal significance, unrelated to contextual information, but reflecting intrinsic psychological associations made by the subject. All four types of additivity can be usefully considered for childhood and adolescence.

The present results suggest that for conduct disorders events may exert a degree of mechanical additivity, as they exert effects independent of the nature of social context (Goodyer *et al*, 1985*a*). However, for the other three groups of mood and somatic presentations there seems likely to be a more qualitative appraisal. Whether this is general, specific, or symbolic for one or all of the groups cannot be clearly stated from the findings.

Finally, rapid effects occur independent of the number of events. This mechanism is not a function of additivity. Since the time period between event and onset of symptoms is short this may reflect a direct effect of an event on well-being; whether this is a result of qualities of the event, for example its severity, or of particular types of appraisal by certain children is unclear.

Perhaps certain types of children are more likely to respond with rapid effects to undesirable events. Factors which make them liable to do so may be intrinsic (temperament, organic factors) or extrinsic (family, social factors). Adverse experiences in the past or persisting as ongoing difficulties (Brown & Harris, 1978) may also contribute but were not examined in this study.

We hoped to provide some answers to four questions from this research. The first three have been addressed: events carry an increased risk of psychiatric disorder; they exert long-term, short-term, and rapid effects dependent to some extent on the number of events; and there is additivity of events in some cases, principally increasing the likelihood of a short-term effect.

The fourth question concerned the specificity between timing and number of events and type of disorder. Overall the proportions of cases reporting events is similar between the clinical groups, with a half to two-thirds of all cases experiencing one or more events. The distribution of events over time indicates little specificity between clinical group and type of effect (long-term, short-term or rapid).

The number of events for each clinical group shows that only conduct disorders have more multiple-event than single-event cases. We have previously shown that events carrying no or mild negative impact are associated with conduct disorder but no other group (Goodyer *et al*, 1985*a*). Since these mild events are not included in this analysis we may have underestimated the effects of multiple events for conduct disorder.

The small numbers in the clinical groups prevent firm statements about the specificity of timing and numbers of events. However, there is a suggestion that conduct disorders are the least likely to experience long-term effects and the most likely to experience multiple events. On the basis of the results this finding is what one might predict, as multiple events are more associated with short-term effects and we have already speculated that mechanical additivity of events may be more likely in conduct disorder.

The lack of specificity between timing and number of events and diagnosis may be a function of sample size, but may also be improved by further clarifying the nature of life events. For example, the findings of Finlay-Jones & Brown (1981) suggest that dangerous events are more associated with anxiety and loss events with depression.

A number of methodological issues require comment. Firstly, we previously indicated that there are likely to be advantages in obtaining life event data from children, particularly as the personal appraisal of stressors by children requires considerable further research (Goodyer *et al*, 1985*a*). Secondly, we cannot be sure that the effects exerted by life events on disorders are not simply a function of some other factor not accounted for in this research. Thus a spurious association between events and disorder, while unlikely in our opinion, may be present. Clearly the relative contribution of events to disorder requires examination. We have already indicated that some 30% of cases did not experience a recent severe life event and 17% of community subjects did. Thus recent events carrying a moderate to severe degree of negative impact may be sufficient in some instances but clearly not necessary in all for the development of psychiatric disorder in young people.

Although these methodological issues must be addressed in future studies the results presented here do indicate that recent stressful events exert different effects on emotional and behavioural disorders, according to their number and timing.

Acknowledgements

This work was partially supported by the Northern Regional Health Authority. The authors wish to thank Dr Ingrid Davidson, Mrs Barbara Kay, Mr Gordon Polinger and Mrs Mary Fleeting.

Appendix

'Relative risk' measures the association between a particular factor and risk of a certain outcome, and is the ratio of the rate of disease among those exposed to the rate among those not exposed. This measure of magnitude depends on a precise enumeration of the number of cases in the general population or sub-groups of it in order to estimate incidence rates in exposed and non-exposed populations. This presents difficulties in psychiatry, and it is not possible to estimate incidence rates in exposed and unexposed populations from a retrospective design. However, an approximation of relative risk can be made from a case control study using a 2 × 2 contingency table. The assumption made is that instead of representing cases and controls the entries reflect the true proportions of the population with and without the disease and with and without the cause. Calculation of relative risk from a 2 × 2 contingency table constructed from a case-control study can be made as follows:

Suspected cause	*Cases*	*Controls*
Present	A	B
Absent	C	D

Therefore:

Risk of disease in those with cause = $A/(A+B)$

Risk of disease in those without cause = $C/(C+D)$

Relative risk = $(A/(A+B))/(C/(C+D))$

If the proportion of the population affected by the disease is relatively small compared with those unaffected then:

$B \approx A+B$

$D \approx C+D$

and the formula reduces to AD/BC.

If the proportion of the population affected by the disease is relatively large, the ratio is a useful measure of disease risk between groups and is best termed 'relative odds', i.e. the ratio of affected to unaffected individuals is one divided by the same ratio in another group.

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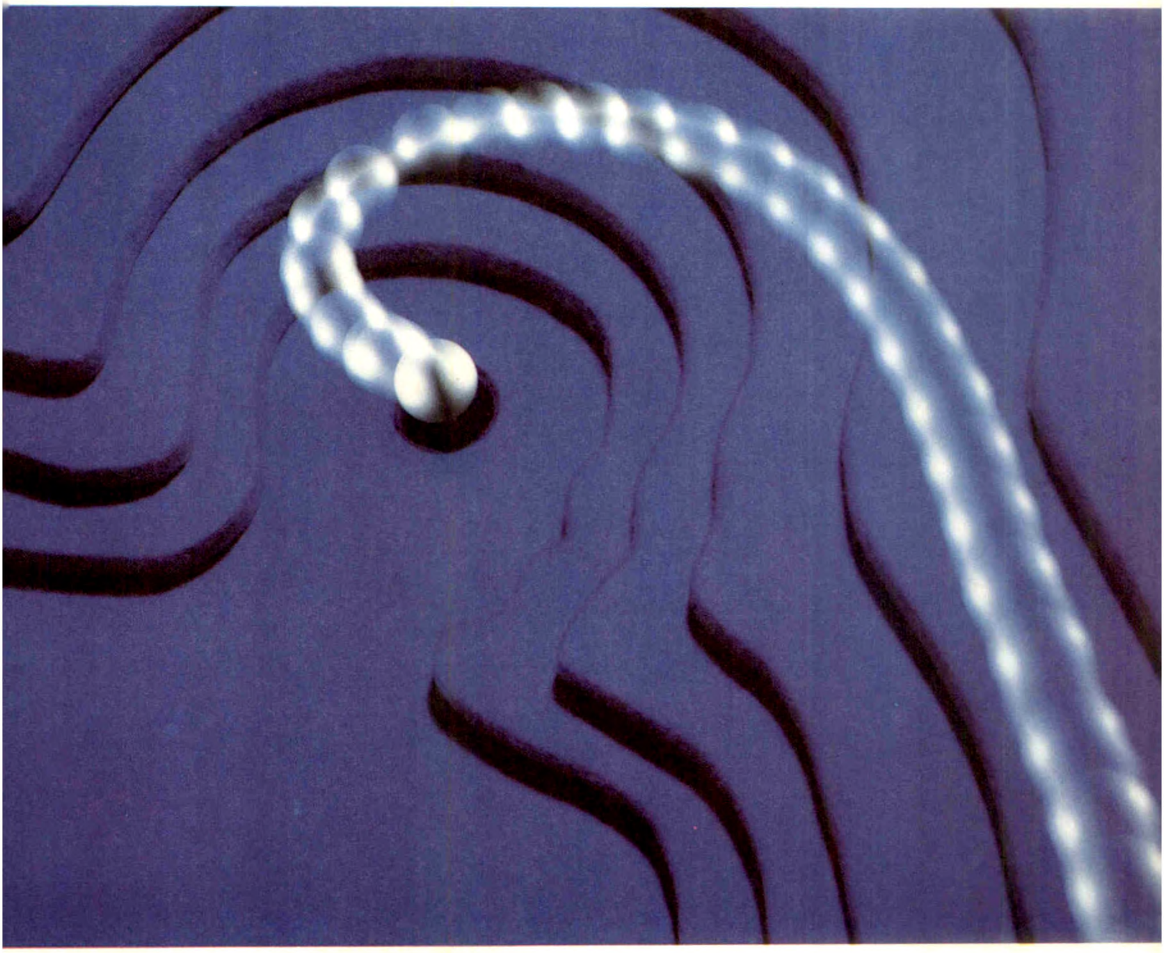
Prescribing Information ▼ **Presentation:** Round, yellow, enteric-coated tablets each
50mg fluvoxamine maleate, imprinted with 'Duphar 291'. Pack of 60 tablets, basic NHS price
ses: Treatment of those affective disorders characterised by persistent low mood, impaired
or activities and psychosomatic complaints. **Dosage and Administration:** The tablets should
ved without chewing and with water. Adults: The effective daily dose usually varies between
200mg and should be adjusted to the individual response of the patient up to a maximum of
e minimum recommended starting dose is 100mg given in the evening. A total daily dosage in
00mg should be given in divided doses. Elderly: The same dosage regimen may be utilised in the
n the general population. Children: There are insufficient data to recommend the use of Faverin in
ontra-indications, Warnings, etc: Faverin should not be given together with or within two
terminating treatment with monoamine-oxidase inhibitors. Patients with hepatic or renal
cy should begin treatment with a low dose and be carefully monitored. Faverin has no effect on
or skills associated with driving and operating machinery. However, in combination with alcohol
irment may be seen. Use in pregnancy and lactation: There has been little human use but there
o adverse animal evidence. Avoid in pregnancy and in nursing mothers if a better known and safer
is available, although safety in pregnancy is likely. Interactions with other drugs: In interaction
studies increased plasma levels of propranolol and warfarin were seen during concurrent admini
Faverin. Care should be taken when Faverin is prescribed to patients already receiving propra
warfarin as the dose of these drugs may need to be lowered. No interactions were seen with digo
atenolol. **Side-effects:** The commonest symptom observed with Faverin administration is nau
usually diminishes early during the course of treatment. Other treatment emergent symptoms
during clinical studies were somnolence, constipation, agitation, anorexia and tremor. Other report
are often associated with the illness and their incidence in controlled trials is comparabl
experienced with placebo. Faverin may cause a decrease in heart rate. During treatment, limited
repolarization were observed in the ECG, but no causal relationship could be demonstrated. The in
postural hypotension was similar to that seen in patients treated with placebo. Treatment of overd
specific antidote is known. The stomach should be emptied as soon as possible after tablet ing
symptomatic treatment should be given. The use of medicinal charcoal is also recommended.
Quantities 50mg tablets: available in packs of 60 (3 strips of 20 blister-packed tablets). **Product
Number** 50mg tablets: 0512/0070. Further information is available from: Duphar Laboratorie
Duphar House, Gaters Hill, West End, Southampton SO3 3JD. Tel: Southampton (0703) 472281.



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Aloise was diagnosed as schizophrenic at the Cery Asylum, Lausanne. She was later transferred to the Asylum de la Rosiere where she remained for over 40 years until her death in 1964.

During the last 20 years of her life she worked steadily at her drawings.

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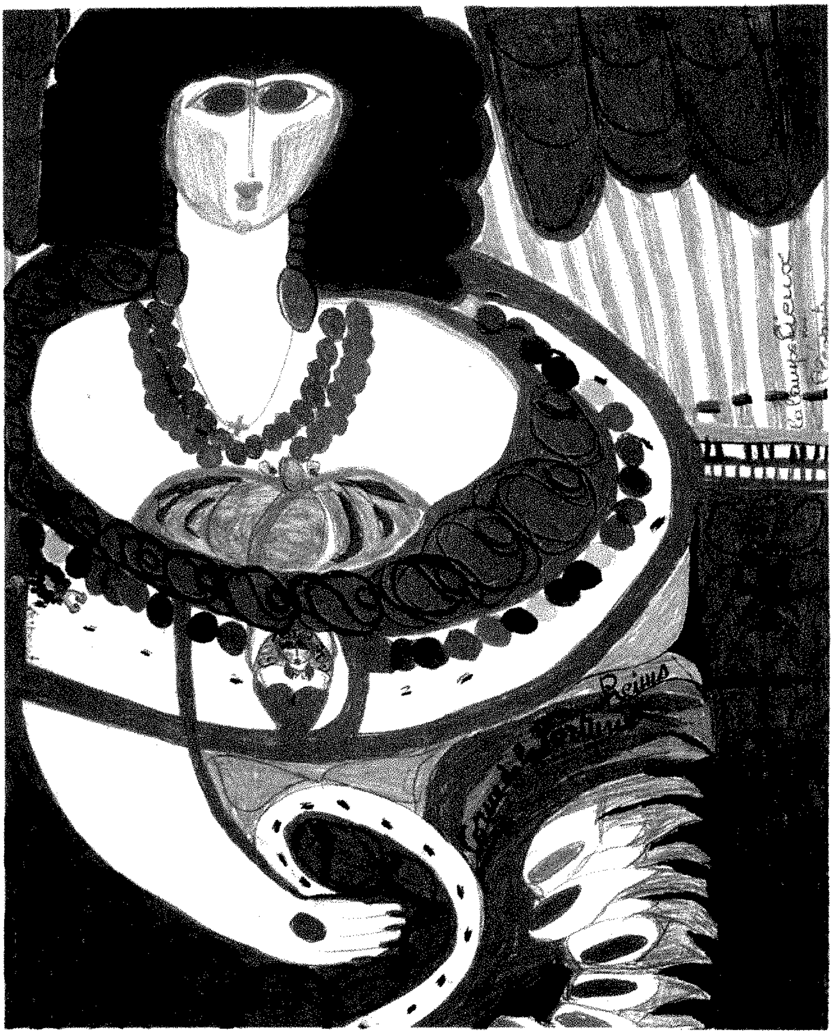

A TREATMENT.

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In mild symptomatology the dosage for Haldol* Decanoate is from 50-100mg given 4-weekly, whereas severe schizophrenic symptoms are usually controlled with up to 300mg.

The schizophrenic patient can benefit from a flexible therapy with little incidence of sedation[1] and a reduced requirement for antiparkinsonian medication,[2] leaving him/her better able to participate in other forms of therapy.

References: 1. Pakes G. E. in 'Therapeutics Today' Ed D. A. W. Johnson, ADIS Press. (1982). **2.**
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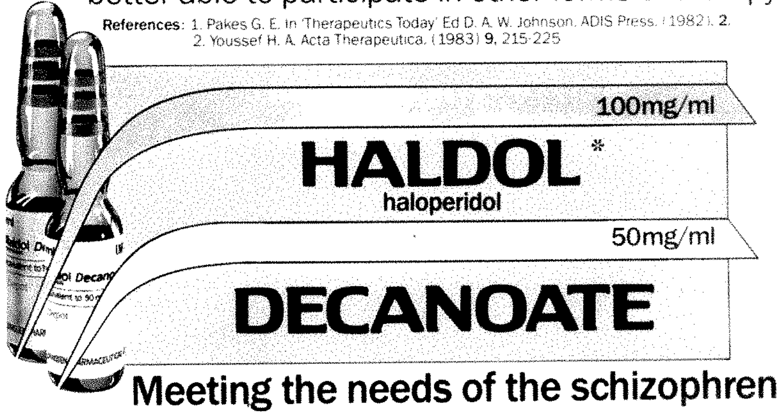

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Presentation: Straw coloured, viscous solution presented in 1ml brown glass ampoules equivalent to 100mg/ml haloperidol (as decanoate ester) and 1ml brown glass ampoules equivalent to 50mg/ml haloperidol (as decanoate ester) both in packs of 5. **Uses**: As a depot injectable form of haloperidol indicated for adults where long term maintenance treatment with a neuroleptic is required, for example in schizophrenia, other psychoses (especially paranoid) and other mental or behavioural problems where maintenance treatment is clearly indicated. **Side effects, precautions, contra-indications**: Haloperidol is not recommended during lactation. Use in Pregnancy: The safety of haloperidol in human pregnancy has not been established. Precautions: Caution in liver disease, Parkinson's disease, phaeochromocytoma, thyrotoxicosis, epilepsy and conditions predisposing to epilepsy. Antagonism of adrenaline, guanethidine, phenindione, anti-Parkinson effects of levodopa and impairment of metabolism of tricyclic anti-depressants have been reported. Haloperidol may increase the effects of CNS depressant drugs, and enhanced CNS effects when combined with methyldopa have been reported. Neurotoxic reactions to combined treatment with lithium and haloperidol have been reported. Side effects: In common with all neuroleptics, the following side effects may be observed, sedation, extra-pyramidal symptoms, tardive dyskinesia, mental dullness, dizziness, headache, excitement, agitation, insomnia, hyperprolactinaemia, galactorrhoea, gynaecomastia, oligo- or amenorrhoea, hypotension. Gastro-intestinal symptoms and weight changes have been reported. Anti-Parkinson agents should only be given as required. The elderly are more susceptible to sedative/hypotensive effects. Occasional local reactions such as erythema, swelling or tender lumps have been reported. In common with other anti psychotics, haloperidol has been associated with rare cases of neuroleptic malignant syndrome. **Dosage**: Haldol* Decanoate provides one month's therapy following a single deep intra-muscular injection in the gluteal region. An initial dose of 50mg every 4 weeks is recommended increasing if necessary after 2 weeks by 50mg increments to 300mg every 4 weeks. The dosage should be tailored to suit the individual patient and to reflect previous oral dosage. In adults 100mg Haldol* Decanoate is approximately equal to 500mg chlorpromazine (or equivalent) per day. Caution is required with the elderly, starting doses of 12.5mg to 50mg monthly should be tried first. **Product Licence Numbers**: 0242/0095, 0242/0094 **Basic NHS Cost**: 1ml x 100mg/ml x 5 £24.00 1ml x 50mg/ml x 5 £15.60. (Correct at time of printing). Further information is available on request from: **Janssen Pharmaceutical Limited**, Grove, Wantage, Oxon OX12 0DQ.

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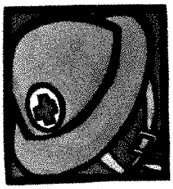

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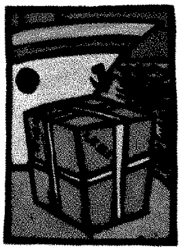

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British Journal of Psychiatry (1987), **151**, 185–191

Brief Psychotherapy in the Treatment of Anorexia Nervosa
Outcome at One Year

A. HALL and A. H. CRISP

Thirty out-patients with severe anorexia nervosa were randomly allocated to either 12 sessions of dietary advice or 12 sessions of combined individual and family psychotherapy. At one-year follow-up both groups showed significant overall improvement, and the dietary advice group showed significant weight gain. A similar mean weight gain for the psychotherapy patients did not reach statistical significance, but this group made significant improvements in sexual and social adjustment.

The long-term outcome of anorexia nervosa (AN) has been reviewed by Hsu (1980). Although outcome at one year bears some relationship to that at four years (Morgan & Russell, 1975; Hsu *et al*, 1979), longer-term studies (Theander, 1983) confirm the view that AN is often a relapsing remitting condition over many years, and that short-term restoration of body weight may have little bearing on future psychosocial functioning. In an attempt to reduce the relapse rate, psychotherapy in various forms has been incorporated into most treatment programmes.

While impressive results have been reported for family therapy (Palazzoli, 1978; Minuchin *et al*, 1978), there is little evidence that this short-term improvement is maintained in the longer term, and there have been no reports of prospective treatment studies evaluating the effectiveness of psychotherapeutic approaches (Garfinkel & Garner, 1982). The specific methodological difficulties of randomly allocating AN patients to different psychotherapeutic treatment modalities within one centre have been described by Szmukler (1983); those general to and inherent in all psychotherapy research have been discussed frequently (Bergin & Strupp, 1972; Luborsky *et al*, 1975; Sloane *et al*, 1975; Malan, 1976; Schlesinger *et al*, 1980; Conte & Karasu, 1981; Brodaty & Andrews, 1983). The dictates of study design and therapist variables in particular may interfere with delivery of a treatment which by its nature must be intuitive, flexible, and tailor-made for the needs of the patient. Frank (1971) identified the therapeutic factors in any patient-therapist relationship, and Strupp & Hadley (1979) attempted to investigate the relative contribution of the therapist's technical skills and of non-specific interpersonal components. DiMascio *et al* (1979) discussed the problems of a control group, and concluded that a non-scheduled treatment group had both ethical and scientific advantages for acutely depressed patients over waiting-list, assessment only, low or minimum contact, or untreated control groups. A controlled psychotherapeutic treatment study for patients with bulimia nervosa has been described by Lacey (1983): a mixture of individual behaviourally orientated therapy and group therapy produced improvement, as compared with no treatment, after initial assessment.

Since AN bears a significant mortality and morbidity, difficult ethical issues must be considered in any research design, but while specialist treatment resources remain scarce and no approach has proved definitively superior, limited treatment trials are justified, although studies with untreated controls probably are not. Morgan & Russell (1975), Crisp *et al* (1977), and Hsu *et al* (1979) have identified factors associated with a worse prognosis, which must be taken into account in any controlled study and in comparing results. Since the number of treatment variables should be kept to a minimum, an out-patient study is more feasible than an in-patient one, overcoming the problems of psychotherapeutic contact with other staff and the difficulties of maintaining blind treatment allocation. This approach seems justified by the findings of the St George's Hospital series (Hsu *et al*, 1979): 71% of the subjects treated with out-patient psychotherapy had good outcomes, compared with 41% after in-patient care. However, although the authors did not attempt to distinguish between the two groups in terms of factors predictive of poor outcome, the in-patient group, who received more intensive individual and family psychotherapy, were almost certainly more chronic and severely ill patients.

The aim of the present study was to investigate the effect on weight, eating behaviour, and social and sexual adjustment of either brief individual and family psychotherapy or dietetic advice for out-patients with AN. The clinical findings at one year follow-up are described.

Method

Subjects

Thirty female subjects who met the diagnostic criteria for primary AN were selected from consecutive referrals to one of the authors. To control for some of the factors found to affect prognosis in previous follow-up studies (Morgan & Russell, 1975; Crisp *et al*, 1977; Hsu *et al*, 1979) the subjects selected were aged 13–27, from social classes I–III (Registrar General's classification, Office of Population Censuses and Surveys, 1985), and unmarried. They all weighed less than 85% of matched population mean weight (MPMW), had amenorrhoea, and had been ill for between 6 and 72 months.

The referring agent–in most cases the subject's general practitioner–was informed that out-patient treatment in the form of either brief psychotherapy or dietetic advice would be offered, and was advised to seek additional treatment elsewhere should this be insufficient to prevent serious deterioration during the period of the study. However, if admission or further treatment were indicated at the one year follow-up appointment it would be made available within the department.

Referrals were screened initially by postal questionnaire, and those meeting the criteria for inclusion were invited, together with their families, for a two-hour assessment interview. Each subject and her family were interviewed by a psychiatrist and a dietitian, to record full clinical and demographic details, including weight, desired body weight, eating pattern, history of AN, past and current relationships with family, and psychosexual history. The clinical and family dynamic assessment took the form outlined elsewhere (Crisp, 1980): it included evaluation of the subject's personality, mental state, family dynamics, and motivation for treatment which had the goal of increasing insight, including psychological change in both the subject and family. Each subject completed a Crown-Crisp Experiential Index (CCEI) (Crown & Crisp, 1979) and scores were calculated for body weight, menstrual function, eating pattern, mental state, and sexual and social adjustment (Morgan & Russell, 1975; Hsu *et al*, 1979). The global clinical score for each subject was the mean of these six scores.

All subjects who met the criteria for inclusion in the study were willing to attend for out-patient treatment and, following assessment, were randomly allocated to treatment with either the psychotherapist or the dietician.

Psychotherapy group

Fifteen subjects were each seen for 12 sessions of one hour's duration at one to two-weekly intervals, by the same psychotherapist. The proportions of individual psychodynamic therapy and family therapy depended on clinical judgement, practicability, and the willingness of the family to be involved. The therapist was trained and experienced in these therapeutic approaches, and in the flexible integration of family and individual work as appropriate. In each case attention was focused on the role AN played in the relationship of the subject with her family and others. An attempt was made to change those aspects of relationships which tended to stifle the subject's development and maintain the anorexia, particularly over-protectiveness, conflict avoidance, enmeshment, and distancing within the family. The specific goals of treatment varied in each case, particularly depending on the age of the patient, but were broadly to encourage her development, both within and separately from her family, and to promote insight. All patients were seen by the dietitian for four 15-minute interviews during the course of treatment, during which they were weighed, their general progress (particularly regarding eating behaviour) discussed, and dietary advice given.

Dietary advice group

Fifteen subjects were seen for twelve one-hour sessions at weekly or fortnightly intervals. Five dietitians were involved; each treated the subjects she had assessed, and all were experienced in the dietary management of AN. Each session was spent discussing diet, mood, and daily behaviour patterns. The family was seen with the subject on some occasions. The goals of treatment were to restore normal eating patterns and dietary constituents, to relate eating behaviour to mood, and to increase the confidence of the subject so that she could remain in control as she gained weight. All subjects were seen by the psychotherapist for four 15-minute interviews, during which they were weighed, then general progress discussed, and advice given, particularly about family and social relationships.

At the end of treatment all 30 subjects again completed the CCEI, and their general practitioners were contacted.

Follow-up

One year after the assessment interview, all the subjects and their families were interviewed by an assessor who was blind to the treatment allocated. The clinical and demographic details recorded initially were reviewed, and the CCEI repeated. Sexual and social adjustment scores were calculated, and the mean of these, body weight, food intake, menstrual function, and mental state scores was calculated to give the global clinical score. Details were taken of treatment received elsewhere and of other life changes in the subjects or families since the last contact with the department. The assessor reviewed each subject's progress in terms of her AN, and her overall development, both social and sexual, as an individual and within the family. Whenever indicated, recommendations for further treatment were made. If the patient and her family did not accept the offer of further treatment, alternative follow-up was offered.

Results

Characteristics at initial assessment

The clinical features at presentation are summarised in Table I. There were no statistical differences between the

TABLE I
Clinical features at presentation

	Psychotherapy group (n=15)		*Dietary advice group (n=15)*	
Age: years				
Mean	19.55		19.57	
Range	14–25		13–27	
Social class[1]				
Groups I+II: *n*	12		13	
Group III: *n*	3		2	
Age at onset of illness: years				
Mean	17.07		17.53	
Range	12–21		12–25	
Age at onset of amenorrhea: years				
Mean	17.77[2]		17.90	
Range	13–21		12–25	
Mean duration of illness: months	29.7		24.5	
Mean duration of amenorrhea: months	27.5		20.1	
Number having had previous treatment	10		8	
Mean sexual adjustment score	5.85		6.75	
Mean social adjustment score	5.50		7.05	
Weight: kg				
Mean	41.00		39.54	
Range	29.4–53.8		33.1–43.3	
Mean height: cm	161.7		162.3	
Deviation below MPMW				
Mean	25.35%		28.16%	
Range	15–37%		16–46%	
Mean desired body weight: kg	42.7		44.2	
Mean weight at onset of dieting: kg	52.50		55.42	
	Usual	*Occasional*	*Usual*	*Occasional*
Reduced food intake	14	1	13	1
Bulimia	3	3	2	5
Vomiting	3	0	2	1
Purgative abuse	2	4	3	6
Anxiety eating with others	6	7	5	7

1. Registrar-General's classification (Office of Population Censuses and Surveys, 1985).
2. Two of this group never menstruated.

two groups on any of these variables, or on individual CCEI scales. The clinical features and CCEI profiles resembled those of other studies (Morgan & Russell, 1975; Crisp *et al*, 1978, 1980).

Treatment compliance

Fourteen of the psychotherapy group (PG) patients completed 12 sessions of psychotherapy. One subject dropped out after six sessions, by which time her weight was within 8% of her MPMW and her menstruation had resumed; her improvement was sustained at follow-up.

Eleven of the dietary advice group (DAG) patients completed treatment. One subject gained 4 kg after the assessment interview, and refused further treatment; at follow-up she had gained more weight, but still had AN. Three subjects attended for three, seven, and nine sessions respectively; two of these gained weight during the period of treatment only and then remained static until follow-up, while the third lost weight during treatment, but had gained 10 kg by follow-up without resumption of her menses. During the period of the study she sought, and obtained, intensive treatment by a hypnotherapist. Another patient in this group was treated by an acupuncturist.

Body weight

Figure 1 shows the mean change in weight, expressed as percentage deviation below MPMW, for each group after 12 sessions of treatment and at follow-up. There was no

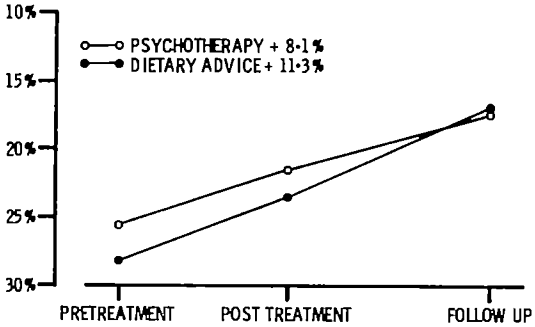

FIG. 1 Percentage deviation below matched population mean weight

statistical difference between the two groups, both of which showed a similar overall gain. This gain was significantly different ($P < 0.001$) from the presentation weight for the dietary advice group only; the PG patients made similar gains, but three showed major weight losses (mean 9.0 kg) after treatment ended, which to some extent masked major gains (mean 10.4 kg) made by eight others, and resulted in a large standard deviation (8.04 kg) in weight at follow-up. In contrast, the DAG patients, with a standard deviation of 6.07 at follow-up, had sustained their gain since treatment, resulting in a significant mean weight gain overall.

Clinical features

The clinical features at one year are shown in Table II.

There were no significant differences between the two groups on any of the individual measures, except in the social ($P < 0.005$) and sexual ($P < 0.10$) adjustment scores, which were significantly higher for the PG. The PG showed a significant improvement in social adjustment ($P < 0.001$) and in sexual adjustment scores ($P < 0.001$) between presentation and follow-up, while the DAG showed slight non-significant improvement. Both groups showed significant improvement ($P < 0.001$ for PG; $P < 0.01$ for DAG) in global scores after one year (Fig. 2).

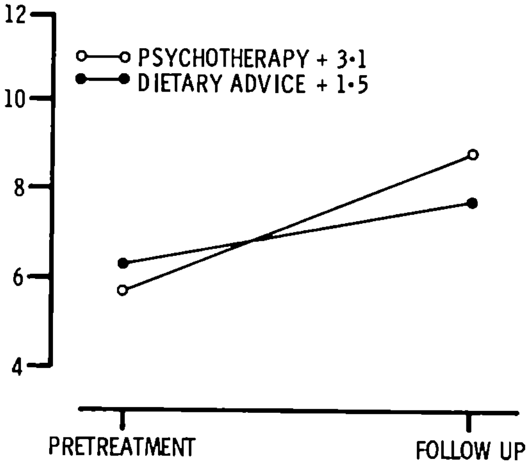

FIG. 2 Global clinical scores

TABLE II
Clinical features at one year follow-up

	Psychotherapy group (n=15)	*Dietary advice group (n=15)*
Weight: kg		
Mean	45.1	46.0
Range	26–56	33–57
Deviation below MPMW		
Mean	−17.4%	−16.9%
Range	−54% to +5%	−37% to +12%
Mean desired weight: kg	47.9	47.8
Menstruation		
Regular	3	3
Irregular	4	2
Absent	8	10
Mean sexual adjustment score	9.2	7.1*
Mean social adjustment score	9.2	7.9**
Mean global clinical score	8.8	7.8

*$P < 0.01$ for difference in mean change between groups
**$P < 0.005$ for difference in mean change between groups

Changes following treatment

The two groups differed in the treatment received elsewhere during the study. None of the PG sought outside treatment, but one patient, who had made progress, deteriorated seriously as soon as treatment ended, and was admitted for 91 days to a medical ward for intravenous feeding, while another was seen as a medical out-patient for diarrhoea contracted during foreign travel. In the DAG, one patient attended an acupuncturist and another a hypnotherapist. After treatment with the dietitian ended, a further four patients attended 68 out-patients appointments for AN. Another patient in the same group, despite gaining weight with resumption of menses, was admitted to a psychiatric hospital for 126 days and attended a day hospital for 78 days; she made seven suicide attempts. Another patient was admitted for 123 days for treatment of her AN.

Nine of the PG patients visited their general practitioner for an average of two visits each after treatment ended, while nine of the DAG who did not receive other treatment made a total between them of 32 visits. Three received medication continuously and one briefly prior to follow-up, while four psychotherapy patients were prescribed medication for a short time.

During the year of the study three of the PG left home and ten acquired boyfriends, compared with one and three respectively of the DAG. The difference in respect of the boyfriends is significant ($\chi^2 = 6.65$, d.f. = 1, $P < 0.01$).

At follow-up, the parents of one subject in each group had separated. The assessor considered that there had been significant family realignment in the families of 12 of the PG subjects, compared with four of the DAG.

Both groups showed marked improvement at follow-up in eating behaviour. The reduction in the symptoms of food avoidance and anxiety about eating with other people was more marked in the PG, while the DAG tended to show a greater improvement in bulimia, vomiting, and purgation. The symptom of 'weight phobia' improved, and was reflected in the desired body weight, which increased by 7 kg for the PG and by 3.5 kg for the DAG (Table II).

Seven of the PG and five of the DAG had resumed menstruation at one year.

CCEI changes

There were no significant differences in CCEI scores between the two treatment groups. The DAG showed significant decreases in the somatic ($P < 0.005$), phobic ($P < 0.025$) and depression ($P < 0.05$) scales (Fig. 3), whereas the PG showed significant ($P < 0.05$) but small falls in the obsessional and depression scores (Fig. 4). One patient, who had deteriorated clinically, refused to complete her CCEI at follow-up.

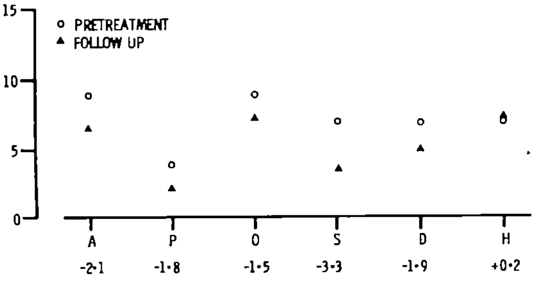

FIG. 3 Mean CCEI scores for the dietary advice group ($n = 14$). The scales are designated as follows: A = anxiety; P = phobic; O = obsessional; S = somatic; D = depressive; and H = hysteria

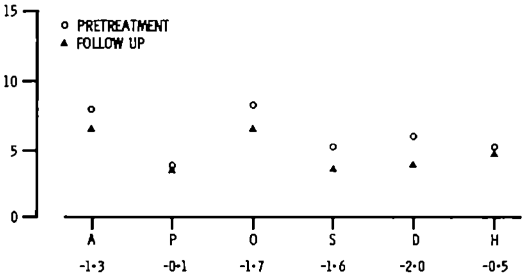

FIG. 4 Mean CCEI score for the psychotherapy group ($n = 15$)

Treatment recommendations

At follow-up, four of the PG were considered recovered and did not require further treatment; all 11 remaining patients accepted and attended the recommended further psychotherapy or infrequent follow-up appointments. All 15 of the DAG were felt to require further treatment but only ten accepted the offer, and two of these dropped out almost immediately. The independent assessor detected a greater degree of motivation to overcome anorexia on the part of the PG; he noted that some of the DAG were reluctantly co-operative in their acceptance of treatment.

Discussion

It has been suggested that it is simplistic to focus on weight gain and return of menstruation alone as indicators of improvement in AN, and that intrapsychic and interpersonal conflicts, sexual and social adjustment, and continuing development are at least as important (Bruch, 1974; Crisp, 1980; Garfinkel & Garner, 1982). This study demonstrates that significant improvement in overall functioning, as reflected in the global clinical scores, may have been achieved by two different brief interventions: dietary advice, or a mixture of individual and family therapy.

On individual measures both groups show modest improvement in weight, menstruation, eating patterns, and sexual and social adjustment after one year, but differ significantly in outcome on three measures. The DAG showed significant weight gain, whereas the PG made significant improvements in sexual and social adjustment. When the patterns of change in weight for each patient are examined more closely, the two groups show distinct differences. The DAG had a more uniform pattern during treatment of modest gains in weight, which tended to be sustained between the end of treatment and follow-up; these patients were more likely to seek alternative treatment elsewhere during the treatment period and afterwards, which may have sustained them. In contrast, the PG was characterised by larger weight gains during treatment, but the subjects tended either to continue to improve, or to relapse–in three cases seriously–during the period in which there was no contact with them.

The changes in CCEI scores, which on some scales reached statistical significance at follow-up, showed trends supporting these findings, and may be related to aspects of the psychotherapeutic process. The PG patients tended to have higher anxiety and depression scores after the 12 treatment sessions, whereas the other group showed a small increase in the obsessional scale; both groups showed lower mean scores overall at one year. Crisp *et al* (1979) have shown that increased anxiety and depression during treatment are associated with improved long-term prognosis, and may indicate a reduction of the denial characteristic in AN. The CCEI changes are difficult to interpret, but might reflect intrapsychic changes and emotional awareness provoked by psychotherapy, whereas dietary advice may reinforce denial, obsessional defences, and control–in some cases stabilising the course of the illness. This provides an explanation for the deterioration of some PG patients when the 12 sessions of treatment ended, and illustrates a major drawback of rigid treatment design in psychotherapy research.

The general problems of study design in psychotherapy research are present in this study. Firstly, patients subsequently allocated to 'dietary advice' nevertheless also receive, together with their families, an initial extensive diagnostic evaluation and related discussion. Crisp (1980) and Malan *et al* (1975) have emphasised the occasional important psychotherapeutic effect of such interventions. Secondly, neither treatment approach can be 'pure'; inevitably, the dietitians discussed family and other issues raised in their sessions, while the psychotherapist at times focused on eating patterns and other matters relating to diet. Treatment of the dietary advice group was additionally complicated by other treatments received simultaneously by two of the patients. A further five patients were treated later in other psychiatric departments. We can assume, therefore, that up to half of the DAG may have received some psychotherapeutic treatment prior to follow-up: all of these had gained some weight between assessment and follow-up, but none had received family therapy or intensive psychotherapy. The contribution of these other treatments needs to be considered. Finally, the independent assessor could not always remain totally blind to the treatment received.

The one-year follow-up period is too short to make any long-term predictions, particularly as the majority of the patients required further follow-up or treatment–either admission to hospital or out-patient individual and family psychotherapy. The greater persistence of the PG in pursuing treatment, both at the outset and after follow-up, is clear.

Although the follow-up period was relatively short, the study supports the findings of others that many patients, even with long-standing AN, show improvement in physical and psychological functioning after a short period of intensive treatment of one kind or another, and that admission to hospital may not be necessary. The differences between the two groups highlight the changes which occur as a result of distinct elements of treatment, which are widely accepted but generally combined in most treatment centres. Our results emphasise that attention to both eating behaviour and psychological change are important in the treatment of AN, but offer a warning about the need for long-term continuity of treatment in some cases, particularly when intrapsychic or interpersonal changes are taking place.

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Anorexia Nervosa: An Object Relations Approach to Primary Treatment

M. HORNE and M. GALLEN

Concurrent behaviour therapy and psychotherapy in the treatment of anorexia nervosa creates significant problems in the development of a therapeutic alliance and in the multiple roles staff are required to assume. Our protocol was developed to keep treatment personnel in their usual roles so that their various treatment skills could be fully utilised. A physician treated the anorexia with naso-gastric feeding, freeing the psychotherapist to act in the usual manner. This avoided role confusion, and the split of 'bad' and 'good' treatment personnel was appropriate and therapeutic, rather than pathological. Four successful cases are described.

Anorexia nervosa (AN) can cause severe cachexia, which can be extremely difficult to treat. A variety of techniques have been used to initiate weight gain, but long-term changes require psychotherapy (Bruch, 1974; Whipple & Manning, 1978). In addition to the physiological effects of cachexia and the ultimate risk of death there are pronounced cognitive and emotional changes which interfere with effective psychotherapy (Fox, 1981). Rapid and effective weight gain is important both for physical and psychological well-being.

Behaviour therapy is currently a fashionable method to induce weight gain. Our own experience with this method has been disappointing, firstly because weight gain is slow, inconsistent, and non-durable, and secondly because the most common behavioural treatment makes the consequence of the failure to gain weight the deprivation of ward privileges, and rewards weight gain by the granting of ward privileges (Agras & Werne, 1978). In this situation the reward is a reintroduction of the *status quo*, and not really a positive reinforcement. Deprivation of ward privileges is therefore the aversive consequence of a failure to gain weight. This aversive consequence is dependent on the outcome (i.e. weight gain) of a series of types of behaviour rather than a direct consequence of a type of behaviour itself as in the usual operant paradigm. The paradigm is therefore not really aversive operant conditioning – it is simply punishment for not gaining weight. The struggle for behavioural control between the mother and the patient is repeated between the therapist and the patient, without the scientific underpinning of a correct operant paradigm.

A working therapeutic alliance is difficult to create with anorexic patients because of their primitive object relations development (Sours, 1980; Masterson, 1977). Using behavioural therapy in conjunction with psychotherapy makes the therapist a bad object in connection with the former and a good one in connection with the latter, inhibiting the development of a working alliance. In addition there is role confusion among staff, as they are required to be both behaviourists and psychotherapists simultaneously. These patients use primitive defences such as projective identification and splitting; this creates additional chaos, as staff already confused in their roles become further split into good and bad objects (Cohen, 1978). On the comprehensive medical unit at Stanford Hospital we developed our treatment programme to avoid these difficulties.

Physicians are required to diagnose and treat physical illness; they work in a well-defined and socially sanctioned paradigm. We reasoned that since the cachexia caused by AN is a severe physical illness we should define it as such and ask the physician to treat the cachexia within the normal medical paradigm. The physician is then in his normal role, and the anorexic patient in this context assumes the role of a physically ill patient. The treatment for bowel disorders where nutrients are not being absorbed is some form of dietary supplementation, or parenteral feeding, depending on the condition. The logical treatment for anorexia is therefore naso-gastric tube feeding. The complications of naso-gastric feeding with a fine bore naso-gastric tube, constant infusion and careful metabolic monitoring are minimal, and far outweigh the dangers of cachexia (Silk, 1984).

In conjunction with the tube feeding, a psychiatrist and psychologist work collaboratively using an administrative therapy model. The psychiatrist deals with administrative, management and psychopharmacological issues, while the psychologist begins his or her work with the patient in the usual psychotherapeutic manner, free from any need to step out of his or her role.

The 'prescribed' splitting which occurs aids therapy, as the phyician and psychiatrist automatically become the bad objects and the therapist the good object, helping the formation of the therapeutic alliance.

We describe the treatment of four typical cases of AN using this method.

Method

The four female patients in this study had an unequivocal DSM-III diagnosis of anorexia nervosa (American Psychiatric Association, 1980). Their ages and weights (as a percentage of ideal body weight are given in Table I. All had begun to show features of the illness as teenagers. Three of the patients were admitted to the psychosomatic ward of Stanford University Hospital and one was admitted to a general medical ward. Two of them were already in psychotherapy and two had just presented for treatment. All patients were resistent to starting tube feeding, but were co-operative once the procedure was begun. The procedure was voluntary in all cases.

A paediatric naso-gastric tube was passed and remained in situ throughout the hospital stay. A nutritionally complete enteral formula (1 kcal/ml) was infused at a rate dependent on the presence of upper gastro-intestinal discomfort or loose bowel motions. A weight gain of 0.1–0.2 kg/day was maintained.

The physician co-ordinated all aspects of the medical management; the psychiatrist determined all aspects of case administration and management, and the psychologist saw the patient for daily psychotherapy. All patients had concurrent family therapy with a separate therapist, and the three patients who were married had some marital therapy.

Results

Weight gains, length of hospital stays and follow-up periods are given in Table I. At follow-up, three patients had successfully terminated therapy, and all had maintained their weight gains. They felt considerable anxiety when the procedure began, and then relief when they realised that they did not have to control their food intake. As treatment progressed, all patients began to eat in addition to the tube feeding. When body changes began to occur the patients became anxious again, but also more affectively responsive, and all therapy situations became more active and intense. This was especially noticeable in family and marital therapy. There were some attempts to split the physician and psychotherapist in relation to bargaining about rapidity of weight gain and the endpoint to treatment. However, all these issues were decided by the physician in relation to the patient's medical condition. Cognitive and general physical abilities improved throughout treatment.

TABLE I
Details of four patients treated by the method described

Variable	*Patient* 1	2	3	4
Age at admission: years	21	18	32	27
Percentage of ideal body weight at admission	74	67	70	71
Increase in weight during treatment: kg	6.6	5.5	7.8	5.5
Length of hospital stay: days	31	24	15	11
Length of follow-up: months	19	3	25	19

Discussion

These four cases demonstrate the effectiveness of this method in instituting rapid weight gain; in addition, the intensity and relevance of therapy were enhanced and the detrimental effects of pathological splitting reduced. All four patients showed such a similar pattern of response: this was characterised by initial anxiety, then relief, then fear, as body changes began, and then more intensity and relevance in the therapeutic work. It was as though the defence had been taken away and the inner conflicts had been laid bare, but in a context where there was adequate therapeutic support to deal with them. This effect was noted by Maloney & Farrell (1980) in their study using hyperalimentation. After conquering the initial fears that tube feeding would take away control of weight gain, patients were relieved that they did not have to engage in the daily struggle with food.

In all cases, the issues revealed in hospital required considerable further therapeutic work, but they could no longer be denied. This was especially important in regard to interpersonal issues; eating behaviour could no longer be the primary issue in the marriage or family. The psychotherapist continued to work with the psychological meaning of the disturbed eating behaviour to the patient. However, we reduced the patient's denial of the medical consequences of cachexia by confronting this as a serious medical issue. By assuming control of the patient's nutrition the physician started a struggle for control of the patient's health which was initially external, but with the ultimate goal of making this internal. We did not expect the patient to return to normal weight; only to stay out of medical danger. We set the threshold weight at 43 kg. The ultimate return to normal eating behaviour involved long and painstaking psychotherapeutic work.

It could be said that the essential element of an object relations paradigm had been satisfied by splitting the weight gain method from psychotherapy; therefore, why not use other methods to induce weight gain? We rejected behaviour therapy, as currently practised, on the theoretical grounds outlined previously. In addition, Agras & Kraemer (1984), in a summary of behaviour therapy studies compared with non-behavioural studies, have shown that behavioural methods are only slightly superior.

In one of the few studies designed to establish the variables responsible for weight gain (Agras *et al*, 1974) it was shown that positive reinforcement (reinstatement of ward privileges) only induced weight gain if accompanied by feedback as to the calories eaten, mouthfuls eaten and weight gained. Patients realised that it was safe to gain a small amount of weight, and therefore gave up control to the therapist and allowed this to happen. This was an important element of our method. Patients were aware of the flow rate and concentration of the material infused. The physician adjusted this to keep weight gain between 0.1 and 0.2 kg/day. Patients would eat as desired, and ate more or less depending on how anxious they felt about weight gain – especially when body changes were occuring. The physician consistently maintained the steady weight gain, with the rationale that health needed to be restored but that the patient's fears would be respected and that some element of control (i.e. the fixed rate of weight gain) would be given to the patient. The feedback created a feeling of safety and added to the patient's trust in her physician. Our paradigm therefore used an essential element of the behavioural method in a more direct and theoretically accurate manner. In essence we provided the 'good-enough parenting' described by Winnicott (1965), making sure that health was restored to reasonable levels so that the struggle with good and bad objects could safely and therapeutically occur.

An important point was that weight maintenance at a safe level was now linked to medical necessity. It could be said that tube feeding would encourage patients to deny responsibility for their behaviour. However, this is an essential element of the illess: all anorexic patients deny the serious consequences of their eating behaviour. Our method focuses serious medical attention on the consequences of the denial, while attempting to undo the denial by psychotherapy.

Nursing and general ward management of these patients was vastly improved compared with previous experiences. The staff had much greater freedom to manage the therapeutic relationship normally, without the need to intrude in a non-neutral manner. The method is cost effective, enhances psychotherapy progress, improves milieu management, and allows safe weight gain.

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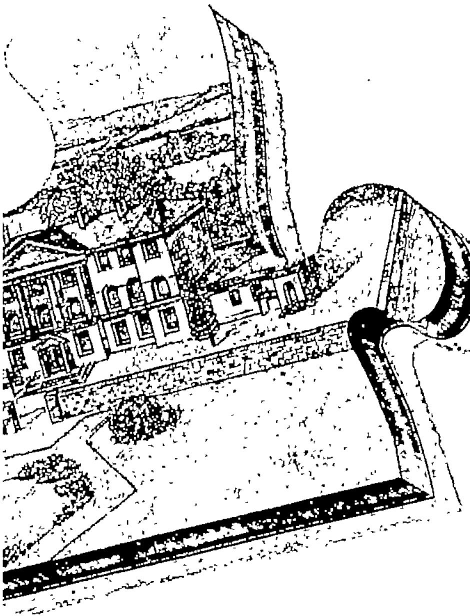

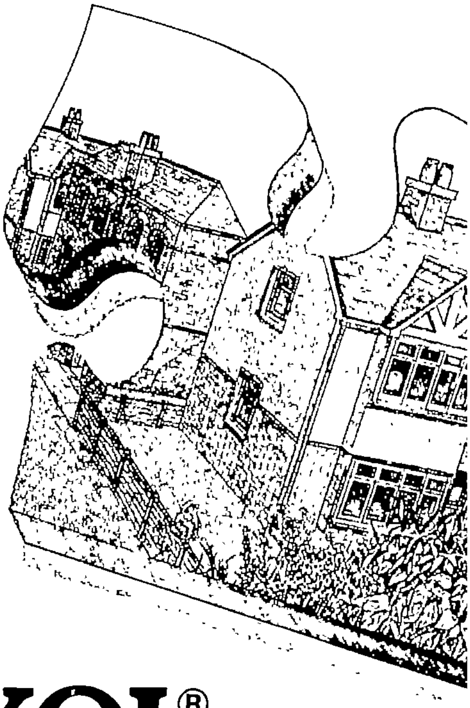

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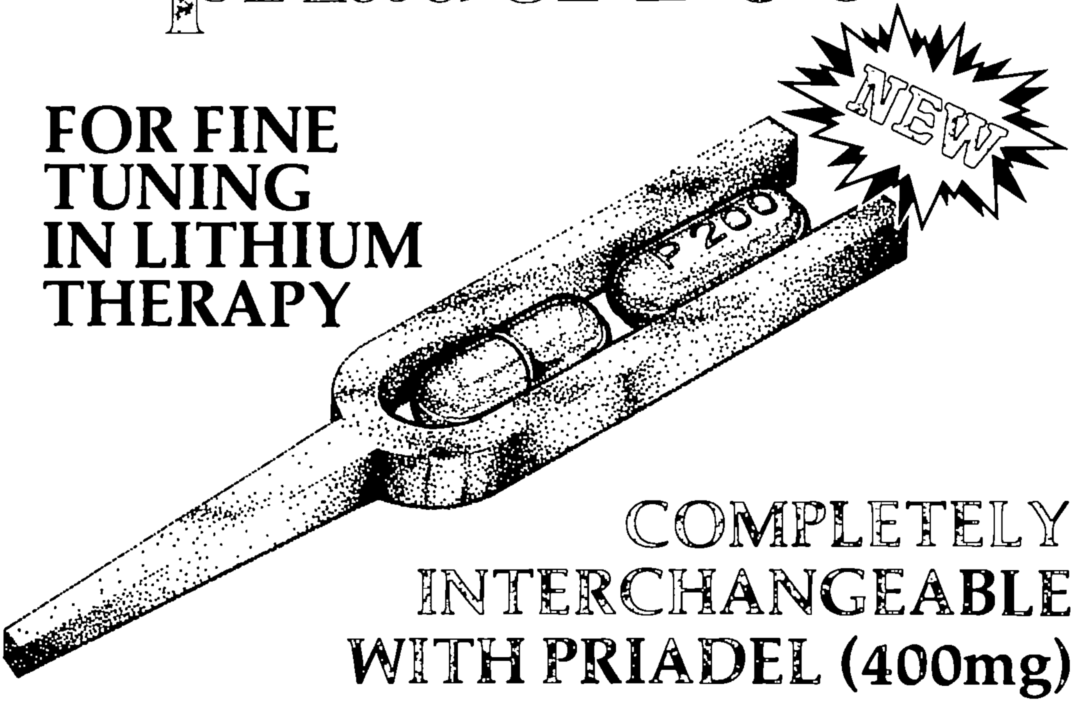

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British Journal of Psychiatry (1987), **151,** 195–199

Hereditary Agoraphobia and Obsessive-Compulsive Behaviour in Relatives of Patients with Gilles de la Tourette's Syndrome

D. E. COMINGS and B. G. COMINGS

We present 11 pedigrees in which a propositus with Gilles de la Tourette's syndrome had first or second-degree relatives with obsessive-compulsive behaviour or agoraphobia with panic attacks, but only partially expressed the TS gene (i.e. had only motor tics or vocal tics, or neither). Of 90 females over the age of 18 presenting with TS, or with motor or vocal tics alone, nine had severe agoraphobia with panic attacks. There may be genetic subtypes of both obsessive-compulsive disorder and agoraphobia with panic attacks that are due to partial expression of the TS gene.

Gilles de la Tourette's syndrome (TS) is an hereditary (Eldridge *et al*, 1977; Nee *et al*, 1980) neuropsychiatric disorder, with the onset between 2 and 15 years of age of multiple motor tics and vocal noises that wax and wane in type and severity. The symptoms can be suppressed for variable periods of time, and must be present for at least one year (American Psychiatric Association, 1980; Shapiro *et al*, 1978; Friedhoff & Chase, 1982). Associated symptoms, not present in all cases, include coprolalia, echolalia, paralalia, attention deficit disorder (ADD) with or without hyperactivity (Comings & Comings, 1984; Cohen *et al*, 1983), learning disorders, exhibitionism (Eldridge *et al*, 1977; Comings & Comings, 1982, 1985), obsessive-compulsive behaviour (Eldridge *et al*, 1977; Nee *et al*, 1980; Shapiro *et al*, 1978; Friedhoff & Chase, 1982; Comings & Comings, 1985; Montgomery *et al*, 1982; Jagger *et al*, 1982), discipline and behaviour problems (Comings & Comings, 1985; Wilson *et al*, 1982), and stuttering. The TS gene can be partially expressed in the form of chronic motor tics alone or vocal tics alone (Eldridge *et al*, 1977; Nee *et al*, 1980; Kidd & Pauls, 1982; Baron *et al*, 1981; Comings *et al*, 1984) and possibly ADD alone (Comings & Comings, 1984). The present study is part of an analysis of 250 cases of TS in which detailed pedigrees were obtained. Complex segregation analysis of these cases for the combined Gilles de la Tourette's syndrome and chronic multiple tic disorder (TS-MT) indicated the involvement of a major gene and excluded polygenic inheritance for a population frequency of the TS-MT trait of less than 1.2% (Comings *et al*, 1984).

We present here some of the pedigrees in this series in which relatives of the TS probands had panic attacks with agoraphobia, or obsessive-compulsive behaviour, but had only motor tics, only vocal tics, or no tics (i.e. did not express the full Gilles de la Tourette syndrome). Since the partial expression of TS, or a family history of TS, can be easily missed unless specifically looked for, we feel these observations may be of value in the further definition of subtypes and causes of agoraphobia and of obsessive-compulsive behaviour.

There is increasing evidence for a significant genetic contribution to panic attacks (Pauls *et al*, 1979 *a*, *b*, 1980; Carey & Gottesman, 1981; Pariser *et al*, 1979; Crowe *et al*, 1983; Harris *et al*, 1983) and panic attacks with agoraphobia (Cohen *et al*, 1951). In a study of 41 probands with panic disorder and 41 controls, Crowe *et al* (1983) found panic disorder or probable panic disorder in 25% of first-degree relatives in the first group, compared with 2% in the controls. In 61% of the families, panic disorder was familial; 11% of the probands also had agoraphobia. These figures are comparable to those of Cohen *et al* (1951), who found that 22% of first-degree relatives were affected and that 67% of the cases were familial. Harris *et al* (1983) found that the frequency of agoraphobia was twice as high in first-degree relatives of probands with agoraphobia than in controls, and concluded that agoraphobia was a familial disorder.

The frequency of obsessive-compulsive disorder is also significantly greater among first-degree relatives of patients with obsessive-compulsive disorder than in the general population (Cohen *et al*, 1951; Kringlen, 1965; Rutter, 1966; Carey, 1978; Cary & Gottesman, 1981). Four identical twin pairs, all concordant for obsessive-compulsive disorder, have been described in which the monozygosity and diagnoses were clearly established (Marks *et al*, 1969; Woodruff & Pitts, 1964; Black, 1974; McGuffin & Mawson, 1980). In two of these the illness occurred at a time when the twins were separated and had little

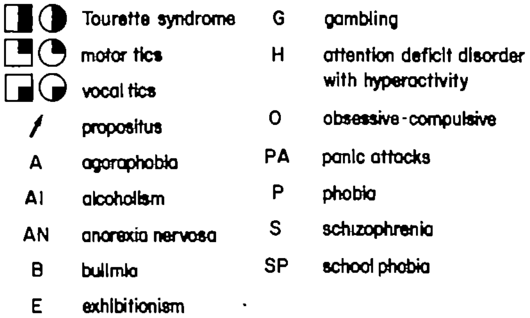

FIG. 1 Key for Figs 2–6

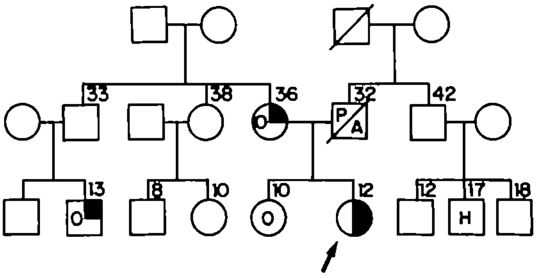

FIG. 2 Pedigree No. 68 (see Fig. 1 for key)

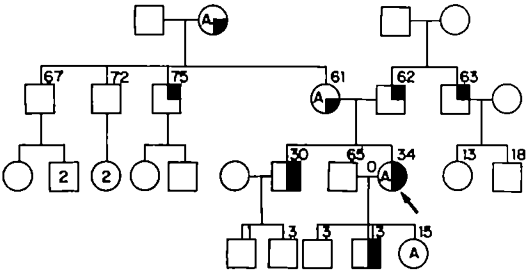

FIG. 3 Pedigree No. 74 (see Fig. 1 for key)

contact with each other (McGuffin & Mawson, 1980). In all four sets, one parent also had an obsessive-compulsive disorder.

Based on an ongoing study of 51 pairs of monozygotic and dizygotic twins, Cary & Gottesman (1981) concluded that "heredity plays a significant part in obsessional disorder and in the phobics who are severe enough to warrant psychiatric care".

Method

The following pedigrees are taken from a series of 250 consecutive cases of Gilles de la Tourette's syndrome, seen in the TS clinic at the City of Hope Medical Center. The clinical, epidemiological, genetic, and other psychiatric aspects of these cases have been presented elsewhere (Comings *et al*, 1984; Comings & Comings, 1984, 1985). All cases conformed to the six criteria of DSM-III, except for nine who did not have vocal tics, but were typical in every other way, and had four or more motor tics. The family pedigrees were based on personal interview (using a structured questionnaire) and examination of every propositus and of every affected first-degree relative. Whenever any other affected relative was geographically available, they were also examined; whenever available, prior medical records were obtained on affected individuals. All patients with agoraphobia had agoraphobia with panic attacks by DSM-III criteria. The obsessive thoughts and behaviour are as described in the case reports.

Case reports

A key to Figs 2–6 is given in Fig. 1.

Pedigree No. 68 (Fig. 2). The ten year-old sister of this propositus had to count to ten many times a day, while taking small steps. The mother had rapid eyeblinking as a child; if she touched one finger, she had to touch them all, and if she touched one tooth with her tongue, she had to touch them all. She had obsessive thoughts in the form of drawing in her mind a square with a cross inside. However, her rules were that she could not lift the pencil from the imaginary paper and could go over a given line only once. Since this is impossible, she dwelled on it constantly, up to 50 times a day. Her 13 year-old niece had hand tics and also took many small steps and counted them. The propositus' father had some panic attacks while driving, a few years before he died of a cerebral aneurysm, but since he had no symptoms of TS he is not included in this series.

Pedigree No. 74 (Fig. 3). The propositus was a 34 year-old female who had an onset of facial tics, grunting, and squeaking at the age of four; mental coprolalia started at the age of ten. At the time of study she had facial and head tics and squeaking vocal noises. She sometimes had to read things a dozen times before it sank in, and complained of racing thoughts and trouble with recent memory. She had obsessive thoughts of sex, violence (killing her mentally retarded son, running her car into a cement wall at 100 mph),kleptomania, and self-destructive acts (cutting off all her hair, cutting herself). She had a responsible job, but had to leave it because of panic attacks while driving; during these she hyperventilated, perspired, had chest pain, felt faint, was tearful, was afraid, and wanted to scream and run away. They came on suddenly, without warning, under a wide range of conditions; this resulted in severe agoraphobia. She had compulsive counting of the number of letters in words, and made mental lists of words according to the number of letters in them. Objects had to be in exactly the right place. Her mother had constant throat clearing and letter reversal in spelling, and experienced panic attacks and agoraphobia for many years; her thoughts also raced, and she was forgetful. Her maternal grandmother also had panic attacks with agoraphobia, and throat clearing. In the six months before the study her daughter, then 15, had panic attacks on elevators, escalators, and while driving, and was increasingly afraid of leaving the house.

Pedigree No. 75 (Fig. 4). The nine year-old sister of this propositus had intermittent facial tics and did numerous

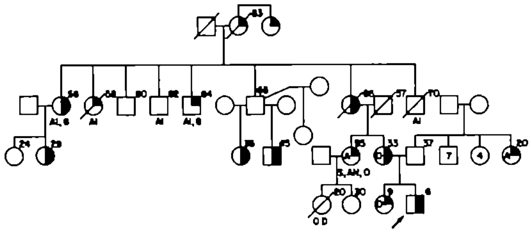

FIG. 4 Pedigree No. 75 (see Fig. 1 for key)

things in a compulsive manner such as touching her head to the ground, or putting both feet together, touching her head, and stiffening up. The father had a 20 year-old sister who had eyeblinding from ages 9 to 12, and who had recently had panic attacks and was afraid to leave the house. The mother was hyperactive as a child, had mild motor and vocal tics from the age of 6 to 15, and compulsively smelt objects. Her 55 year-old sister had multiple psychiatric problems. She was admitted to the hospital on eight occasions, and received chlorpromazine and ECT for schizophrenia. Although medical records were not available, ten of our TS proposita were incorrectly diagnosed as schizophrenic, and she may have been so as well. She had anorexia nervosa as a teenager, has been agoraphobic for the past 20 years, and was obsessive about repetitive touching, smelling, and sniffing. The maternal grandmother had a life-long history of throat clearing, coprolalia, spitting, and facial tics. The remainder of the family history is shown in Fig. 4, and includes four other individuals with TS, four with motor tics, five alcoholics, and two compulsive gamblers.

Pedigree No 76 (Fig. 5). The mother of this 14 year-old boy with TS, ADD, and exhibitionism had vocal noises and facial tics as a child. She had compulsive behaviour, consisting of vacuuming the house many times a day, every day. She had had severe agoraphobia with panic attacks for five years, and was unable to come to the clinic unless someone brought her. She took diazapam to relieve her anxiety about being on the motorway.

Pedigree No 184 (Fig. 6). The father of a 16 year-old boy with TS and ADD had a "nervous breakdown" at the age of 40; he then began having panic attacks when driving, and developed severe agoraphobia. He did not leave the house for years and still has difficulty with panic attacks when he goes out. He tried to keep his business going for five years by phone, but it eventually collapsed and he is now on a total disability pension. He was also hyperactive as a child, but had no tics or vocal noises. The 57 year-old sister developed severe agoraphobia with panic attacks at age 32, and still has these problems. She had three children, including a 26 year-old son who had such severe school phobia that he required home tutors.

Pedigree No. 192. The mother of a 15 year-old boy with TS had grunting vocal noises, rapid eyeblinking, and facial tics for several years as a child. At the time of study she had panic attacks and felt paralysed when driving on the motorway, and had to have others drive for her. She had a 17 year-old brother with head tics, eyeblinking, vocal noises, coprolalia, learning disorders, short temper, and a paranoid personality. The parents are divorced. The father had a head tic as a child.

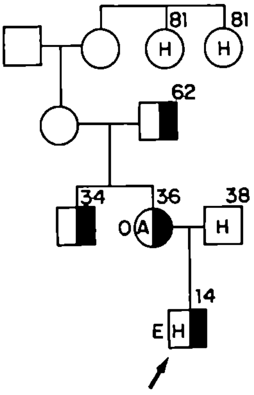

FIG. 5 Pedigree No. 76 (see Fig. 1 for key)

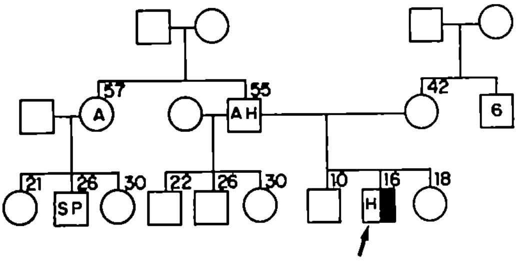

FIG. 6 Pedigree No. 184 (see Fig. 1 for key)

Pedigree No. 316. The mother of identical twin girls with TS had severe mental coprolalia for years, and was afraid she would say the word 'fuck' out loud. She had a compulsion to fold and unfold the pages of books and could sit doing this for hours; if she went somewhere without a book she felt uncomfortable until she had one in her hands again. She had an obsession with dates, fixing on the day's date and saying it over and over in her mind, hundreds of times; she combined the compulsion and obsession by carrying around a diary. She had numerous panic attacks over many years, and felt tense and nervous unless at home; this was slowly getting worse. There had been problems with stuttering since childhood.

Pedigree No. A1. A 40 year-old female was referred because of familial agoraphobia. She had an onset of panic attacks with associated agoraphobia at the age of 22 and has had attacks almost daily ever since, despite years of varied types of therapy. In the past year she has obtained significant improvement of symptoms with medication (alprazolam). Her mother had suffered from agoraphobia with panic attacks since the age of 24, and the maternal grandmother and grandaunt also had agoraphobia. The propositus had a 14 year-old son with an onset of eyeblinking, facial grimacing, head jerking, and hyperactivity at two years of age; he later developed ritualistic licking of his shoulders and feet, paralalia, numerous phobias, and obsessive-compulsive behaviour. Except for the absence of vocal noises, he was a classic, undiagnosed case of TS. In this family, the TS was ascertained through a patient with agoraphobia, rather than vice versa.

Discussion

Many studies have shown the presence of an obsessive-compulsive disorder in individuals with TS (Eldridge *et al*, 1977; Nee *et al*, 1980; Shapiro *et al*, 1978; Cohen *et al*, 1983; Comings & Comings, 1985; Montgomery *et al*, 1982; Jagger *et al*, 1982). Although Shapiro *et al* did not find evidence of a greater frequency of obsessive-compulsive behaviour in TS patients than in a general psychiatric clinic, its presence in up to two-thirds of TS patients in some series suggests a significant increase compared with the general population. The pedigrees studied here suggest not only that may TS patients themselves manifest an obsessive-compulsive disorder, but that their first and second-degree relatives may also present with such behaviour in the absence of full-blown TS. We believe this may constitute a genetic subtype of obsessive-compulsive disorder, due to the incomplete expression of a TS gene. Montgomery *et al* 1982 also reported (in a selected population of TS patients) the presence of obsessive-compulsive disorder and panic attacks, without tics or vocal noises, in first-degree relatives of patients with TS. The presence of an obsessive-compulsive disorder in first-degree relatives with either very mild motor or vocal tics, or no tics, is important in that it tends to negate the theory that this behaviour in TS patients is simply a psychological reaction to the presence of tics and vocal noises (Shapiro *et al*, 1972).

In this series of families there were 463 individuals manifesting the TS-MT gene by the presence of chronic motor tics, vocal noises, or both; nine of these (2.0%) had agoraphobia. (There were nine additional cases–five females and four males – in other relatives, but without tics or vocal noises; these are excluded from the 463 cases since there is no direct evidence that they also had the TS gene, although in six of them other members in that branch of the family showed complete or partial manifestation of the TS gene). Many of the 463 cases were less than 18 years of age. Agoraphobia usually has an onset between 18 and 35 years of age and affects primarily women, with a sex ratio of one male to nine females (Burns & Thorpe, 1977; Marks & Hearst, 1970; Mathews *et al*, 1981). If all patients younger than 18 years are excluded, there were 231 patients manifesting the TS-MT gene with motor or vocal tics or both. Of these, 141 were men. None of the men had agoraphobia (although some had panic attacks without agoraphobia); of the ninety women, nine had agoraphobia with panic attacks.

While the frequency of agoraphobia in females manifesting with TS-MT appears to be greater than that of the general population, the degree to which it is greater depends on which frequency figures are used for the general population. If the figure of one per 1000 of agoraphobia among women sufficiently severe to cause disability (Mathews *et al*, 1981; Agras *et al*, 1969; Hollingshead & Redlich, 1958; Lemkau *et al*, 1942) is used, agoraphobia is 100 times more common in women with TS-MT. If a higher estimate of 1.8% of women having agoraphobia-panic attacks (Uhlenhuth *et al*, 1983) is used, the rate observed here is only about five times that in the general population. The highest frequency we could find in any control series was 4.2% among relatives of 20 surgical patients and hospital employees, reported by Harris *et al* (1983).

There are several possible explanations of these data. Firstly, females carrying the TS gene, whether they have tics and vocal noises or not, might have an increased susceptibility to agoraphobia. Secondly, if females carrying a TS gene also inherit a gene (or genes) for panic attacks, they might be more likely to have panic attacks and agoraphobia than if they only had the gene for panic attacks. Thirdly, the TS gene and agoraphobia, whatever its cause, might be unrelated, and agoraphobia might be no more common in individuals with the TS gene than the general population. Our bias is towards the first explanation, but these issues will probably only be resolved by the development of genetic markers for the specific disorders.

We believe these observations are important for several reasons. Firstly, in most of these patients with incomplete expression of TS, the motor or vocal tics may no longer be present, may be relatively mild, or may be easily suppressed during an out-patient visit. As such, they are easily missed unless the patient is specifically questioned about them. Secondly, patients with agoraphobia or obsessive-compulsive behaviour should be questioned about the presence in relatives of motor or vocal tics, or both, as well as about agoraphobia and obsessive-compulsive behaviour.

Both the literature and the above observations suggest that agoraphobia and obsessive-compulsive behaviour may be subdivided into the following categories: (a) sporadic (no family history); (b) familial (positive family history); and (c) cases possibly due to a TS gene (positive history of motor or vocal tics in the patient or family members). This subdivision may have important implications about the best form of therapy for that individual.

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Depressed Mood After Stroke

A Community Study of its Frequency

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A community study on stroke registered 976 patients. Over 60% of survivors at three weeks, six months and 12 months after the stroke were assessed for depressed mood, using the Wakefield self-assessment depression inventory, and on other functional and social activities. At each point, 25–30% of those assessed were depressed; over 50% of patients depressed at three weeks remained so at one year. Factors associated with depression included loss of functional independence, a low level of other activities, a low reasoning ability, and being female and living with someone; path analysis showed that most depression was not explained by these factors. Few depressed patients at six months were on antidepressant medication.

The physical disability caused by stroke is obvious but, despite the attention drawn to the "hidden barriers to recovery" (Adams, 1974), little attention has been paid to the emotional aspects of stroke. Reasons may include the difficulty in assessing emotion accurately, and the feeling that it is only to be expected that stroke patients will be depressed. It is only in the past few years that the frequency of depression after stroke has been investigated. One group have found depression to be present in about 30% of patients (Robinson & Price, 1982; Robinson *et al*, 1983); they have suggested that it may be specifically associated with localised damage near the left frontal lobe (Robinson *et al*, 1984*a*) and that treatment with nortriptyline may relieve it (Lipsey & Robinson, 1984). A second group also found depression in about 30% of patients, and related this to their loss of social activities (Feibel & Springer, 1982).

The pioneering work of these two groups of investigators needs to be replicated. We have investigated the frequency of depressed affect after stroke in a community survey. In this paper we examine the following questions:

(a) How prevalent is depression at various times after stroke?

(b) What is the natural history of depression after stroke?

(c) What factors relate to the presence of depression at any time?

Method

Over 28 months, a total of 976 acute cases were registered in a community survey of stroke-related disability, based on a population of 215 000 patients registered with 96 general practitioners in Bristol. Of these, 249 patients (26%) were not admitted to hospital over the first six months after the stroke. Patients were identified by direct notification from anyone involved in stroke care (e.g. family or hospital doctors, nurses, therapists) and also by obtaining the records of all patients known to have been discharged from any hospital in the county of Avon with a primary or secondary diagnosis of stroke (ICD-9 classifications 430–438; World Health Organisation, 1978). A clinical diagnosis was used for this study, based upon the WHO definition (Aho *et al*, 1980): "Rapidly developed clinical signs of focal (or global) disturbance of cerebral function, lasting more than 24 hours or leading to death, with no apparent cause other than of vascular origin". The diagnosis was confirmed by a neurologist, by personal examination in 713 (73%) cases or by reading hospital notes if a patient had died or was registered late.

Each patient was seen as soon as possible after the stroke, at 3 weeks, at 6–7 months, and at 12–13 months; at each point, information was recorded on many aspects of the neurological, functional, emotional, and social status.

Depression was assessed using the Wakefield self-assessment depression inventory (Snaith *et al*, 1971), which has 12 questions giving a score between 0 and 36; although not validated in the elderly or in stroke, it was derived from the Zung scale, which has been validated in stroke research (Robinson & Price, 1982). In the original validation, a score of 15 or more was found to be the best cut-off to identify those with depression. We have classified those scoring 15–18 as exhibiting 'probable depression' and 19–36 as 'definitely depressed', because there is some evidence that older people tend to have higher scores (Snaith *et al*, 1971). The Hopelessness scale (Beck *et al*, 1974), comprising 20 statements for the patient to agree or disagree with, was also used, but many found it difficult to understand, and so the results were only used to help validate the Wakefield scale. Since the Wakefield inventory was not developed as an accurate diagnostic instrument, but rather as a way of assessing the severity of depression, our results concern the

TABLE I
Characteristics of 976 patients studied

		Time	
	3 weeks	*6 months*	*12 months*
Died: *n*	350	432	484
Notified late/lost to follow-up: *n*	57	69	58
Aphasic or confused: *n*	190	98	86
Assessed for depression: *n*	379	377	348
Average age: years			
Mean	69.9	69.7	69.3
s.d.	9.6	9.8	10.3
Male: *n*	203 (54%)	209 (55%)	180 (52%)
Living alone: *n*	87 (23%)	88 (23%)	92 (26%)
Not admitted in 6 months after stroke: *n*	159 (42%)	165 (44%)	151 (43%)
Side of weakness: *n*			
Right	170 (45%)	174 (46%)	160 (46%)
Left	178 (47%)	166 (44%)	152 (44%)
Both or neither	31 (8%)	37 (10%)	36 (10%)
Disability–Barthel scores: *n*			
0–14 (severe/moderate)	99 (26%)	48 (13%)	45 (13%)
15–19 (mild)	123 (32%)	128 (34%)	117 (34%)
20 (no disability)	157 (42%)	201 (53%)	186 (53%)
Depression–Wakefield scores			
0–14 (not depressed)	252 (67%)	257 (68%)	240 (69%)
15–18 (probably depressed)	43 (11%)	46 (12%)	45 (13%)
19–36 (depressed)	84 (22%)	74 (20%)	63 (18%)

extent of any feeling of depression or sadness, rather than clinically defined 'depression'.

Social function was assessed using the Frenchay Activities Index (FAI) (Holbrook & Skilbeck, 1983); this records the frequency with which a patient has performed various activities over the preceding months, and gives a score ranging from 0 (no participation) to 45 (full participation in all 15 items). Physical function was measured using the Barthel Activities of Daily Living (ADL) scale (Granger *et al*, 1979). This scale measures ten self-care items, and gives a score of 0–20. A score of 0–9 indicates 'severe' disability; 10–14, moderate disability; 15–19, 'mild' disability; and 20, functional independence. Each patient's reasoning ability (IQ) after the stroke was measured using Raven's coloured progressive matrices (Raven, 1965): this is a non-verbal measure with normative data for the elderly. Motor loss was assessed using the Motricity index (Demeurisse *et al*, 1980), which gives a score ranging from 0 (complete paralysis) to 100 (normal strength). Two self-assessment procedures were also used. Firstly using a visual analogue scale 150 mm long, patients were asked to indicate the degree of recovery of independence they had achieved: the end-points were "the worst you were with your stroke" and "as you were before your stroke". Secondly, using a five-point scale, patients were asked to indicate their depression relative to before their stroke as much less, less, the same, more, or much more (scored 1–5).

Results

Details on the patients seen and time-point assessed are shown in Table I. Patients with a wide range of disabilities and ages were included. The number of patients not assessed due to late notification, and because of communication problems (either aphasia or confusion) is also given. Only ten patients refused to answer the questions. The high proportion of patients not admitted reflects the fact that many of those admitted were severely affected, and died within a short time. The prevalence of a depressed affect at three weeks, six months and one year is also shown: approximately 20% of patients were 'definitely depressed' at any point, and a further 10% 'probably depressed'. The Hopelessness scale was completed by 317 patients at one year: their average score was 5.64 (s.d. 4.02), ranging from 0 to 19, with only 26 scoring 13 or more.

Recovery from depression did occur. Of the 127 scoring 15 or more on the Wakefield scale at three weeks, 26 had died by six months, and a total of 46 had died by one year. At six months, 33 (33%) of the surviving 101 patients scored 14 or less. At one year, 28 (35%) of the surviving 81 patients were not depressed.

To determine how much variability occurred with time, details of the 255 patients with assessments at all three points were analysed: 175 (69%) scored 0–14 at three weeks and 80 (31%) scored more than 15; 133 (52%) were consistently

TABLE II

Some correlation coefficients with Wakefield scores (all significant at $P<0.01$)

	n	*Coefficient*
At 3 weeks		
Barthel ADL score	379	−0.329
IQ (Raven's matrices)	331	−0.257
Motricity score	342	−0.324
At 6 months		
Barthel ADL score	377	−0.219
IQ (Raven's matrices)	345	−0.146
Current social activities (FAI score)	375	−0.353
Loss of social activities (FAI score)	357	+0.229
Carer's depression (Wakefield)	192	+0.345
Self-image of recovery (line scale)	293	−0.430
Self-image of mood change	359	+0.539
At 1 year		
Barthel ADL score	348	−0.247
IQ (Raven's matrices)	330	−0.203
Current social activities (FAI score)	348	−0.371
Carer's depression (Wakefield)	200	+0.253
Other correlations		
Wakefield 3 week and 6 month scores	312	+0.690
Wakefield 3 week and 1 year scores	261	+0.630
Wakefield 6 month and 1 year scores	299	+0.750
Wakefield 6 month and Barthel 3 week scores	365	−0.165

'not depressed', while 44 (17%) were consistently 'depressed' at all three points. Thirty-nine (15%) depressed patients had recovered by one year, but 14 (5%) who were not initially depressed became so by six months, while 25 (10%) became depressed by one year; 19 (7%) patients fluctuated between the three assessment points.

One can conclude that approximately half of the patients who were depressed at one year had been depressed since soon after their stroke. Also, a quarter of those not initially depressed became so six months to one year later.

To elucidate the factors associated with depression, simple correlation coefficients were calculated between a patient's Wakefield score and his score on other scales (Table II). A few patients had to be excluded, as data for them were not available, but most analyses contained over 300 patients. All coefficients are statistically significant ($P<0.01$), with the highest associative factors of items not measuring mood being self-image of recovery ($r=-0.430$) and FAI score at one year ($r=-0.371$). The negative sign indicates that as these scores increase, so less is scored on the Wakefield Depression Inventory (i.e. showing a less depressed mood). Similarly, as functional and intellectual performance improve, showing higher scores, so Wakefield score decreases. However, these coefficients are not high enough to imply necessarily clinical significance; e.g. the Raven's progressive matrices score at six months only accounts for 2% of the variance seen, despite its statistically significant coefficient of $r=-0.146$. Furthermore, the existance of a significant correlation cannot be assumed to indicate a causal relationship.

The strongest correlations are those between measures of mood, either using different instruments (e.g. the Hopelessness scale) or at different times. Wakefield scores are also strongly correlated with patients' self-assessment of mood change from before their stroke.

At three weeks after the stroke there was a significant association ($\chi^2=7.56$, $P<0.05$) between depression and side of stroke symptoms: 75 (42%) of the 178 patients with left-sided symptoms were probably or certainly depressed, compared with 48 (28%) of the 170 patients with right-sided symptoms. Their mean scores were significantly different (left $=13.1$, s.d. $=8.6$; right $=11.2$, s.d. $=7.8$; $t=2.17$; $P<0.01$). The mean score (8.3, s.d. $=7.1$) of the 31 patients with no unilateral symptoms or signs (e.g. brief or mild aphasia) was significantly lower than in the patients with left-sided symptoms ($t=2.94$, $P<0.01$). There was no significant association between level of depression and laterality of stroke at six months or one year. Patients with aphasia or other communication problems sufficient to preclude assessment – 86 (20%) at one year – were excluded. Most of these patients had right-sided weakness, which complicates interpretation of results.

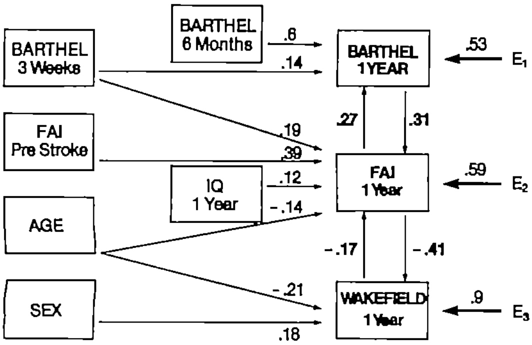

FIG. 1 Path diagram showing influences on depression, social activities and ADL in 348 patients seen one year after stroke.

Women appeared to be significantly more depressed at all three assessment points ($P<0.01$). The number of men/women scoring 19 or more (certain depression) was 38/46 (19%/26%) at three weeks; 29/45 (14%/27%) at six months, and 28/35 (15%/21%) at one year. This sex difference was not simply because more women lived alone: at one year, the mean score of the 45 women living alone was 10.3 (s.d. = 7.2), identical to that of the 27 men living alone. In fact, the difference in depression was most marked in those living with someone: the 180 men had a mean score of 9.7 (s.d. = 7.1), as against 12.0 (s.d. = 7.6) for the 121 women ($t=2.44$, $P<0.02$). The remaining patients were in institutional care. Overall, there was no significant difference in average depression scores between the patients living alone and those living with someone.

Few patients were on treatment for depression six months after the stroke. Information on drugs being taken was available for 274 of the patients assessed, but this information had not been collected for those seen in the early part of the study. Only 23 (8%) were on antidepressant medication; their average Wakefield score was 14.9 (s.d. = 8.0), which was significantly higher ($t=2.35$; $P<0.01$) than the remaining patients (mean = 10.8; s.d. = 8.0). Eleven (48%) had scores of 15 or more, compared with 76 (30%) of the remaining patients, 50 of whom had scores of 19 or more. It seems that most 'depressed' patients were not being treated for depression.

Path analysis (Blalock, 1971) was used to develop a model linking the various factors thought to influence depression: as results were very similar after both six months and one year, only the latter are illustrated in Fig. 1. This technique allows a visual appreciation of the associations between several variables simultaneously. Repeated multiple regression analyses identify the strength of interactions between pairs of variables, while controlling for the effects of the other variables. Path coefficients (i.e. standardised regression coefficients) represent the relative strength of each association.

Figure 1 illustrates the influences on a patient's mood (Wakefield), social function (FAI), and functional ability (Barthel ADL) at one year after the stroke. The arrows to these variables, together with corresponding path coefficients, indicate the relative (but not absolute) strength of influence of static or preceding factors. For example, 'sex' indicates that, when controlling for all other factors, sex has a relatively small influence on depression.

The path diagram further illustrates how a significant correlation, for example that between ADL score and Wakefield score ($r=0.247$), can be 'lost' when controlling for other variables. That is, the path coefficient between ADL and Wakefield scores did not reach a significant level (zero path coefficient), and was therefore ommitted from the diagram. The significant correlation can now be explained by means of an intervening variable – FAI – which may act as a mediator between ADL and depression.

Where reciprocal statistical influences exist between outcome variables, these have been indicated; it is difficult, however, to envisage how social functioning might influence ADL function, and an unidentified intervening variable must be assumed. The consequence of depression in terms of fewer activities (lower FAI score) and vice versa is, on the other hand, more readily understood.

Finally, in understanding the three outcome factors, much of the variance has not been accounted for in terms of the variables measured, so that unmeasured extraneous variables must account for the remainder; 28% of the variance in Barthel ADL is unaccounted for, and extraneous variables are represented by E_1 in the diagram with a path coefficient of 0.53. Similarly, 25% of the variance in FAI (E_2) and 40% of the variance in Wakefield (E_3) are unaccounted for.

Discussion

This analysis confirms that a depressed mood is common after stroke and shows that, although other stroke-induced losses do relate to depression, most depression cannot be explained simply by reference to physical or social disability. One possible explanation is that pre-existing personal or social characteristics may have an important influence on the presence of depression after stroke. It also seems that few depressed patients receive specific medical treatment, despite evidence that tricyclic antidepressants may be effective (Lipsey & Robinson, 1984).

This study included all patients admitted to hospital, but might have missed some of those treated at home; however, our overall incidence of 1.90/1000 per year is not very different from other surveys (aho *et al*, 1980), suggesting relatively complete notification. At one year after stroke, 88% of surviving patients were seen, but 20% of these could not be assessed due to communication problems. Although 'objective' biochemical methods of diagnosing depression have been proposed (Carroll *et al*, 1981), their sensitivity and specificity are still debated (Holden, 1983), while their use in a community study would raise many problems. However, as a wide spectrum of patients was included, with no obvious selection according to stroke severity or hospital admission, the sample should be representative of patients suffering stroke but with no communication problems.

The validity of the Wakefield depression scale needs discussion. In this study, the Wakefield score was strongly related to two other measures of depression–the Hopelessness score and the patient's own assessment of his mood change–and it was also related to the prescription of antidepressant medication. We conclude that the Wakefield did measure depressed mood in our patients, although this is not to say that patients with a depressed mood necessarily suffered a depressive illness.

Patients were categorised as 'probably' or 'certainly' depressed because of uncertainty as to cut-off values with an elderly population. Furthermore, it is possible that many 'normal' elderly people are 'depressed': the average Hopelessness score of 5.64 in our study is not significantly different from that recorded in 148 normal people aged over 50 in Dublin (Greene, 1981). Depression may have been present in many patients before their stroke. However, one survey (Bergmann, 1982) of elderly people suggested that 'depressive neurosis' was present in only 3.8% of the 360 tested and 'depressive illness' in 0.6% (all women). These figures are much lower than those seen in our patients with stroke.

Our results confirm the frequency of depression found in other large-scale studies (Robinson & Price, 1982; Robinson *et al*, 1983, 1984*b*; Feibel & Springer, 1982): approximately one-third of patients were depressed at any time after the stroke. We found that the severity of the stroke, as measured by the Barthel ADL scale, did have a direct correlation with depression. One other study (Feibel *et al*, 1979,) noted such a correlation, although the strength of their correlation increased with time, in contrast to the fall we found; other studies (Robinson & Price, 1982; Feibel & Springer, 1982) failed to find any correlation with ADL loss. Two points need to be made concerning this correlation. Firstly, when present, it is relatively small, accounting for 5–15% of all variance; this is probably not clinically important. Secondly, path analysis suggests that non-ADL activities are intervening factors in the relationship between ADL function and depression. We thus confirm the relationship between depression and low social functioning, as previously found (Feibel & Springer, 1982; Feibel *et al*, 1979).

It has not previously been reported that women are more depressed; this difference applied only to those living with someone. Social isolation did not appear to be associated with depression. The other major identified influence on depression at one year was depression seen earlier in the recovery process. Over half of those depressed at three weeks or six months were still depressed at one year. This is a little less than found in another follow-up study, where 10 out of 13 initially depressed patients were still depressed at six months (Robinson *et al*, 1984*b*).

Previous reports on small numbers of selected patients (Robinson *et al*, 1984*a*) have suggested that there is a higher rate of depression in those with lesions in the left frontal region, but the same workers failed to confirm this finding when studying larger groups of patients (Robinson *et al*, 1983). We did not have any information concerning the anatomical location of the stroke, but we have failed to confirm any association between right-sided weakness and depression. Indeed, in the first few weeks those with left-sided weakness were more depressed, although this may be due to exclusion of patients with aphasia. We conclude that even if lesions near the left frontal pole are associated with an increased risk of depression, this cause of depression is overshadowed by other factors in the majority of cases.

This study confirms previous observations that most depression is untreated (Robinson & Price, 1982; Feibel *et al*, 1979,) and also shows that most depression cannot be explained simply as a natural response to physical disability. Indeed, much of the variance in depression is unaccounted for by any of the variables studied, suggesting that other personal or social factors may be important. This study suggests that attempts to reduce depression through improving a patient's social or ADL functioning may not be very successful, although this is not to deny the important association between depression and both low social and ADL functioning.

In conclusion, this study found that a depressed mood was present in about one-quarter of surviving and assessable patients at any time up to one year after stroke. Less than half of those depressed in the early stages were free of depression at one year. The presence of depression was associated with the degree of physical disability, loss of social function, and being female living with someone, but these and other factors measured do not account for much of the depression seen. The path diagram derived from these data gives a broad outline, in visual form, of some important inter-relationships, but will need modification in the light of future research.

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Correlation between the 37-Item Mental Test Score and Abbreviated 10-Item Mental Test Score by Psychogeriatric Day Patients

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Groups of organically and functionally mentally ill psychogeriatric day patients were assessed by the 37-item Roth-Hopkins test and the abbreviated ten-item Roth-Hopkins test. The scores correlated highly, correlation being equally good for both diagnostic groups. Among the functionally ill and the mild organically ill, scores improved between first and second test. The quicker, better tolerated ten-item test is a useful test of mental impairment for psychogeriatric patients in the community, as well as in institutional care.

A mental test of orientation and information was devised by Roth & Hopkins in 1952, and shown to distinguish between diagnostic groups of organically impaired and functionally disordered elderly psychiatric patients (Roth & Hopkins, 1953; Hopkins & Roth, 1953). It was modified by Blessed *et al* (1968), who added tests of memory and concentration previously shown by Shapiro *et al* (1956) to distinguish between similar categories of patients. This 37-item Roth–Hopkins mental test score (MTS), when applied to elderly psychiatric patients who were followed to post-mortem, was shown to correlate significantly with quantified measures of cortical degeneration and with levels of choline acetyl transferase in the neo-cortex (Blessed *et al*, 1968; Perry *et al*, 1978).

Hodkinson (1972) and Qureshi & Hodkinson (1974) developed an abbreviated form of MTS which condensed the most discriminating aspects of the MTS to ten items (AMTS). They showed with patients recently admitted to hospital, in long-stay wards, or in residential homes, that the AMTS was quicker to perform, better tolerated by patients, and correlated highly significantly with both the MTS and the Modified Tooting Bec questionnaire—a further test of cognitive function used extensively in the assessment of memory and orientation in elderly patients (Denham & Jeffreys, 1972). Qureshi & Hodkinson administered the AMTS twice to 72 patients, and found no evidence that patients' performance improved with practice.

This study was carried out to select a suitable test for rapid screening of elderly people living in their own homes for signs of dementia. In seeking a test, the authors chose the AMTS because it contains only items from the MTS, which has the advantage—rare among clinical tests of cognitive function—of having been validated against neuropathological and neurochemical changes in the brain. However, in the previous studies, the AMTS had been administered to institutionalised patients only, and it was felt necessary to show that inter-test correlation was equally high for patients living in their own homes.

Method

Fifty-two patients living in the community but currently attending a psychogeriatric day hospital for treatment were selected for testing if they satisfied the following criteria:

1. They were willing to participate.
2. They had completed a full diagnostic assessment procedure.
3. They were attending at least once per week, so that both tests could be administered within seven days of each other.
4. They could hear well enough to participate in testing, and speak sufficiently clearly for their answers to be recorded.
5. Their psychiatric disorders could be reliably classified by the International Classification of Diseases (ICD-9) criteria (WHO, 1978), on the basis of the history of the illness, previous psychiatric and family history, and in certain patients, electro-encephalographic and radiographic examination, more complex psychometry, etc. Those with ambiguous or mixed diagnoses were excluded.

Patients were allocated at random to two groups: one group had the MTS (Appendix I) followed by the AMTS (Appendix II) at their next attendance. The other group received the AMTS first and the MTS subsequently. Possible improvement of scores over time was looked for by assigning a score (MTSA) derived from the performance of the patient on the ten items of the AMTS as they occur in the 37-item MTS. A comparison of the first score on AMTS or MTSA was then made with the second score.

All tests were given by one interviewer, under identical conditions.

Results

All 52 patients completed the study; there were 13 men and 39 women, with a mean age of 75.75 (±6.82) years. There was no significant difference by sex or age between the two groups. The diagnoses of the patients are shown in Table I.

Twenty-seven patients had chronic organic brain syndrome (dementia), while 25 suffered from functional psychiatric illnesses. Comparison of test performances for these two major sub-groups is shown in Table II.

The functionally ill not only scored better than the organically ill, but were also consistently better, with much less individual variation. This is revealed in the histograms (Figures 1 & 2).

The organically ill seemed to fall into two groups of roughly equal size: 13 obtained scores equal to or greater than 75% correct answers, while the remaining 14 scored 70% or less correct. The former are referred to as 'mildly impaired' and the latter as 'moderately to severely impaired'. The organically ill group as a whole scored significantly worse than the functionally ill on both the MTS and the AMTS ($P<0.001$ for both tests).

The scores that patients attained on the AMTS correlated highly significantly with those of the MTS and the MTSA (Table III).

These correlations are comparable to those found by Qureshi & Hodkinson (1974), which ranged from +0.87 to +0.96.

By Sign Test analysis, it was found that all diagnostic groups except the moderately to severely organically ill improved from the first to the second test, reaching varying levels of significance (Table IV). This was unaffected by the period between tests (two to seven days).

TABLE I
Diagnoses of the patients

Diagnosis	*ICD 9*	*No*
Organic group	290	27
Senile dementia	290.0	20
Pre-senile dementia	290.1	1
Arteriosclerotic dementia	290.4	3
Other/unspecified	290.8–9	3
Functional group	295–8/300	25
Depression	296.1/298.0/300.4	20
Hypomania	296.4	1
Paraphrenia	295.3	1
Schizo-affective disorder	295.7	3
Total		52

TABLE II
The differences between the scores of two groups of patients

Test	*Diagnostic group* *Organic*	 *Functional*
MTS (out of 37)	22.90	34.48*
AMTS (out of 10)	6.37	9.50*

Mann–Whitney Test, $*P<0.001$

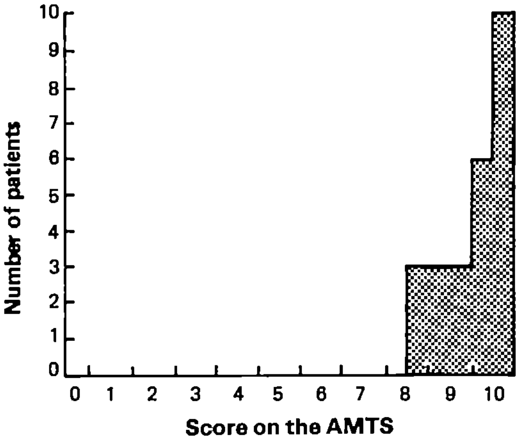

FIG. 1. Distribution of the scores of functionally ill patients

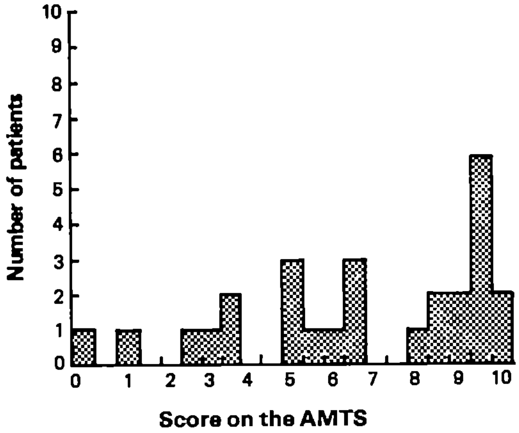

FIG. 2. Distribution of the scores of organically ill patients

TABLE III

Correlation between the tests for the various groups of patients

Tests under comparison	*Diagnostic groups* Organic group	Functional group	All patients
MTS AMTS	+0.91*	+0.93*	+0.92*
AMTS MTSA	+0.95*	+0.96*	+0.96*
MTS MTSA	+0.95*	+0.96*	+0.96*

Pearson's Correlation Coefficient (r)
*$P<0.001$

TABLE IV

Analysis of changes between the first and second test scores in the various groups

Diagnosis	*Score change* improved	same	worse
Organic psychosis (mild)	7	5	1*
Organic psychosis (severe)	8	2	4
Organic psychosis (all)	15	7	5*
Functional	12	12	1*
All patients	27	19	6†

Sign test analysis
*$P<0.05$, †$P<0.01$

Discussion

The results of this study agree with those of Qureshi & Hodkinson, that the shorter, quicker AMTS correlates well with the MTS. No disadvantages of the AMTS came to light during the performance of the study; it was still possible to draw a more subjective appraisal of the patients in the quality of their answers. It was also felt subjectively that patients tolerated the shorter test better.

The 'assigned' MTSA correlated well with both the MTS and the AMTS and it seems valid to assign a score in this way. The effect of improvement in score shown by the functionally ill and the mildly organically ill was independent of age or sex, and is probably a result of a learning process, since in a period of less than seven days, it is unlikely that any improvement due to treatment had occurred.

This finding is at variance with that of Qureshi & Hodkinson, which is probably related to a greater number of their cohort being of the moderately to severely organically impaired category.

Despite the considerable differences in mean scores, on both tests, between functionally and organically impaired patients there is a considerable overlap between scores obtained by the functional group and by the mildly impaired of the organic group. This may reflect a group of patients with early dementia at the upper end of the range of cognitive ability who would progressively worsen. It may also be due to a group of organically ill patients whose cognitions are relatively spared and have been referred for other reasons. Under-education may play a part, but would be expected to affect all groups of patients equally (Bergmann *et al,* 1971). Whatever the reasons for this overlap, it appears that the mildly impaired have a capacity for improvement which is not demonstrable in the severely impaired.

The fact that improvement is possible supports the use of cognitive rehabilitation methods such as Reality Orientation Treatment for the less severely impaired (Woods, 1979), but also suggests that serial testing should be used with caution in mildly organically and functionally ill patients.

Thus, as in patients in institutional care, for a psychogeriatric day-patient who has been diagnosed on the clinical picture as suffering from an organic brain syndrome, the AMTS is a useful, commendably brief interview for quantifying the severity of the cognitive impairment, comparable to the MTS.

Mental tests alone cannot replace careful clinical assessment in the diagnostic process in psychiatry of the elderly. They remain an important adjuvant and, in this context, a brief test is preferable to a longer one of similar sensitivity and reliability. The AMTS appears to satisfy these criteria admirably.

Appendix I

37-Item Roth–Hopkins Test

Name	1
Age	1
Time (hour)	1
Time of day	1
Day of week	1
Date	1
Month	1
Season	1
Year	1
Place—Name	1
Street	1
Town	1
Recognition of 2 people	2
Date of birth	1
Place of birth	1
School attended	1
Occupation	1
Name of spouse or sib	1
Town where worked	1
Employer	1
Date of World War I	1
Date of World War II	1
Monarch	1
Prime Minister	1
5 minute recall— John Brown, 42 West St, Gateshead	5
Months of year in reverse order	2
Counting 1–20	2
Counting 20–1	2

Appendix II

Abbreviated Ten-Item Roth–Hopkins Test

Age	1
Time (hour)	1
Year	1
Name of place	1
Recognition of 2 people	1
Date of birth	1
Date of World War I	1
Monarch	1
Counting 20–1	1
5 minute recall—42 West St	1

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Monoamine Oxidase Inhibitors and Narcotic Analgesics
A Critical Review of the Implications for Treatment

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There has been a recent renewal of interest in the use of monoamine oxidase inhibitors, but the concurrent administration of narcotic analgesics is often a cause for concern. This review clarifies the different types of MAOI/narcotic interactions and offers guidelines for the use of narcotic analgesics in the presence of MAOIs. The MAOI/pethidine interaction has two distinct forms: an excitatory and a depressive form. Pethidine must never be used in the presence of MAOIs because of the risk of a fatal excitatory interaction. Morphine does not cause this excitatory interaction, and is the drug of choice provided an allowance is made for possible potentiation of the depressive narcotic effect. It is inevitable that strong analgesia will occasionally be required as an emergency measure in patients on MAOIs, and insufficient attention is paid in psychiatric textbooks to the two different types of interactions and their therapeutic implications.

There has been a recent renewal of interest in the use of monoamine oxidase inhibitors (MAOIs) in this country, the USA (Quitkin *et al*, 1979) and Australia (Brown & Cass, 1979). As a British Medical Journal (1976) editorial put it, "This resurgence is timely. Other types of treatment for depression and phobias are far from satisfactory, especially in those patients who seem to respond to MAOI". Although the precise clinical indications remain unclear, they have potential application in anxiety states, phobic disorders, hypochondriacal and depressive neuroses, obsessional neuroses, and possibly more severe depressive illness (Tyrer, 1982).

The main concern in the clinical use of MAOIs relates to the risk of dietary and drug interactions, although in spite of many potential risks, problems are uncommon in practice when care is taken (Crammer *et al*, 1982). Nevertheless, when patients on MAOIs urgently require a narcotic analgesic for emergency surgery or the relief of severe pain there is concern about a potentially fatal interaction on the one hand, or inadequate treatment of the emergency on the other. Current textbook guidance tends to be unhelpful in these circumstances. Pethidine is usually regarded as dangerous in combination with MAOIs, but morphine, methadone, and opiates in general are often also included in lists of contra-indicated drugs. Further advice, when given, usually refers to the test dose procedure of Churchill-Davidson (1965), which involves giving small incremental doses of pethidine or morphine over three hours and is hardly suitable in an emergency.

What is insufficiently appreciated is that the MAOI/narcotic interaction has two distinct forms. There is an excitatory form, characterised by sudden agitation, unmanageable behaviour, headache, hyper or hypotension, rigidity, convulsions, hyperpyrexia, and coma, which is thought to be due to central serotonergic over-activity, and a depressive form, consisting of respiratory depression, hypotension, and coma, due to inhibition of hepatic microsomal enzymes by MAOI with accumulation of unmetabolised narcotic. Pethidine and possibly dextromethorphan are the only analgesics which have elicited the excitatory response.

Interaction with pethidine

The first report of pethidine interaction (Mitchell, 1955) concerned a 60-year-old doctor treated for pulmonary tuberculosis with iproniazid. He developed severe muscle twitching, profuse perspiration, hyper-reflexia, and upgoing plantar responses within 20 minutes of treatment with pethidine (100 mg, i.m.). He recovered and his death a year later was probably unrelated. Iproniazid also had a vogue in the treatment of intractable angina, and three cases were reported of similar sudden deterioration after the additional administration of pethidine (Papp & Benaim, 1958; Shee, 1960). Further reports appeared of seven psychiatric patients on a variety of MAOIs who were given pethidine for surgery, labour pains, severe headache, and backache (Palmer, 1960; Clement & Benazon, 1962; Reid & Jones, 1962; Cocks & Passmore-Rowe, 1962; Denton *et al*, 1962; Taylor, 1962). Six patients had a sudden excitatory reaction; two of these died. The seventh had a gradual depressive response and recovered. Two patients who were treated for hypertension with the MAOI pargyline also received pethidine (Vigram, 1964); one of them quickly became rigid and comatose, and the other one became hypotensive over four hours. Both recovered. One of the few recent reports (Meyer & Halfin, 1981) described a man with coccidioidomycosis who developed 'atypical depression' and was treated with phenelzine. He was also given pethidine for headache, and suddenly became angry, excited, and tearful and made purposeless writhing movements.

On the basis of the results of animal experiments, it has been postulated that the excitatory response is caused by an elevation of 5-hydroxytryptamine (5HT) levels. This follows monoamine oxidase inhibitors (Rogers, 1971) and is potentiated by pethidine, which blocks neuronal uptake of 5HT (Brown & Cass, 1979). Animals pre-treated with inhibitors of 5HT synthesis do not exhibit a rise in cerebral 5HT or the excitatory response (Penn & Rogers, 1971).

Interaction with morphine

In a comprehensive review of MAOI interactions, Stockley (1981) wrote that "well documented, serious, and potentially fatal pethidine/MAOI interactions also cast a shadow over morphine, and resulted in the appearance of morphine in a number of lists and charts of drugs which were said to interact with the MAOIs". However, he found only one case report of a severe morphine/MAOI interaction (Barry, 1979). A woman on tranylcypromine (40 mg/day) underwent a pre-operative test of morphine and became unconscious and hypotensive (but continued to breathe) after receiving morphine (6 mg, i.v.). Neither the weight of the patient nor duration of injection were stated, although the latter appears to have been about ten minutes. This reaction was of a different character to that with pethidine, and the patient became fully conscious and normotensive within two minutes of receiving naloxone intravenously.

An American review (American Pharmaceutical Association, 1976) agreed that there was no evidence for the involvement of narcotic analgesics other than pethidine with MAOIs. Two patients who had severe excitatory reactions to pethidine (one fatal) had previously been given morphine with no adverse effects (Shee, 1960; Palmer, 1960). Brown & Waldron (1962) safely gave morphine (20 mg, i.m.) to five patients who developed severe headaches and retrosternal discomfort while on tranylcypromine. Sargant (1975) knew of some ten cases with no side-effects other than a more prolonged morphine action. Morphine has thus been recommended as the narcotic analgesic of choice in the presence of MAOI administration (American Pharmaceutical Association, 1976).

Interactions with other narcotics

Dextromethorphan, like pethidine, blocks neuronal 5HT uptake. Rivers & Homer (1970) reported a fatal interaction. A 26-year-old woman receiving phenelzine (15 mg q.d.s.) for depression consumed 60 ml of a cough mixture containing dextromethorphan. Within an hour she became unconscious, rigid, and severely hypotensive with fixed dilated pupils. Her body temperature was 42°C. She died despite attempted resuscitation. Dextromethorphan should be considered as dangerous as pethidine and is absolutely contra-indicated.

There is little information about interactions with other narcotics. Methadone has been given concurrently with tranylcypromine, both at 30 mg/day, for three months without mishap in a drug addict who developed depression while on a withdrawal programme (Mendelson, 1979).

Pentazocine has been studied in animals in which interactions with MAOIs have occurred (Rogers & Thornton, 1969), but it is not clear if this occurs in man. Relevant clinical and experimental reports regarding the recently-introduced analgesics buprenorphine, meptazinol, and alfentanil, together with fentanyl and phenoperidine (which are used exclusively in anaesthetics), are scanty. All of these narcotics are best avoided in the presence of an MAOI.

Management

Morphine does not block neuronal 5HT uptake, but its narcotic effects may be potentiated in the presence of an MAOI because of the inhibition of hepatic microsomal enzymes. In an emergency, when pain relief is required immediately, small doses of morphine may be given with caution, intravenously, and carefully titrated against clinical response. Should a depressive reaction occur, treatment is primarily supportive. Naloxone may reverse respiratory depression (Barry, 1979), but assisted ventilation might also be necessary. If conservative methods for the treatment of hypotension fail, noradrenaline is the most reliable vasopressor. Indirectly acting sympathomimetics (such as metaraminol), which act by releasing endogenous noradrenaline, risk provoking a hypertensive overshoot and should not be used.

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Clomipramine, Tryptophan and Lithium in Combination for Resistant Endogenous Depression: Seven Case Studies

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Seven patients suffering from severe endogenous depression who had proven resistant to lengthy trials of treatment with tricyclic and other antidepressants are described. Their successful treatment with a combination of clomipramine, L-tryptophan and lithium is discussed in the context of other strategies for the treatment of resistant depression.

About one-third of patients presenting with a major depressive illness fail to respond to the usual doses of tricyclic antidepressants (Klein *et al*, 1969). A variety of strategies have been suggested for the management of these cases, which have included simply increasing the dose. Many patients with resistant depression respond when the dose is increased to the 200–300 mg/day range (Kotin *et al*, 1973; Quitkin, 1985; Schaztzberg *et al*, 1983). Schuckit & Feighner (1972) and Nelson *et al* (1982) report that some patients seem to require doses in excess of 400 mg/day before a response is seen. Quitkin *et al* (1984) discussed the length of such medication trials, and concluded that a tricyclic must be given in adequate doses for at least six weeks before it may be considered to have failed.

Even with high doses given for an adequate period of time there remains a group of depressed patients with endogenous features who fail to respond to tricyclics alone. The addition of lithium to a tricyclic in non-responders has been demonstrated to produce rapid remission in some cases (Lingjaerd *et al*, 1974; de Montigny *et al*, 1983; Heninger *et al*, 1983; Schrader & Levien, 1985). Similarly, the addition of the serotonin precursor amino-acid, tryptophan, has been shown to potentiate the effect of tricyclic antidepressants (Shaw *et al*, 1975; D'Elia *et al*, 1978; Chouinard *et al*, 1979; Thompson *et al*, 1982). The combination of lithium and tryptophan has also been reported to be more effective than either drug separately (Chouinard *et al*, 1979; Worrall *et al*, 1979; Brewerton & Reus, 1983) although there is little evidence for the antidepressant efficacy of tryptophan alone (Burns & Mendels, 1979; Tissot & Shafner, 1980).

The monoamine oxidase inhibitors (MAOIs) may have a unique role in the treatment of some cases of resistant depression (Quitkin *et al*, 1979). Various authors have advocated the cautious use of combined tricyclic and MAOI antidepressants (Sargant, 1963; Ayd, 1966; Sethna, 1974; Goldberg & Thornton, 1978), but Razani *et al* (1983) compared separate high dose tricyclic and MAOI regimens with a combination, albeit in lower doses, and found that the combination did not prove more effective than either drug alone, although there was no higher risk of side-effects.

Bridges (1983) described preliminary experience with the combination of lithium, tryptophan and tricyclics (in particular clomipramine) in resistant depression, following reports by Eccleston (1981) and Shaw (1983). A similar regimen using an MAOI instead of a tricyclic has recently been reported by Barker & Eccleston (1984). We have used clomipramine rather than an MAOI because the side-effects of the tricyclics are generally less potentially dangerous, especially in higher doses. We report further clinical experience with the clomipramine combination, illustrated by seven cases.

Case studies

Case 1

Mrs J. N. is 60 years old, with a definite family history of affective disorder. Her mother suffered from recurrent depression and had frequent psychiatric admissions. A brother died by suicide, and another brother and a nephew have had hypomanic episodes requiring admission. Mrs N had a normal early development. She left school at the age of 14, and was employed in full-time factory work both before and after her marriage at the age of 36. She continued to work until her first admission to hospital for depression, aged 54. Her only son was delivered by caesarian section when she was 40, with no psychiatric complications.

Her psychiatric illness began fourteen months after her husband died from a brain tumour, when she was 53. She had some social difficulties immediately after his death but generally coped well, looking after her son, then aged 13, and continuing with part-time work. Following a short coryzal illness she gradually became increasingly depressed, with psychomotor retardation, diminished appetite, poor sleep and frequent crying. Admission became necessary; at this time she was not noted to be suicidal or deluded, and had intact cognitive functioning.

She was initially treated with nortriptyline (150 mg/day) for two months. Her condition continued to deteriorate – she became confused and disorientated, and developed memory impairment, a presentation which was increasingly suggestive of a cerebral organic pathology. Investigations, including a computerised tomography scan and lumbar puncture, were normal. Nortriptyline was stopped and she was given a course of ECT (11 treatments over six weeks), with little benefit.

Over the next three years she remained in hospital, wandering about the ward entirely mute and self-absorbed, and was quite unable to look after herself. She was doubly incontinent, and would remain in a soiled bed until prompted to rise. She ate voraciously with her fingers, sometimes on inappropriate foods such as a quarter pound of butter. She would often openly masturbate and remove her clothes in public.

Further unsuccesful attempts at treatment included amitriptyline (150 mg/day) for six weeks, mianserin (60 mg/day) for one year, isocarboxazid (30 mg/day) for eight weeks, and intramuscular flupenthixol decanoate (up to a maximum of 100 mg/week). A further course of bilateral ECT (9 treatments) was given, without benefit, while she was taking a combination of mianserin (60 mg/day) with oral flupenthixol (3 mg/day).

A number of psychological factors were thought to be important in the early stages of her illness, including the grief following the death of her husband and her son's disapproval of a liaison the patient later had with a neighbour. She was also thought by some to demonstrate 'learned helplessness'. Accordingly, family therapy and behavioural programmes were attempted, with no success.

During these three years in hospital she remained mute and in a state of virtual stupor. She was then referred to the Geoffrey Knight Psychosurgical Unit for Affective Disorders at the Brook Hospital in London. There she received a full neuropsychiatric assessment with detailed physical investigations, but again there were no abnormal findings. Taking this into account, together with the strong family history and the initial presentation of an unambiguous depressive picture, the Unit recommended a trial of high dose and then combined antidepressants. She was therefore given clomipramine (up to 225 mg/day, the maximum dose tolerated without hypotension), and later also tryptophan (2 g at night). This regimen produced no benefit, so after four weeks lithium carbonate, increasing to 600 mg daily, was added.

At a ward round three weeks after lithium was started, she casually replied 'not too bad' when asked how she felt, the first comprehensible words that she had spoken for at least three years. Thereafter, her recovery was uncomplicated and quite rapid. She became sociable, involved herself in ward activities, and reported no depression. She remained on this medication and, following rehabilitation, was discharged back to her own home four months after she resumed speaking.

Mrs N. formed a relationship with a male patient during the last weeks of her stay in hospital, and on his discharge he went to live with her. However, there was an argument about money and he attacked her, causing severe lacerations. This necessitated surgical treatment which was associated with her medication being stopped apart from the lithium. She subsequently resumed attendance at a psychiatric day hospital and remained well, so the clomipramine and tryptophan were not restarted.

Three months after her injury, her psychiatric condition deteriorated. She again became confused, untidy and took her lithium only erratically. She was readmitted to hospital, where she again wandered about, and sometimes lost herself in the hospital. She neglected herself and became increasingly withdrawn. Her condition again came to suggest an organic brain syndrome, but investigations were all negative. Clomipramine (225 mg/daily) was then added to the lithium, together with tryptophan (2 g at night). Within three weeks she was well enough to leave hospital. To date she has remained well on these three drugs for four years, still at the original doses. She is now a chatty, extravert, cheerful woman with many interests. She lives independently at home, attends a day centre and goes to a sea-side holiday camp by herself twice a year.

Comment

High-dose clomipramine with tryptophan was not effective on this patient. However, these two drugs, with the subsequent addition of lithium, resulted in rapid recovery after a totally refractory illness lasting three years. When the medication was reduced to lithium only, because of surgical treatment, there was a relapse to the patient's earlier stuperose psychiatric state; lithium alone was not effective, therefore. The addition of clomipramine and tryptophan to the lithium again resulted in recovery, and these three drugs have kept the patient entirely well for the past four years. Reviewing this patient's three years of treatment during her admission, it seems that the triple drug combination succeeded with an illness which did not respond to ECT. However, one could only be certain of this if these drugs had followed soon after ECT had been ineffective.

Case 2

Mrs P. B. is a 54 year-old woman with a 23 year history of recurrent endogenous depression. She had previously been cheerful, well-adjusted and happily married. There is no family history of affective disorder.

She was first admitted to hospital, aged 31 years, with depression; she received no physical treatments and discharged herself after three months. She was readmitted ten years later with depression following redundancy, and she made a slow recovery after ten treatments of ECT. Two years afterwards she again became depressed, and recovered with dothiepin (100 mg/day) after four weeks. She remained well for a further two years, when there was another episode of depression and she took an overdose of her benzodiazepine night sedation. She then failed to respond to amitriptyline (150 mg/day) and to a further course of ECT, and she continued to express suicidal intent.

She was referred to the Geoffrey Knight Unit, and a stereotactic subcaudate tractotomy was performed which produced remission for one year. She then again became depressed, but responded to a combination of dothiepin (50 mg/day) and lithium carbonate (800 mg/day). On this regimen she remained well for nine months, but she then relapsed into a state of agitated depression. She responded slowly to a combination of trimipramine (50 mg/day) and phenelzine (45 mg/day), and on this regimen remained well for two years.

Nine months after this, combination therapy was discontinued. She again relapsed and showed no reponse to its being recommenced, nor to a combination of Parstelin (4 tabs daily) with oral flupenthixol (3 mg/day). Cognitive therapy was tried, to no effect. Clomipramine (100 mg/day) was then given without improvement until tryptophan (1 g/day) was added during the fourth week, following which there was a slow but steady return to normal functioning. The patient remained well on this regimen for two years, but relapsed soon after it was stopped. She then showed a clinical picture of severe retarded depression with suicidal ideas.

Clomipramine was restarted, the dose increasing to 150 mg/day. Above this dose postural hypotension occurred. There was no response after eight weeks and lithium was added, producing a blood level of 0.9 mmol/dm^3. A partial response was seen after two weeks, but complaints of early morning wakening, diurnal variation of mood and lethargy continued. Clomipramine was now tolerated up to 200 mg/day but there was no improvement after a further six weeks. Tryptophan (2 g/day) was added, and the following week the patient attended again with her husband, who volunteered that she was completely well: "back to her old self for the first time in twenty years". She has remained completely well for eighteen months with no change in medication.

This case suggests that L-tryptophan can be essential for the maximum benefit from clomipramine and lithium.

Case 3

Mrs J. C. is a 32 year-old mother of three who presented with a third episode of depression, biological features having been evident on each occasion. She had complete remissions after treatment with dothiepin (150 mg/day) for each previous illness, remaining completely free of symptoms for seven years after the first and two years after the second illness. She has a strong family history of depression, bipolar on the father's side and unipolar from her mother's family. Her three brothers are all affected. Her younger brothers are dizygotic twins; one shows bipolar and the other unipolar symptom patterns. The patient herself has not shown evidence of bipolarity.

Mrs J. C. initially received psychotherapy for the most recent episode of depression, which began with free-floating anxiety and somatic complaints, but she was referred for physical treatments once early morning waking, morning worsening of mood and weight loss become apparent. She received the following medication with little success: dothiepin (200 mg/day) for eight weeks; amitriptyline (200 mg/day) for six weeks; and clomipramine (250 mg/day) and tryptophan (2 g/day) for six weeks.

The patient was initially unwilling to take lithium, and the additon of carbamazepine (300 mg/day) produced little improvement. The patient rejected admission to hospital for ECT, but eventually agreed to a trial of lithium. She was symptom-free within three weeks of achieving a plasma lithium of 0.8 mmol/dm^3 on a dose of 800 mg/day and has remained well over the subsequent eighteen months while also continuing the clomipramine and tryptophan. There was an attempt to reduce the dose of clomipramine, but early morning waking recurred; this cleared up when the medication was returned to the original level.

Carbamazepine, at least at 300 mg/day, thus did not prove to be an effective substitute for lithium in this case.

Case 4

Mrs J. S. P. is a 42 year-old mother of two, referred following the sudden onset of severe endogenous depression with occasional panic attacks. She had suffered an episode with similar symptoms during a pregnancy nineteen years earlier, but this had cleared at the time of the delivery and she had remained well since. There is no family history of affective illness. She had failed to respond to the following medication regimens: mianserin (30 mg/day) for three weeks; dothiepin (200 mg/day) for eight weeks; dothiepin (200 mg/day) and tryptophan (2 g/day) for three weeks.

Drowsiness prevented a further increase in the dose of dothiepin, and the tricyclic was changed to clomipramine, up to a dose of 250 mg/day. This produced some improvements, but significant symptoms remained after six weeks. Lithium carbonate was then added, giving a blood level of 0.65 mmol/dm^3 on a dose of 400 mg b.d. Significant improvement was seen within a week; complete remission was achieved after four weeks, and continues to date (over one year later).

Case 5

Mrs A. B. is a 49 year-old woman with a unipolar depressive illness which began at the age of 44, shortly after a hysterectomy and salpingo-oöphorectomy for endometriosis. Her father had committed suicide while depressed.

She has had two episodes of depression, each requiring hospital admission following self-mutilation and suicide attempts. On her first admission she recovered with ECT (12 treatments) and phenelzine (45 mg/day) with trimipramine (50 mg/day). Although she remained on this combination she rapidly relapsed and was readmitted following a serious suicide attempt six months later.

Her treatment during this second admission included: phenelzine (45 mg/day) and trimipramine (50 mg/day) for one month; imipramine (150 mg/day) for one month; imipramine (150 mg/day) and lithium (600 mg/day) for two months. She then received clomipramine (150 mg/day), tryptophan (2 g/day) and lithium (600 mg/day). She began to improve within six weeks of starting this regimen, and so far has remained fully recovered for eighteen months.

Imipramine and lithium were not effective, but clomipramine, tryptophan and lithium were. This could reflect a possible greater potency for clomipramine compared with imipramine, or the improvement may be related more to the addition of tryptophan.

Case 6

Mr B. P. is a 40 year-old man who had suffered from a single episode of depression varying continuously for five years. He had no previous psychiatric history and no family history of affective illness.

He was admitted to hospital at the age of 36 with severe depression and depressive delusions. He responded partially to ECT (6 treatments) but remained anxious, and he still expressed ideas of worthlessness. He then received the following medication: clomipramine (150 mg/day) for three months; clomipramine (75 mg/day) for two months; imipramine (200 mg/day). Imipramine produced a partial recovery such that he was able to be discharged and resume part-time work. He remained very anxious and depressed, and he received weekly interpretive psychotherapy for two years with little change in this condition.

He was readmitted to hospital, as he had not improved, and he received the following treatment: phenelzine (60 mg/day) for two months; ECT (6 treatments) with little benefit; imipramine (225 mg/day) for four weeks; imipramine (225 mg/day) and tryptophan (1.5 g/day) for four weeks. He was then given clomipramine (300 mg/day) and tryptophan (2 g/day) for four weeks. As there was little improvement, lithium (400 mg/day) was added. Within two weeks he made a full recovery, and he remained well on this triple medication for over a year. Since then he has continued well for another year on clomipramine only.

Case 7

Mrs J. S. is a 58-year old woman who has been admitted to hospital six times with unipolar depressive illness which began at the time of her menopause, at the age of 48. She has no family history of affective illness. Her first five episodes of depression responded rapidly to antidepressants of ECT, and she completely recovered each time.

The last episode of depression began when she was 51; she received the following treatment over the next six years with little effect: amitriptyline (175 mg/day) for two months; amitriptyline (100 mg/day) and imipramine (75 mg/day) for two months; nortriptyline (150 mg/day) for two months; clomipramine (75 mg/day) for three months; phenelzine (60 mg/day) for four weeks; ECT (8 treatments) produced a little improvement, but was not repeated; nomifensine (150 mg/day) for three months; phenelzine (60 mg/day) for three months; mianserin (30 mg/day) for two months; clomipramine (100 mg/day) for three months.

The clomipramine was then increased to 150 mg/day and tryptophan (1.5 mg/day) added. Lithium, increasing to 800 mg/day, was later also prescribed. Within six weeks of receiving these three drugs the patient showed a marked improvement and has remained well on this combination for four years.

Discussion

All seven of the cases reported received lengthy trials of tricyclic and other antidepressants. The doses in Quitkin *et al*'s (1979, 1984) studies varied between 200–300 mg/day, levels considerably higher than are usually used in Britain; Paykel (1979) has recommended that doses should be pushed to the point where side-effects are definite and moderate but still tolerable. In the cases reported this was usually at the onset of persistent postural hypotension, as discussed by Eccleston (1981). In each case several tricyclics had been used, the different side-effects permitting higher doses with some than with others. Management then progressed variously through a variety of other types of antidepressants, combination regimens and then ECT. Two patients received the reputedly hazardous combination of a tricyclic and an MAOI, in both cases with some short-lived benefit. In both, phenelzine and trimipramine was used, as recommended by Young *et al* (1979). Neither these authors, nor studies by White *et al* (1980) or Razani *et al* (1983), found such a combination superior to either drug used alone in adequate doses. Davidson *et al* (1978) compared five weeks of combined treatment with phenelzine and amitriptyline with ECT in resistant depressives. Although the study may be criticised for the low drug doses used, ECT emerged as clearly superior. However, ECT also failed in the cases we have reported.

It may thus be argued that these cases illustrate the unique efficacy of the triple combination of lithium, clomipramine and tryptophan in treating a small group of patients with endogenous depression resistant to many other forms of treatment, and that a trial of this combination should be carried out for apparently intractable cases, because patients might otherwise be condemned to chronic illness. It seems clear that the combination should be tried before a referral for psychosurgery.

There remains uncertainty as to whether the triple combination with clomipramine is more or less potent than with an MAOI instead. Price *et al* (1985) found tranylcypromine and lithium to be effective with a group of patients whose illnesses were resistant to a tricyclic and lithium, and the literature suggests that tryptophan potentiation alone is more convincing with monoamine oxidase inhibitors than with tricyclic compounds (Paykel & Hale, 1986).

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Suicide in Scottish Prisons

S. A. BACKETT

A consecutive series of prison suicides in Scotland between 1970 and 1982 was examined. The initial phase of imprisonment was found to be the most vulnerable stage of confinement, with almost two-thirds of the deaths having taken place within the first month. A disproportionately high number were found to be on remand. A history of some form of psychiatric involvement was common, and a third of the group had received previous psychiatric in-patient treatment. In contrast to results from general population studies, there was little evidence to link suicide with a history of depressive illness. A record of problems with either alcohol or drug dependence was found in almost half the cases. The importance of general measures designed to reduce stress and promote coping mechanisms is emphasised.

Considerable concern has been expressed about the number of suicides and parasuicides which take place in prison. There is disquiet that such an event could take place in an environment in which there is a high level of surveillance, and there is a possibility that the prison regime itself may be seen to be instrumental in the process. While few would deny that imprisonment is an unpleasant experience, society accepts responsibility for it in the belief that it will deter future criminal activity. However, the punitive aspect becomes unacceptable if it is thought to be a causal factor in a prisoner's death.

In 1982 the suicide of a young man in a remand centre near London was followed by an inquest verdict which described a 'lack of care' on the part of the prison department. This was the stimulus for the newly formed Inspectorate of Prisons to undertake both an Inquiry into this death and a more general study of the adequacy of arrangements for the prevention of suicide. The report (Home Office, 1984) considered the prevention procedure and summarised the views of a number of interested bodies. Examination of a sample of suicides showed that about two-thirds of them were regarded as having psychiatric problems, with a smaller proportion (not specified) having been patients in psychiatric hospitals. This supports the earlier finding by Topp (1979), who also examined prison suicides in England and Wales. He found that, while in custody, 69% had seen doctors for complaints with a psychiatric connection and 39% were receiving treatment as a consequence. Despite this, the risk of suicide had been recognised in only 15% of the deaths. The Inspectorate of Prisons acknowledged the importance of general measures to reduce suicide, at the same time emphasising the central role that the prison medical officer should have in 'individual prevention'. The medical nature of the task resulted from an understanding that suicide was often associated with mental illness, alcoholism, and drug abuse, and that it required a doctor to identify these conditions and recommend treatment.

A more recent report (Scottish Home and Health Department, 1985) reviewed the suicide precautions in the Detention Centre and Young Offenders Institution at Glenochil in Scotland. This review followed a series of five deaths which had occurred between 1981 and 1985 and which had been the source of increasing public alarm. Recognising the vulnerability of some of the young offenders, many of the recommendations made and provisionally accepted by the Secretary of State could be considered as general measures designed to make the environment more humane and caring.

Problems are encountered when international comparisons are made. Tuskan & Thase (1983) illustrated this when they drew attention to the wide discrepancies that exist between prison suicide rates in different countries. Variations in reporting methods and inmate populations have been suggested to explain these differences.

The United States Bureau of Prisons administers 35 different forms of penal facility, ranging from maximum security penitentiaries to community treatment centres. County jails are the most widely used form of confinement and are the source of the majority of studies into prison suicides (Smialek & Spitz, 1978; Fawcett & Marrs, 1973; Danto, 1973; Helig, 1973). Even these jails differ from one county to the next, which further complicates comparisons either between them or with state or federal facilities. The studies themselves have varied considerably, both in size and objectivity. While some have defined what they consider to be the characteristics of the suicidal inmate, others have demonstrated that these descriptions are not specific enough to accurately identify the individual and intervene effectively. In this respect it is unfortunate that the most detailed and comprehensive study in this area examined suicide in four maximum security penitentiaries in Canada (Burtch & Ericson, 1979); while data presented in this study are impressive by comparison, it is doubtful that consideration of such a specialised form of imprisonment can contribute to our understanding of suicide in other, more widely used penal institutions.

The present study set out to describe the characteristics of a group of prisoners who committed suicide. In view of the reported association between mental illness and suicide in the general population, it was of particular interest to consider the psychiatric contact, and where possible the diagnoses, of a group of prison suicides.

Method

This study examined a consecutive series of deaths classified as suicide in the Annual Report on Scottish Prisons. In Scotland this process of classification starts with the notification of all deaths which have taken place in custody to the Procurator Fiscal. A report is then sent to the Crown Counsel, who act on behalf of the Lord Advocate and decide which of these deaths will proceed to a Fatal Accident Inquiry. Having heard the evidence the Sheriff, who presides over this Inquiry, is asked to give an opinion on the cause of death. Over the years there has been some inconsistency in these determinations: of those deaths subsequently classified in the Annual Report as suicide there have been some in which a specific reference had been made in the Enquiry that the death was due to suicide; others have been less specific, but have resulted in the same classification.

The Scottish Home and Health Department made available, where possible, the prison records, medical files and Fatal Accident Enquiry reports on each of these deaths. In addition, the Information Services Division of the Scottish Health Service provided data on those individuals who had received in-patient treatment in psychiatric hospitals in Scotland. No corresponding information was available for those who may have had in-patient treatment outside Scotland.

In common with other studies (Fawcett & Marrs, 1973; Esparza, 1973) there were many instances in which the files were not complete. When information on areas of social or psychological importance was recorded both the quantity and quality varied considerably, and many items may have been under-reported.

Suicide rate

The estimation of the suicide rate in a changing population, as found in prisons, requires modification before it can be compared to the rate in the general population. Such a situation has been described for in-patients in a psychiatric hospital (Copas & Robin, 1982). Unfortunately the relevant data necessary to make a similar adjustment for this study were not available. In common with other studies (Topp, 1979; Home Office, 1984) the rate was calculated using the mean daily average prison population for the period in question.

Results

General features

Between 1970 and 1982 there were a total of 33 deaths in Scottish prisons recorded as suicide. All were male prisoners. Suicide was the single largest cause of death, and accounted for 37% of the total. Using the mean daily average prison population (1970–1982), the suicide rate was found to be 51.8 deaths per 100 000 per year. The ages ranged from 14 to 59 years, with a mean age of 28.6 years. Eighteen were single, nine married, and six divorced or separated. Eleven of the group were 'under observation' at the time of their death, with four of these having been identified specifically as a suicide risk.

The early phase of imprisonment appeared to be the most vulnerable stage of confinement. Four deaths occurred in the first 24 hours, thirteen within the first week, and twenty in the first month. Two deaths took place within days of the prisoner's intended release. At the time of death fourteen had been sentenced and nineteen were on remand; of the latter group approximately two-thirds were untried, while the remainder were awaiting sentence or further reports prior to sentence. During the period under consideration the percentage of the total prison population classified as 'on remand' varied between 12.2% and 16.9%, with a mean of 14.1%.

Previous criminal involvement was common. Twenty-nine had a criminal history and 24 had experience of imprisonment. Of those that had been convicted, the majority were serving short sentences for non-violent crimes such as theft. The longest sentence being served was four years.

Psychiatric history

Fifteen members of the group had a record of a previous parasuicidal episode, and in half the cases of two or more. Thirteen had given some indication of suicidal intent in the period immediately before the death. This involved self-inflicted injury in seven cases; in the remaining six cases there was some verbal communication, either to a fellow inmate or to a Prison Officer.

Twenty had a history of psychiatric contact, and for seven this had been within the month prior to the suicide (in some instances this was for the purpose of a psychiatric report). During this period there was only one recorded case in which a depressive illness had been identified and treatment commenced.

Eleven were identified as having received in-patient treatment in psychiatric hospitals in Scotland before their imprisonment. These eleven cases accounted for a total of twenty-four separate admissions (Table I). Depressive disorder had been diagnosed in only two instances.

TABLE I
Psychiatric diagnoses of those prisoners who had received in-patient psychiatric treatment

Case no.	*ICD–9*[1]	*Discharge diagnosis*	*No. of admissions*
1	311.9	Depressive disorder (not elsewhere classified)	1
	307.9	Special symptoms or syndromes (unspecified)	1
2	295.9	Schizophrenia (unspecified)	2
	303.2	Alcohol dependence syndrome	1
3	300.4	Neurotic depression	1
4	301.9	Personality disorder (unspecified)	2
	301.7	Personality disorder (sociopathic)	1
5	295.9	Schizophrenia (unspecified)	2
6	303.9	Alcohol dependence syndrome	3
	301.7	Personality disorder (sociopathic)	1[2]
7	301.7	Personality disorder (sociopathic)	1[2]
	301.6	Personality disorder (asthenic)	1[2]
	304.1	Drug dependence (barbiturate)	1[2]
8	304.9	Drug dependence (unspecified)	1
	303.9	Alcohol dependence syndrome	1
	965	Poisoning	1
9	304.9	Drug dependence (unspecified)	1
10	312	Conduct disorder	1
11	301.6	Personality disorder (asthenic)	1

1. Diagnosis classification according to the International Classification of Diseases (World Health Organization, 1978).
2. Self-inflicted injury on admission.

It had been documented in the records of fifteen of the group that they had either an alcohol or drug-related problem. While the majority of deaths for the group as a whole occurred during the initial period of imprisonment, this was particularly apparent for those prisoners with these problems: nine of these fifteen deaths took place within the first week. Examination of the medical files of this subgroup provided evidence which suggested that a third of them were experiencing withdrawal symptoms at the time of, or immediately before, the suicide.

Discussion

Any conclusions that can be drawn from these data must be qualified by the fact that the study considered only those deaths which had been officially classified as suicide. Apart from noting the numbers involved, no attempt was made to examine critically the other prison deaths.

Studies by both Robins *et al* (1959) and Barraclough *et al* (1974) have shown that in the general population mental illness can be identified in virtually all suicides. Depressive illness was the most common diagnosis, and a previous history of the same illness was found in almost half the cases. If a similar situation exists for those suicides in prison, it follows that any attempt at prevention should involve the early diagnosis and treatment of this illness.

The suicide group in this study had experienced a considerable amount of psychiatric involvement, with some of this contact taking place in the period immediately preceding the death. In addition, one in three had a history of psychiatric in-patient treatment. There was little evidence that depressive illness at the time of death, or a previous history of this illness, was common; in only one case from the prison records and two from the in-patient group could this diagnosis be found. This suggests that factors other than depressive illness may be important in those suicides which take place in prison.

An alternative approach is to view these deaths as the result of varying degrees of stress rather than formal illness. Support for such a situation can be found in the study by Gunn *et al*, (1978), which examined the prevalence of psychiatric disorder in prison. They examined a sample of prisoners with a combination of psychiatric interviews and questionnaires. The results showed that a high degree of disturbance and psychological discomfort was experienced by the prison population as a whole. The level of this distress appeared to be maximal during the initial phase of imprisonment and, although remaining comparatively high, diminished with the passage of time. This change was attributed to a subsequent adjustment to prison life.

Prison suicides follow a similar pattern. This study, in common with many others, found that the majority of deaths occurred shortly after admission to prison. This is the time when the stress factors are most numerous and when the prisoner faces the greatest amount of uncertainty. It could be argued that this results in a continuum of distress, rather than a specific illness with a recognisable constellation of symptoms. In this situation the suicide may take place if, and when, a critical threshold is exceeded. The threshold varies, and is dependent on a balance between the factors themselves and the individual's resources to cope with them. One of the stress factors identified by this study was the symptoms associated with withdrawal from either drugs or alcohol.

Coping mechanisms which might be used by the prisoner have recently been discussed in a review by Coid (1984). He described the way the prison subculture may facilitate the prisoner's adjustment to prison life. In the first place, membership of this group may itself lead to a reduction in the feeling of isolation. In addition, by standing in opposition to the prison staff and management, and by the creation of an 'us' and 'them' situation, the prisoner may regain a feeling of control. This in turn may restore some of his self-esteem and promote a sense of autonomy. The problems arise because acceptance to such a group is neither an immediate process nor equally available to all prisoners. Some will be denied membership owing to the nature of their offence, or aspects of their mental state or personality. As a result of this exclusion they may remain in a vulnerable state. The combination of the clustering of stress factors, and the inadequately developed coping mechanisms, may help to explain the timing of the suicides and the period of adjustment noted by Gunn *et al* (1978).

Finally, it is important to remember that suicides, whether they take place in or outside prison, are rare events, and that it is difficult to predict such an event. This study supports the suggestion that the suicidal prisoner presents with few characteristics which would assist the process of identification. While alcohol and drug related problems may be associated, an identifiable depressive illness does not commonly precede the suicide.

When considering the prevention of suicide in prison it may be more appropriate to emphasise general measures designed to reduce stress and promote coping mechanisms, rather than concentrate on the recognition of the suicidal prisoner. The prison Medical Officer undoubtedly has an important role to play in this. Nevertheless, he will require support from many different areas if the necessary changes are to be made.

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Reality Orientation and Reminiscence Therapy

A Controlled Cross-over Study of Elderly Confused People

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We compared reality orientation with reminiscence therapy for elderly people in a large residential home, using a controlled cross-over design. Both kinds of therapy group were enjoyed by both staff and residents, and enabled staff to get to know moderately and severely confused residents. The group that received reality orientation followed by reminiscence therapy showed improvement in cognitive and behavioural measures which was not found in the other two groups. It may be important to use reality orientation techniques with confused residents before involving them in a reminiscence group.

In spite of the predominantly negative attitude of Western culture to old age (Butler, 1974; Stearns, 1977; Blythe, 1979; Emery, 1981; Thomas 1981) and the many physical, social and psychological difficulties encountered by this age group, the majority of elderly people appear to live busy, meaningful lives. Like younger people, many live relatively independently in the community; either alone, with spouses, or with families or friends. Less than 10% show the kinds of confused behaviour associated with dementia, i.e. disorientation, withdrawal, bizarre habits, or distressed feelings; and even when they do, the majority remain at home or spend only a limited time in hospital (Kay *et al,* 1964; Kay *et al,* 1970; Bergman *et al,* 1978).

Nevertheless, a significant number of elderly people live permanently in residential homes or hospitals, for various reasons. In these populations the proportion of confused residents is much higher than in the community. Recently it has been found that approximately one-third of residents in non-specialist local-authority homes could be described as moderately or severely confused (Evans *et al,* 1981). These residents tend to be dependent and withdrawn and to take little part in the social and recreational activities provided by the home.

For many years it was felt that nothing could be done to improve the quality of life for these residents, apart from providing good physical care. The situation changed, however, when researchers and other workers in the caring professions began to recognise that the environment could exert a powerful effect on behaviour, even when there was a physical basis for that behaviour. They found that elderly confused people could respond favourably to social and behavioural therapy (Taulbee & Folsom, 1966; Folsom, 1968; Burnside, 1976; Citrin & Dixon, 1977; Miller, 1977; Hennessey, 1978). The most widely accepted and researched technique up to now has been *reality orientation* (RO) (Greene *et al,* 1979; Holden & Woods, 1982).

More recently another technique called *reminiscence therapy,* which is based partly on one of the components of RO, has been gaining popularity (Ebersole, 1978; Norris, 1981; Lesser *et al,* 1981). However, there appear to have been no major studies of the effects of reminiscence therapy on the elderly confused.

Both reality orientation and reminiscence therapy aim to help the elderly confused towards three objectives: relearning conversational skills, co-operating in social activities, and taking an interest in the world. Reality orientation aims also to restimulate basic orientation.

The contents of reality orientation programmes vary but all are based on the idea that certain kinds of basic orientating information are essential if the confused person is to function at a reasonable level. Emphasis is therefore placed on teaching the day, date, weather, time and use of names (Barnes, 1974; Holden & Sinebruchow, 1978; Taulbee, 1978; Greene *et al,* 1979; Woods, 1979). Reality orientation, conducted individually or in groups, can lead to a significant reduction in disorientation (Harris & Ivory, 1976; Greene *et al,* 1979; Jeffery, 1981, 1982; Merchant & Saxby, 1981; Powell-Proctor & Miller, 1982) and an improvement in communication for those who are mildly or moderately confused (Brook *et al,* 1975; Holden & Woods, 1982). However, most improvement appears to be fairly specific to verbal reorientation and there appears to be little measurable behaviour change (Powell-Proctor & Miller, 1982). This could be either (*a*) because specific intervention techniques lead only to specific and not generalised results (Hanley *et al*, 1981); or (*b*) because the measures used are not sensitive enough to detect changes in behaviour.

If reality orientation is successful only in improving certain kinds of basic knowledge, it is important to know whether or not this knowledge really is essential for the individuals concerned. Even when the gain in information is accompanied by a more generalised improvement in well-being and an accompanying improvement in staff morale, it may well be that the important elements of an RO programme are not the orientating information but the stimulating influence of the social group. Woods (1979) found that residents in an RO group improved more on tests of information, orientation and various aspects of memory than similar residents in an unstructured social-therapy control-group. It is possible that these improvements were brought about because the RO groups were structured: the actual content of the RO programme may not have been important. Rating-scale assessments of the residents' functioning in the home showed no differential effect; but the staff running the groups felt that the social therapy was more effective and that these groups were easier to lead. This suggests that the staff, and probably the residents, found the social groups more enjoyable. If one of our aims in running groups is to improve the quality of institutional life, it is important that both staff and residents find the experience generally enjoyable or worthwhile; and since the gains achieved during a formal RO programme dissipate quickly once the programme has ceased (Miller, 1977; Feier & Leight, 1981; Powell-Proctor & Miller, 1982), it is important that staff and residents find the experience rewarding so that groups continue and perhaps diversify into different kinds of specialist groups.

This paper reports on an attempt to find out whether our particular RO programme and reminiscence programme were effective in bringing about cognitive, emotional and behavioural changes in the subjects, and whether the effects of the two programmes could be differentiated. It was hoped also to gain more information about the effects of such programmes upon the staff running the groups, since it has been suggested that the main value of RO may be its positive impact on the staff (Powell-Proctor & Miller, 1982; Hanley *et al.* 1981).

Method

Subjects

Subjects were chosen from the residents of a large (62-place) local authority home for the elderly. Out of 20 residents selected by staff as being confused, three were excluded because of severe communication problems. Of the remainder, 15 obtained scores on the 'Information/Orientation' and 'Mental Ability' portions of the Clifton Assessment Procedures for the Elderly (CAPE) (Pattie & Gilleard, 1979) which suggested moderate-to-severe impairment of cognitive functioning. The two with adequate cognitive functioning were excluded from the study. All 15 confused subjects had sufficient sight and hearing to participate in a small group: these 15 subjects were assessed by an independent psychologist, matched for age and cognitive functioning, and randomly assigned to one of three groups.

Some details of the subjects are summarised in Table I: there were no statistically significant differences between Groups A, B and C. All the subjects had been in the home for some time (mean 3 years 4 months, range 8 months–10 years). Their ages ranged from 72 to 90 (average 81.5), and 14 of the 15 were female. Although the residential home regularly provided activities such as bingo sessions, singing groups and outings, our subjects rarely participated and spent a great deal of time in social isolation.

Design

The 15 subjects were divided into three matched groups of five, designated A, B and C. The subjects in the control

TABLE I

Characteristics of subjects at the beginning of the study. The mean value is given for each matched group of five subjects: there were no statistically significant differences between groups

	Group A	*Group B*	*Group C (controls)*
Age: years	81.4	82.7	80.4
Length of residency: years	3.52	4.6	1.92
Information/orientation score[1]	5.4	5.8	5.9
Mental ability score[1]	6.8	8.2	7.4

[1] Measured by the cognitive assessment scale of CAPE (Pattie & Gilleard, 1979)

group (C) received no treatment. Those in Groups A and B were exposed in turn to both reality orientation sessions and sessions of reminiscence therapy, in a cross-over design of study.

Six members of staff were allocated randomly to one or the other of the experimental groups: three worked throughout with Group A and the other three with Group B. Each group session was run by a member of staff and a research clinical psychologist (SB). Preliminary training for the staff consisted of six hours of introductory talks, videos, discussions and hand-outs.

Procedure

Each group met for half an hour a day at the same time Monday–Friday for two 4-week periods. All sessions were tape-recorded. A comfortable, conventionally furnished sitting room was used. Tea was served at all sessions to help create a social atmosphere.

The *reality orientation* sessions followed the format outlined by Holden & Woods (1982) for the 'standard group'. We used a large board for recording the day, month and weather, as well as writing materials, old and current newspapers, and personal and local photographs from past and present. We also used materials to stimulate all five senses (not just sight and hearing); e.g. substances with a distinctive smell, such as vials of rosewater or sachets of cinnamon; materials with a distinctive feel, e.g. silk or sandpaper; and foods with distinctive taste and texture, like pickles or honey.

The *reminiscence therapy* sessions were based on the format suggested by Andrew Norris (Norris, 1986). We used a set of six audio/slide programmes designed to facilitate reminiscence in the elderly (Help the Aged, 1981), old photographs of local scenes, residents' personal photographs, books, magazines, newspapers and domestic articles.

When reminiscence material was used in the reality-orientation sessions, comparison was always made with the present day. These explicit comparisons were not made in the reminiscence therapy sessions, althoug some residents spontaneously commented on the differences between past and present. Historical accuracy was not deemed to be important during reminiscence therapy, but it *was* during reality orientation.

Subjects in all the groups were told that they were helping in an experiment to find out what techniques could help elderly people who were having problems with their day-to-day memory or past memories.

At the end of each session, each staff member made a subjective assessment (on a 5-point scale) of how much each resident had enjoyed the group that day. Staff not running the groups but working closely with the residents involved were asked to make the same assessment, after observing the subjects' behaviour and chatting informally to them about their day.

Measures

Assessment of staff

Staff members running the groups were interviewd individually using a semi-structured open-ended format, and the interviews were tape-recorded. Staff also completed questionnaires about the perceived effects of reality orientation and reminiscence therapy. This was done three times: before training to run groups; again at the end of Stage I; and again at the end of Stage II (see Fig 1). On each occasion they were also asked to complete a personal information questionnaire for each resident in their group and in the control group, without referring to records. The Personal Information Questionnaire (Hanley, 1982) asks basic questions about the resident's family, former work roles, and past and present hobbies and interests.

Assessment of residents

The following measures were taken before and after each period of group therapy, and again one month after sessions ended (see Fig. 1):

1. The Cognitive Assessment Scale (CAS) of CAPE (Pattie & Gilleard, 1979), excluding the Gibson Spiral Maze, and giving measures of:
   (a) Information/orientation
   (b) Mental ability
2. Life Satisfaction Indices (Gilleard *et al*, 1981).
3. CAPE Behavioural Rating Scale (BRS), (Pattie & Gilleard, 1979).
4. Problem Behaviour Rating Scale (Jeffery, 1981), which assesses (on a 7-point scale) specific behaviour patterns associated with confusion.
5. Holden 5-point Communication Scale (Holden & Woods, 1982).

Measures 1 and 2 were completed by the residents with the aid of an independent psychologist. Measures 3, 4 and 5 were completed by staff who knew the residents well but were not involved with the therapy groups.

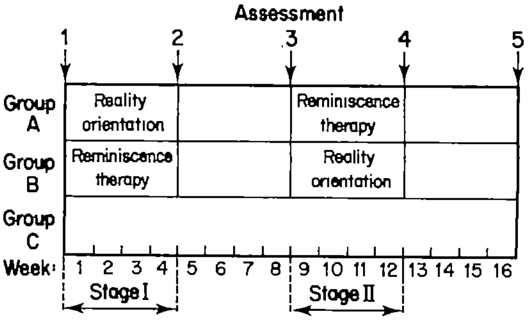

FIG. 1 Design of the study.

Results

End of Stage I

Effects on residents

The results after four weeks of regular sessions are shown in Table II (assessment 2). At this stage, Group A (after four weeks of reality orientation) had improved on measures of information/orientation and communication, and on both

TABLE II

Scores on measures of cognitive ability, emotional state and behaviour, assessed every four weeks throughout the study period. Data represent mean ± s.d. for each group of five subjects. On measures of information/orientation, mental ability and life satisfaction, higher scores represent improvement; on measures of behaviour and communication, lower scores represent improvement

Experimental group	*Assessment*	*Information/orientation*[1]	*Mental ability*[2]	*Life satisfaction*[3]	*Behaviour*[4]	*Communication*[5]	*Problem behaviour*[6]
Group A							
(*Reality orientation*)→	1st	5.4±2.86	6.8±4.76	11.0±2.74	12.6±2.88	16.6±5.55	16.6±9.04
	2nd	6.8±3.59	5.3±5.12	9.8±5.44	*7.8±2.59	14.6±5.13	13.0±7.00
(*Reminiscence therapy*)→	3rd	5.2±3.42	5.6±5.23	9.4±2.7	9.1±4.34	13.2±3.24	11.6±6.07
	4th	*6.4±3.65	5.8±4.97	10.6±3.78	9.0±4.53	12.3±3.99	10.8±4.87
	5th	*6.0±3.39	6.0±5.15	10.2±3.19	9.2±3.96	12.1±2.86	10.6±4.93
Group B							
(*Reminiscence therapy*)→	1st	5.8±3.09	8.2±3.7	9.6±4.93	11.2±4.71	15.4±4.29	14.4±6.35
	2nd	5.8±2.95	7.4±3.58	10.2±7.26	*13.7±9.07	14.7±4.51	14.0±7.21
(*Reality orientation*)→	3rd	*6.0±3.54	6.8±3.83	11.2±7.07	11.4±6.19	15.4±4.51	12.6±5.98
	4th	*5.2±2.28	6.8±3.49	8.6±4.62	11.8±5.93	15.4±3.85	12.4±5.64
	5th	5.4±2.30	7.4±3.13	9.6±6.27	11.2±6.50	13.6±3.29	13.6±5.13
Group C							
	1st	5.9±4.74	7.4±4.22	9.4±3.91	15.8±3.96	20.4±10.31	18.2±8.14
	2nd	6.0±4.3	6.0±4.24	9.6±2.51	*17.0±8.49	23.0±7.07	20.0±9.9
	3rd	*3.6±3.58	5.6±5.23	8.0±6.33	15.8±3.83	19.2±7.61	18.0±7.58
	4th	5.0±4.53	6.0±5.61	8.8±4.87	16.0±5.29	19.0±6.94	17.2±6.30
	5th[7]	*4.2±4.03	5.6±5.18	8.8±5.85	15.2±5.26	18.9±5.76	18.2±7.92

1. Information/orientation: Cognitive Assessment Scale of CAPE (Pattie & Gilleard, 1979)
2. Mental ability: Cognitive Assessment Scale of CAPE (Pattie & Gilleard, 1979)
3. Life satisfaction: Gilleard *et al* (1981)
4. Behaviour: CAPE Behavioural Rating Scale (Pattie & Gilleard, 1979)
5. Communication: Holden & Woods (1982)
6. Problem behaviour: Jeffery (1981)
7. Group C, assessment 5: scores for four subjects only, as one died

*Statistically significant difference between groups behaviour scales. The improvement on measure 4 (BRS) was significantly greater than in the other two groups (Kruskal Wallis $P<0.009$, Group A over B and C; Mann Whitney $P<0.008$, Group A over B; Mann Whitney $P<0.009$, Group A over C). Group B (the reminiscence group) showed a slight improvement in life satisfaction compared with the other two groups, but this difference was not significant.

Since it has often been suggested (Miller, 1977; Powell-Proctor & Miller, 1982) that any positive effects of reality orientation disappear rapidly once sessions are discontinued, the residents were tested again a month after the groups ended (Assessment 3). As can be seen from Table II, the improvements shown by the RO group (A) on measures 3, 4 and 5 (BRS, Holden and Problem Behaviour scales) were maintained, but the improvement in information/orientation score was lost. However, both therapy groups did maintain their scores for information/orientation, whereas the no-treatment control group showed a marked decline: this difference from the control group was significant for the reminiscence group (Mann Whitney $P<0.0259$). It is possible that the therapy groups had some positive interaction with the residents in the control group in the experimental period (see below).

Enjoyment of groups by residents

The residents in both groups A and B seemed to enjoy the sessions: their daily attendance record was high (mean 82% for Group A residents, range 41%–100%; mean 86% for Group B residents, range 65%–100%). This regular attendance might be interpreted as compliance to staff encouragement. However, it was exceptionally high, and in marked contrast to their previous behaviour. Reitz & Hawkins (1982) found that less than 50% of the nursing-home residents in their study attended group recreation sessions, even when staff personally invited them to attend.

Although there were fluctuations in individual scores, staff assessment suggested that all the residents had enjoyed the groups (see Table III).

TABLE III

Enjoyment of sessions by residents (assessed by staff). Rating scale from 1 (non-enjoyment) to 5 (maximum enjoyment). No significant difference was found between groups or between types of therapy

	Staff running therapy groups	*Other staff*
First therapy period		
Group A		
Mean	4.06	4.86
Range	3.25–4.58	4–5
Group B		
Mean	3.58	4.0
Range	3.2–4.0	3–5
Second therapy period		
Group A		
Mean	4.02	4.5
Range	3.31–4.28	3–5
Group B		
Mean	3.67	3.93
Range	3.39–3.85	3–5

Effects on staff

The results from the Personal Information Questionnaire (Table IV) supported the impressions of staff that taking part in group sessions helped them to get to know the individuals in their own groups. Before sessions began, the staff knew very little about the residents in any of the three groups: out of a possible total of 40 items, an average of only 5.33 was known about each resident. By the end of Stage I staff knew much more about the residents in groups A and B: there was no difference in their knowledge between the two therapy groups, but the difference between them and the control group was very large (Kruskal Wallis, $P<0.009$).

All six staff were enthusiastic about the positive effects the sessions had on the residents, although they felt the effects were more marked for the less severely confused. The staff themselves enjoyed taking part in the groups. Five out of six felt that the group experience had helped them to talk more freely to residents on a variety of topics, both outside and inside the group sessions. They also found it easier to allow residents to express their feelings and found that they were no longer avoiding talking to residents about potentially emotive topics.

End of Stage II and follow-up

Effects on residents at end of Stage II

During this stage the treatments were crossed over: Group A residents received four weeks of reminiscence therapy and Group B four weeks of reality orientation. The results are shown in Table II (assessment 4).

Although neither group had met for a month, there was some indication that the previous group experience had affected their behaviour.

Group A residents continued to function as they had done at the end of the previous series of sessions: from the beginning they spoke to one another as well as to staff; they addressed people by name; and they talked spontaneously about current topics of interest in the residential home such as recent illnesses and deaths, and recent or forthcoming outings. They then moved on to the reminiscence topic of the day but continued to compare and contrast past and present.

Group B residents were less willing to use names or discuss day-to-day events and this made it difficult for staff to initiate reality orientation techniques. At first the residents spoke very little to one another and addressed their remarks to the staff. They seemed to remember the room and wanted to talk about their own past experiences, but appeared detached from the other residents. This may explain the drop in information/orientation scores during this period.

TABLE IV

Staff's knowledge of residents: Personal Information Questionnaire scores before and after therapy (maximum possible information = score of 40)

	Scores for individual residents[1]					*Mean*
Group A						
Before group sessions	6	2	5	12	1	5.2
Reality orientation→						
End of Stage I	27	30	29	19	15	24.2*
Reminiscence therapy→						
End of Stage II	37	39	33	22	19	30.0*
Group B						
Before group sessions	4	4	4	11	6	5.8
Reminiscence therapy→						
End of Stage I	28	28	23	24	25	26.6*
Reality orientation→						
End of Stage II	32	34	24	25	27	28.4*
Group C						
Before group sessions	3	4	8	6	4	5.0
End of Stage I	3	4	8	6	4	5.0
End of Stage II	3	4	8	6	4	5.0

*Statistically significant difference from control group

[1] Each score represents the pooled knowledge of the three members of staff involved in the relevant group (A or B); pooled knowledge of all six staff for patients in Group C

Nevertheless, by the end of Stage II they were interacting with one another as well as they had been doing at the end of the first therapy period.

Meanwhile, Group A residents again improved their scores on Information/Orientation, Holden and Problem Behaviour scales: compared with Group B, the improvement Information/Orientation scores was significant (Mann Whitney, $P < 0.0413$). These results support our impression that Group A residents had gained a group identity in the reality orientation sessions which helped them benefit from the reminiscence therapy which followed.

Residents in the control group also improved their Information/Orientation scores but not to the pre-therapy level; and there was no steady improvement on the Holden or Behavioural Ratings, with the exception of one resident who shared a room with a Group A resident. The individual scores of just two residents in the control group were largely responsible for the increase in the mean information/orientation score, and each of these two shared a room with a resident in Group B. No other subjects shared rooms with each other, so it would appear that these residents were obtaining some benefit from the attendance of their room-mates at group sessions.

The changes in the life-satisfaction scores showed the same trend as had been seen in the first experimental period: residents in the reminiscence group improved their scores while those in the reality-orientation group decreased theirs and the control group showed little change.

Effects on residents at follow-up

The residents in the three groups were tested again one month after the experimental period had ended. The results are shown in Table II (assessment 5). Although there was a slight decline in their Information/Orientation scores in the follow-up period, Group A maintained most of the improvements gained in the second therapy period. The scores for the other two groups showed little or no change: but on the Information/Orientation measure Group B residents maintained the scores attained at the end of the therapy period while the average scores of Group C residents declined.

Enjoyment of groups by residents

As in Stage I, although day-to-day fluctuations in individuals' mood and physical state produced some variation in enjoyment, the generally high level of attendance suggested that on the whole residents enjoyed attending both types of group. The daily attendance rate for Group A was 91.8% (range 58.2–100%), with four of the residents attending every meeting. For Group B it was 75.3% (range 52.9–94.1%). The difference in attendance between the two groups was significant (Mann Whitney $P < 0.048$) and may have been due to shortage of staff rather than residents' unwillingness to attend.

Staff ratings of residents' enjoyment was high for both groups (see Table III).

Overall effects of therapy programmes

Over the four-month period of the study CAPE Mental Ability scores declined for all three groups, but less for the two therapy groups than for the control group (see Table II). In spite of this decline, Group A residents improved on the other measure of cognitive ability (CAPE Information/Orientation): By the end of the second stage, Group A residents had improved an average of 18.5%, compared with declines of 10.3% for Group B and 15.2% for Group C (see Figure 2). This improvement was largely maintained one month later, while Group C residents continued to decline, producing a significant difference at this stage between Group A and Group C (Mann Whitney, $P<0.048$).

On the Holden Communication Scale, which reflected the residential staff's estimates of residents' awareness, responsiveness and communication abilities, Group A residents had improved by an average of 25.9% after the two therapy stages, and by 27.4% at follow-up a month later (see Figure 3). In contrast, Group B residents had improved by only 11.7% at the final assessment, and Group C by 7.4%.

Group A residents also improved markedly on the behavioural measures compared with the other two groups. On the CAPE Behaviour Rating Scale they improved an average of 28.6% over the therapy periods and maintained a 27.0% improvement at follow-up (Fig. 4). In contrast, Group B and Group C scores showed little change. Similarly, on Jeffery's Problem Behaviour Scale (Fig. 5). Group A residents had improved an average of 34.9% by the end of therapy and maintained this at follow-up (36.1%; whereas Group B residents only improved by 13.9% and Group C by 5.5%, and even these slight gains largely disappeared at follow-up.

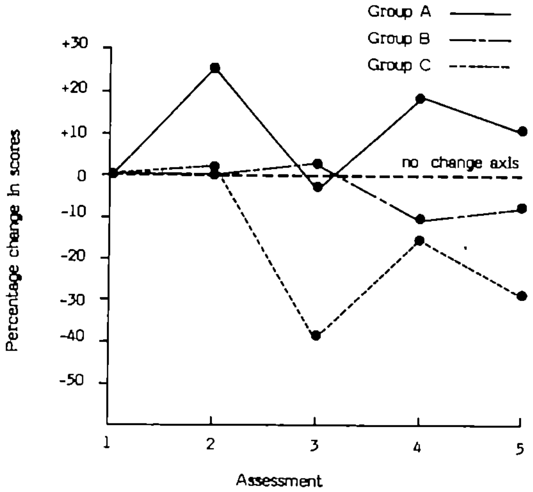

FIG. 2 Changes in mean CAPE "Information/orientation" scores for each of the experimental groups over the 4-month study period, showing percentage improvement (+) or decline (−) compared with the group's initial score.

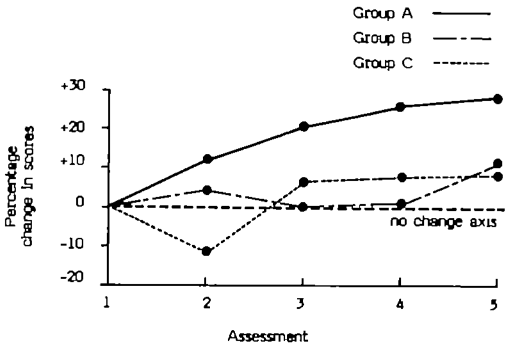

FIG. 3 Changes in mean Holden Communication Scale scores for each of the experimental groups over the 4-month study period, showing percentage improvement (+) or decline (−) compared with the group's initial score.

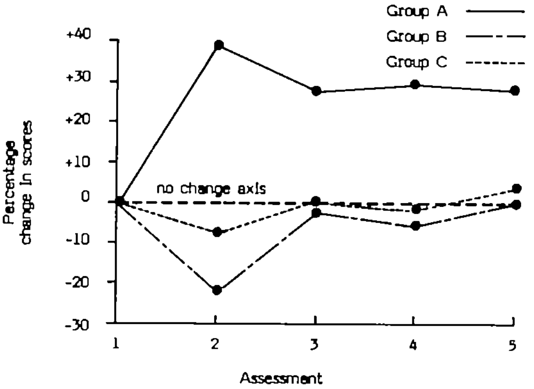

FIG. 4 Changes in mean CAPE Behavioural Rating Scale scores for each of the experimental groups over the 4-month study period, showing percentage improvement (+) or decline (−) compared with the group's initial score.

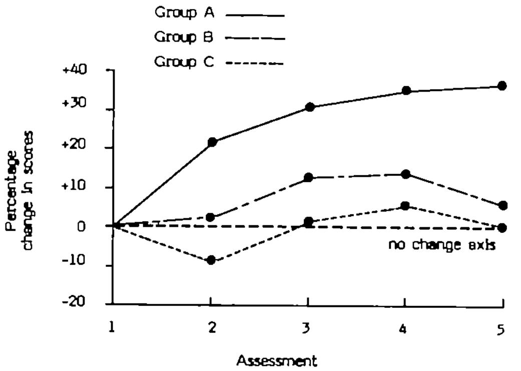

FIG. 5 Changes in mean Problem Behaviour Rating Scale scores for each of the experimental groups over the 4-month study period, showing percentage improvement (+) or decline (−) compared with the group's initial score.

Effects on staff

At the end of the therapy sessions the staff involved said that they knew and understood the residents in their own groups better than they had done before. The experimental evidence to support these statements is shown in Table IV. By the end of the study there was a very significant difference in the staff's knowledge about both therapy groups compared with the control group (Kruskal–Wallis $P < 0.009$, A *vs* B *vs* C; Mann Whitney $P<0.009$, C *vs* A; Mann Whitney $P<0.009$, C *vs* B).

All six staff said that they had enjoyed taking part in both types of groups and thought that the groups should be continued, and extended to other residents who were socially isolated but not necessarily confused; for example, to those who were avoided because they talked repetitively.

Once initial difficulties with a few non-involved staff and residents had been openly discussed, staff running the groups had encountered little resentment and much cooperation from their colleagues.

Discussion

The results of this study suggest that both reality orientation and reminiscence groups can be effective in helping staff to get to know moderately or severely confused residents. It also seems apparent from the high level of attendance, involvement and commitment that both kinds of group can be enjoyable and stimulating for both residents and staff. There was some evidence that the study had positive long-term effects on the residential staff; six months after the study ended, four of the six staff therapists (two from each therapy group) were running weekly sessions for three groups, using a combination of reality orientation and reminiscence techniques. The officers in charge, the staff who had run the groups (and some other staff) were enthusiastic about the effects of the research on the home: they said they were eager to receive more training of a similar nature and had already re-organised staff meetings so that they occurred regularly and were orientated towards staff training. Our results add some experimental support to previous suggestions that reality orientation (Powell-Proctor & Miller, 1982) and reminiscence groups (Norris & Abu el Eileh, 1982; Lesser *et al*, 1981) lead to both increased job satisfaction on the part of staff and improved quality of life for elderly institutional residents.

There was also evidence to suggest that some of the positive effects on group members were passed on to their non-participating room-mates, since room-mates improved on several measures during the therapy periods. This could be because of the increased communication skills of staff and group members. Our finding that these improvements in the control group were short-lived is in agreement with the findings of Brook *et al* (1975) that active involvement of therapists and clients is necessary for sustained improvement.

Group A residents showed greater overall improvement than Group B residents, which suggests that it may be important to use reality orientation techniques with confused residents *before* trying to involve them in reminiscence therapy. However, the results of our life-satisfaction questionnaire suggest that taking part in reality orientation sessions (as opposed to reminiscence therapy) can, for a short period, cause some individuals to report a lower level of life satisfaction. Even though the effect is not statistically significant, the trend is noteworthy since it was shown by Group A and Group B residents. It could be that initially reality orientation had a depressing effect on the mood of the residents because it forced them to face up to the reality of their situation. Indeed, before the sessions began some staff had expressed doubt about the wisdom of making mentally and physically impaired elderly people aware of the reality of their limited existence: they thought it might be kinder to allow them to continue to believe they were in a different time or place, even though that would mean that they were never able to adjust to the loss of their loved ones/or to the impairment of their own abilities; that they constantly asked for people who would never appear; that they remained isolated from the other residents, failed to recognise their visitors, and rarely participated in activities. However, our results suggest that this temporary but realistic lowering of mood may have been a necessary precondition for enjoyment of the present. Tape recordings from sessions and records of observations made by staff show that, as groups progressed and members got to know one another, members began to discuss openly both negative and positive feelings. They talked, mostly in groups but also after sessions, about themselves, about friends, their families, other residents, about their present situation and about their reasons for entering the home. This new-found ability to express emotions, including anger and sadness, appeared initially to alarm some members of staff and some non-group residents: before matters were resolved by discussion there were isolated attempts to discourage residents from attending group sessions. These discouragers reacted as if the groups were *causing* the anger and sadness, rather than confirming the reality of such factors as infirmity, bereavement and loneliness. The fact that the residents refused to be put off suggests that they felt a need to express these emotions.

The improvements in Information/Orientation achieved by Group A residents during their RO sessions (Stage I) were lost in the following month: this accords with the results of previous studies, (Miller, 1977; Greene *et al,* 1979; Feier & Leight, 1981; Powell-Proctor & Miller, 1982). However, in contrast with previous studies (Woods, 1979; Hanley *et al,* 1981; Zepelin *et al,* 1981; Powell-Proctor & Miller, 1982) these residents showed a significant improvement in behaviour compared with the other two groups, and this improvement was maintained in the follow-up period. Furthermore, the improvements in cognitive functioning of Group A residents during subsequent reminiscence therapy suggest that skills learned during RO may have continued to have a positive effect on their behaviour in this period. Although staff placed no emphasis on orientation-promoting material during reminiscence therapy, the residents talked spontaneously about topics such as recent events, planned outings, and current gossip about fellow residents at the beginning of each meeting. Similarly, the reminiscence therapy in Stage I for Group B residents seemed to affect their behaviour in Stage II, when they showed a preference for talking about their own past experiences and, initially, little interest in current events inside or outside the home. Many people enjoy reminiscence and, particularly if their present life situation is unfavourable, look back upon the past with nostalgia rather than as a rich storehouse of experience which could help them enjoy the present and look forward to the future. It is possible that reminiscence therapy reinforced the residents' and staff's inclination to avoid potentially upsetting topics, while the reminiscence component of reality orientation, with its emphasis on comparing both good and bad aspects of past and present, helped them to view both past and present experiences more realistically. Thus one might have anticipated our finding that residents who experienced RO first would do better than residents who took part in reminiscence first, and would have a better chance of deriving longer-term benefits.

The results indicate that a reasonably high level of functioning can be maintained for some time after formal sessions have ended and that losses in functioning which occur while residents are not in a group can be made up once groups restart; this suggests that groups need not necessarily run continuously to be effective. This could be important when considering the feasibility of running small groups in residential homes where staff have little time to devote to the social and psychological care of their residents (Evans *et al,* 1984). When we interviewed staff at the beginning of the study they expressed a desire to spend more time talking to the residents and getting to know them but said there was little opportunity to do so because they spent most of their working hours doing domestic activities and taking care of the physical needs of residents. Staff were also concerned that time spent talking to confused residents in small groups would be wasted, since the residents would not be able to contribute much and would not remember having taken part. However, our results suggest that the group experience prompted the staff to reappraise the ability of disabled residents to derive pleasure and other psychological benefits from small-group therapy. The staff also discovered that these kinds of sessions could be a useful means of increasing their knowledge and understanding of mentally disabled residents. Since staff continued to run groups and take part in in-service training long after the end of the study period it seems likely that their experience in the research project had caused them to reappraise their roles in caring for the elderly, and had increased their feeling that they were doing a worthwhile, rewarding job.

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Explorations of a Crisis Intervention Service

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Descriptive studies of a crisis intervention service replicated previous findings of relationships between staff members' attitudes towards treatment and their 'personal styles'. The attitudes and personal styles of crisis team members and non-members differed, largely because of differences in the professions making up these groups. While all staff groups shared the same concept of crisis, they differed in their discrimination of crisis cases from 'furores'. Initial crisis intervention interviews exhibited more confrontation and less exploration by therapists than did initial psychiatric out-patient clinic interviews. Patients referred to the crisis team differed from significant other people in their lives, and from control patients referred to the ordinary out-patient clinic, in aspects of their perception of problems.

A 24-hour, family-orientated, psychiatric crisis intervention service was introduced at Napsbury Hospital in 1970, resulting in a significant reduction in psychiatric admission rates and in the number of new long-stay patients (Scott, 1980). It is now accepted as the model for psychiatric services in the London Borough of Barnet. The studies reported below focus on three aspects of this service: staff attitudes, therapeutic strategies, and the construing of clients referred to the crisis team.

Staff attitudes

The introduction of the Napsbury crisis intervention service was not without its problems, for "the old institution tried to terminate the existence of the team which initiated community practice" (Scott and Seccombe, 1976). Such resistance to change in the treatment policies of a clinical team or institution has also been noted elsewhere. Caine *et al* (1981) report how resistance to the introduction of a therapeutic community approach in a previously traditional psychiatric hospital ultimately led to the virtual abandonment of this 'adventure in psychiatry' (Martin, 1962). It also provided the impetus for a series of research studies which demonstrated that the attitudes to psychiatric treatment held by clinicians and by patients are a reflection of their more general attitudes to life and their 'personal styles'. More specifically, individuals favouring a medical/behavioural approach to treatment, in which an 'expert' therapist takes a predominantly directive role, were shown to be significantly more conservative and outer-directed than those whose preference was for a psychological approach in which the focus is more likely to be on interpersonal issues and where the therapist-patient relationship is less formal. Corresponding differences in attitudes and personal styles were found between staff working in different treatment settings, and the tension noted by Scott & Seccombe between staff involved in 'old' and 'new' approaches at Napsbury may also have reflected such differences. It was hypothesised, therefore, that members of the crisis team were more inner-directed and radical and had more 'psychological' attitudes to treatment than staff who were not involved in the crisis intervention service.

A further focus of the present study was the concept of 'crisis' held by crisis team members. Bloom (1963) demonstrated that American crisis theorists define a crisis as "an episode beginning with a known precipitating event followed by either no discernible reaction or by some reaction which required a month or somewhat longer to resolve". An elaboration of this concept, as used at Napsbury, was Farewell's (1976) introduction of the term 'furore' to describe the situation which occurs when behaviour, apparently precipitated by some current event, in fact reflects an unresolved crisis masquerading in a different form. It was predicted that crisis team members, unlike staff not involved in the intervention service, would differentiate between true crises (as defined by Bloom), furores, and non-crisis situations in terms of the management options which they considered appropriate for these.

Therapeutic strategies

Reviewing the literature on crisis intervention, Hobbs (1984) remarked that "there is little consensus among practitioners as to what constitutes crisis intervention. A variety of techniques is applied, in a variety of settings, to a wide variety of problems, by practitioners with a variety of skills and qualifications, and with a variety of aims". Similarly, Butcher & Koss (1978) pointed out that the "actual processes by which various crisis interventions bring about desired individual changes have not received enough attention to enable evaluation of them".

In view of such considerations, one of the aims of the present study was to unravel the components of the initial crisis interview, as practised at Napsbury. The concepts on which this interview was originally based were derived from crisis theory (Caplan, 1964) and from Scott's research on the families of schizophrenics (Scott & Ashworth, 1965, 1967; Scott & Alwyn, 1978). A central tenet of crisis theory is that there is a critical period in a person's response to crisis events during which he or she is extremely responsive to therapeutic intervention. For this reason, Scott & Seccombe (1976) saw the first crisis interview as being of supreme importance and as the beginning of treatment. They also considered that this interview should normally be conducted in the patient's family environment, as only there could the therapist prevent 'closure'–the severance of family bonds and avoidance of sensitive issues which contain the key to managing the crisis. In Scott's view, despite demands by the family for the patient to be pronounced mentally ill, the therapist should not succumb to the pressures of a lay diagnosis (Mechanic, 1962), but rather should keep in sight the basic human issues which have precipitated the crisis. His family research suggested that these issues concern a discordance between the perceptions of patients and parents–often between the patients' negative view of parents and the parents' view of themselves. Therefore, a major therapeutic task is "to get the patient to stand by his perceptions of the parents and not to abdicate them in favour of inner states" (Scott & Ashworth, 1967).

On the basis of these theoretical concepts, it was predicted in the present study that the initial crisis interview would be found to be characterised by therapeutic strategies in which the therapist took an active, confrontational role. In contrast, the initial psychiatric out-patient interview–the other major assessment option available to psychiatric patients living in the Borough of Barnet–was expected to be characterised by strategies in which the therapist's role was more passive and facilitatory.

Client constructions

In a pilot investigation of characteristics of patients referred to the crisis intervention service it was hypothesised that if a crisis is indeed a time of greater openness to change and flexibility, such patients would have less tightly organised personal construct systems (Kelly, 1955) than would patients referred to the out-patient clinic. It was also predicted that among the crisis referrals, the views which family members held of each other would be indicative of 'untenable' family relationships (Scott & Alwyn, 1978). Specifically, patients would be expected to perceive their family members more negatively than the family members perceived themselves. Conversely, crisis patients would construe themselves more favourably, and view their predicament less in terms of psychiatric symptomatology and illness than their family members construed them and than patients referred to the out-patient clinic construed themselves.

Scott & Alwyn (1978) used the Family Interpersonal Perception Test, asking family members to apply a set of attributions to themselves, other family members, and the others' view of them. There is now considerable evidence, however, that individuals find their own constructs more meaningful than supplied constructs when rating perceptions of themselves and others (Bonarius, 1977). Accordingly, the present study employed a repertory grid, using constructs elicited from the family members themselves, which it was assumed would be a more sensitive measure of clients' constructions than would the Family Interpersonal Perception Test.

Method

Staff attitudes

A battery of questionnaires was distributed to all clinical staff working in the two NHS psychiatric facilities serving the Borough of Barnet, and to all the general practitioners (GPs) in the Borough who agreed to be visited by a research assistant. The number of members of each professional group who completed the questionnaires, and the percentage of the total possible response from the group, are as follows: 12 psychiatrists (41%); 12 psychiatric social workers (63%); 3 clinical psychologists (75%); 11 occupational therapists (73%); 78 psychiatric nurses (12%); and 54 GPs (31%).

The questionnaire battery had five sections.

(a) Personal Information Sheet. This included questions on the subject's professional background and main and preferred areas of work. With the GPs, it was replaced by a structured interview focusing on attitudes towards the crisis intervention service.

(b) Attitudes to Treatment Questionnaire (ATQ) (Caine *et al*, 1982). High scores on this questionnaire indicate organic, and low scores psychological attitudes to psychiatric treatment.

(c) Direction of Interest Questionnaire (DIQ) (Caine *et al*, 1982). Introversion, in Jung's sense of inner direction of interest, is reflected in high, and extraversion in low scores on this questionnaire.

(d) Wilson-Patterson Attitude Inventory (Wilson, 1975). This measures conservatism (W1), defined as resistance to radical change, together with five of its components, referred to as 'realism' (W2), 'militarism-punitiveness' (W3), 'anti-hedonism' (W4), 'ethnocentrism-intolerance' (W5), and 'religion-puritanism' (W6). High scores indicate conservative attitudes.

(e) Case vignettes: These consisted of ten short case descriptions, four of which described cases which would be considered to be crises according to Bloom's (1963) definition, but which would not fall within Farewell's (1976) category of 'furore'. A further two vignettes described furores, while the situations outlined in the remaining four were neither crises nor furores. Subjects were asked to rate each case on four dimensions concerning their view of the problem; to rank alternative management options for the case in terms of their degree of appropriateness; and to place in preferred rank order different treatment possibilities (which were defined in an attached glossary). An example of each of the three types of case is provided in Appendix I as are the particular questions which were asked.

Therapist strategies

A series of 17 initial crisis interviews and 13 initial interviews at the psychiatric out-patient clinic were tape-recorded, and the tapes analysed in random order by a researcher who was blind as to the type of interview conducted. The system of analysis used was based on the method of Hawton *et al* (1982), which involves allocating every therapist statement (defined as all that a therapist says between two of the patient's statements) to one of nine possible strategies: Facilitation of communication; Exploration; Goal-setting; Informing; Interpretation; Approval/disapproval; Facilitation of emotions; Therapy rules; and Confrontation. The manual of definitions of these strategies was employed by the rater in deciding on appropriate strategy allocation, and where more than one strategy was used in one therapist statement, the predominant one was chosen. Modifications to the original procedure were that allocations to categories were made directly from recordings (i.e. typed transcripts were not used), and that entire interviews were analysed rather than excerpts. In addition to analysis of the whole interview, each interview was divided into four equal sections, and separate analyses carried out on each section. Raw scores for each strategy were expressed as percentages of the total number of interventions, to allow for differences in the length of interviews.

Client constructions

All contructs used by clients in describing themselves and other people were extracted from the tapes. Ten of the constructs elicited from the clients (identified patient and any significant others present at the interview) at each interview were selected (on the basis that they appeared dissimilar in meaning and relevant to aspects of the client's presenting problem) for use in a repertory grid which, if they agreed, was administered to the clients within a week of the initial interview. In the grid each client used the ten constructs elicited from his or her initial interview to rate (on a seven-point scale) the following people: the patient now; the patient before present problems; the patient as he or she will be after treatment; four significant others present at the initial interview (or the patient's mother, father, spouse, and one other significant person); the patient's GP; and two therapists present at the initial interview. Grids were administered to 17 of the patients referred to the crisis service and to 18 of their significant others. They were also completed by 13 patients referred to the psychiatric out-patient clinic, but it did not prove practicable to administer grids to the significant others of these patients. All grids were analysed by Slater's (1972) INGRID computer programme, and group comparisons were carried out on the variation accounted for by the first two principal components, and on measures of distance between various elements, between certain constructs, and between constructs and elements.

Results

Staff attitudes

Relationships between questionnaire scores

Significant inter-relationships were demonstrated between organic attitudes to treatment, outer direction of interest, and conservative social attitudes (correlations: ATQ with DIQ, -0.53, $P<0.001$; ATQ with W1, 0.50, $P<0.001$; DIQ with W1, -0.52, $P<0.001$; one-tailed tests). The only variables to show a significant relationship with age were conservatism and anti-hedonism.

Differences in questionnaire scores between staff members

Members of the crisis team had significantly more psychological attitudes to treatment and were more inner-directed than non-members, as well as obtaining lower scores on the 'militarism-punitiveness', 'anti-hedonism', 'ethnocentrism-intolerance', and 'religion-puritanism' scales (Table I). A further comparison of scores of staff members with a preference for working in the crisis team and those with no such preference revealed similar differences, in addition to the lower conservatism and higher realism of the former group.

In view of the differences in composition of the samples of crisis team members and other psychiatric staff, analyses of variance were carried out on the questionnaire scores, with crisis team membership and professional affiliation as independent variables. The results indicated that the differences between the crisis team members and non-members on all variables except conservatism, militarism-punitiveness and religion-puritanism were due to the differences in composition of these groups in terms of professional disciplines; the significant difference in conservatism between the groups was removed when age was included as a covariate in the analysis. Table II presents mean questionnaire scores of the different professional groups, together with the results of Scheffé tests on those variables with which the analyses of variance indicated significant differences

TABLE I

Differences between questionnaire scores of crisis team members and non-members

	Questionnaire							
	ATQ	*DIQ*	*W1*	*W2*	*W3*	*W4*	*W5*	*W6*
Crisis team members ($n = 25$)								
Mean	40.72	18.24	30.04	29.72	7.16	10.48	5.16	5.88
s.d.	13.11	7.51	13.73	6.04	4.78	4.51	3.30	3.27
Non-crisis team members ($n = 115$)								
Mean	47.70	12.88	34.97	29.54	15.60	15.60	12.97	13.97
s.d.	14.84	11.18	18.62	8.41	13.26	13.26	13.20	16.34
Student's t-test								
t	2.17	2.29	1.25	0.10	2.78	1.90	2.93	2.46
P (one-tailed)	<0.05	<0.05	NS	NS	<0.01	<0.05	<0.01	<0.01

TABLE II

Questionnaire scores of professional groups

	Mean score of group[1]				*F* (*P*)	*Homogeneous subsets* (*Scheffé tests*)	*Mean score: GPs* ($n = 52$)
	SW ($n = 12$)	*P* ($n = 12$)	*OT* ($n = 11$)	*N* ($n = 77$)			
ATQ							
Mean	34.42	42.50	47.00	53.46	6.20	1.SW,P,OT	49.92
s.d.	9.61	16.41	10.62	12.25	(<0.001)	2.OT,N	11.13
DIQ							
Mean	22.67	17.17	15.00	12.09	8.98	1.SW,P, 2.OT,P	14.00
s.d.	4.12	7.73	6.32	4.85	(<0.001)	3.OT,N	7.00
W1							
Mean	20.83	38.67	37.00	44.90	7.74	1.SW	38.27
s.d.	10.83	8.97	12.80	12.71	(<0.001)	2.OT,P,N	12.88
W2							
Mean	31.33	30.83	29.55	28.30	0.70		25.87
s.d.	4.72	6.28	8.42	7.50	(>0.05)		6.92
W3							
Mean	3.00	12.08	10.73	11.93	13.69	1.SW	10.23
s.d.	1.76	4.14	3.13	3.80	(<0.001)	2.OT,P,N	4.40
W4							
Mean	9.00	12.00	11.36	13.41	1.85		14.37
s.d.	2.86	3.67	4.43	4.95	(>0.05)		4.36
W5							
Mean	2.50	8.58	6.36	7.80	5.80	1.SW,OT	6.08
s.d.	1.51	3.09	3.80	3.94	(<0.001)	2.OT,N,P	2.98
W6							
Mean	3.67	8.08	7.55	9.61	3.69	1.SW,OT,P	7.23
s.d.	4.72	6.28	8.42	7.50	(<0.05)	2.OT,P,N	4.79

[1] SW = social workers; P = psychiatrists; OT = occupational therapists; GP = general practitioners; N = nurses.

between these groups. On every measure social workers and nurses occupy the extreme positions, the former group being significantly more inner-directed and radical, and having more psychological attitudes to treatment, than the latter. Furthermore, these were the professional groups which differed most from the GPs, who were significantly more conservative, outer-directed, and organically orientated than the social workers, but significantly less conservative than the nurses.

Concept of crisis

Using Wilcoxon's test,[1] separate comparisons of median scores on response categories for the three types of case vignette were carried out on the responses of crisis team members, psychiatric staff not involved in the crisis team, and GPs. All staff groups were found to rate the crisis cases higher on the crisis dimension than the non-crisis cases, and to consider referral to the crisis team a more appropriate management option for them and crisis intervention a more appropriate treatment approach. Psychiatric staff, whether or not they were members of the crisis team, also regarded psychiatric admission as more appropriate for the crisis cases than for the non-crisis cases. However, only the crisis team members considered crisis intervention a more appropriate treatment option for the crisis cases than for the furores. The GPs considered referral to the crisis team a more appropriate form of management for the furores than for the crises; in common with psychiatric staff not involved in crisis intervention, they also regarded admission as more suitable for the furores than for the crises. Compared with the non-crisis cases, all staff groups rated the furores more highly on the crisis dimension, as well as considering psychiatric admission and referral to the crisis team more appropriate management options for such cases and crisis intervention a more appropriate treatment option.

Mann-Whitney U tests[1] indicated that crisis team members rated cases more highly in terms of their suitability for crisis intervention and for referral to the crisis team than did other psychiatric staff and GPs. In addition, crisis team members gave crisis cases higher ratings on the crisis dimension than did GPs.

Therapeutic strategies

Mean lengths of interview were 42 mins and 52 mins for crisis and out-patient interviews respectively; the difference was not statistically significant. The only significant differences between interview types in mean percentage scores on therapeutic strategies were the greater use of confrontation ($z = 1.85$; $P < 0.05$; one-tailed), and less frequent use of exploration ($z = 1.89$; $P < 0.05$; one-tailed) in the crisis interviews. Taken together, exploration and facilitation of communication accounted for the majority of therapist statements, while approval/disapproval was used least frequently in both types of interview.

However, as therapists conducting the two types of interview were not matched, the differences observed between interviews could have simply reflected differences in particular therapists' styles. In fact, it transpired that in each interview one of the therapists concerned made most of the interventions, and could be designated the 'primary therapist'. Only two of the primary therapists actually conducted both types of interview, while all other therapists conducted only one or the other. Data for these two therapists were therefore analysed separately;. and differences in exploration and confrontation between interviews were not only maintained thereby but actually increased ($z = 2.86$, $P < 0.001$ and $z = 2.01$, $P < 0.01$ respectively). Despite these differences in frequency of strategy usage between the two types of interview, there was little difference in their pattern of usage, exploration being employed less frequently and confrontation more frequently as interviews progressed, regardless of the type of interview (table available on request).

Client constructions

The construct systems of patients referred to the crisis intervention service were less tightly organised, in that the first principal components of their grids accounted for less of the variation than did those of patients referred to the out-patient clinic (Table III). Out-patients saw themselves as more highly characterised by their presenting complaints than did crisis patients, and imagined that they would change more, and become more similar to their ideal selves, following treatment. In the subsample of patients who used a construct of illness, out-patients also viewed their presenting complaints more in those terms than did crisis patients ($U = 2$; $P < 0.05$; one-tailed).

Table IV shows differences between the repertory grid scores of the crisis patients and the mean scores of their significant others. The significant others were more dissatisfied with the patients than the patients were with themselves, as well as seeing them as more highly characterised by their presenting complaints. They also showed a greater tendency to see the patient's present situation as representing a change from his or her previous state. The tendency for the patients to be more dissatisfied with their significant others than the latter were with themselves did not reach statistical significance.

Discussion

Staff attitudes

This study has provided cross-validation data for the Attitudes to Treatment and Direction of Interest Questionnaires, and has replicated the finding of Caine *et al* (1981, 1982) that clinicians' attitudes to psychiatric treatment reflect their more fundamental constructions of the world, i.e. their personal styles. Kelly (1955) has pointed out that any challenge to such core constructs is a threatening prospect, and so the introduction of the crisis intervention service at Napsbury may have posed a threat to those staff members who did not share the particular philos-

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[1] The results of these tests are available on request

TABLE III

Differences in repertory grid scores between crisis patients and out-patients

	Component 1[1]	*Component 2*[2]	*PC*[3]	*PPA*[4]	*IPA*[5]
Crisis patients					
Mean	54.18	19.79	63.58	1.04	0.85
s.d.	14.66	6.00	25.95	0.44	0.43
Out-patients					
Mean	61.90	17.30	44.22	1.34	0.57
s.d.	12.78	5.24	33.54	0.43	0.61
Student's *t*-test					
t	1.47	1.20	1.93	1.88	1.87
d.f.	28	28	21	28	28
P (one-tailed)	<0.10	NS	<0.05	<0.05	<0.05

1. Variation accounted for by the first principal component.
2. Variation accounted for by the second principal component.
3. Mean distance of 'patient now' element from constructs describing patient's presenting complaint.
4. Distance between patient and patient after treatment elements.
5. Distance between ideal self and patient after treatment elements.

TABLE IV

Differences in repertory grid scores between crisis patients and their significant others

	PI[1]	*PC*[2]	*PPB*[3]	*SI*[4]
Crisis patients				
Mean	1.40	74.25	0.90	0.89
s.d.	0.30	25.02	0.34	0.15
Significant others				
Mean	1.66	59.04	1.13	0.75
s.d.	0.20	17.85	0.43	0.39
Student's *t*-test				
t	2.34	1.96	1.44	0.82
d.f.	9	8	9	9
P (one-tailed)	<0.05	<0.05	<0.10	NS

1. Distance between patient and ideal self elements.
2. Mean distance of 'patient now' element from constructs describing patient's presenting complaint.
3. Distance between patient and patient-before-problems elements.
4. Mean distance between significant other and ideal self elements.

ophy which it represented. This possibility is highlighted by the predicted divergences in attitudes and personal styles observed between members and non-members of the crisis team, albeit largely because of their different composition in terms of professional affiliation. However, despite these divergences, some consensus of views was also apparent. For example, most of the GPs who were interviewed expressed general satisfaction with the crisis intervention service. Responses to the case vignettes also indicated that all staff members shared the concept of a crisis case which Bloom (1963) found to be held by American crisis theorists, although only crisis team members distinguished crisis cases from furores in terms of the greater appropriateness of crisis intervention for the former.

The questionnaire scores of crisis team members were similar to those of the therapeutic community staff studied by Caine *et al.* This may reflect the fact that both these approaches view psychiatric problems and their treatment in the context of social systems rather than in terms of 'mental illness', and it is of some interest that several of the staff who developed the crisis intervention service at Napsbury had been involved in a previous, unsuccessful attempt to introduce a therapeutic community. The scores obtained by different professional groups were also very similar to the normative data for such groups presented by Caine *et al.* Group differences of this order would suggest that interdisciplinary conflicts, ostensibly focusing on clinical issues, may in fact reflect divergences in more basic assumptions, values, and social and political attitudes. Although the customary low response rate from a postal survey casts doubts on the representativeness of these findings, it seems likely that the research will have been identified with the crisis intervention service in the hospitals studied, and that staff favourably inclined towards the service may therefore have been more likely to respond to the questionnaires. Such a sampling bias would lead to underestimates of the differences between the staff groups compared.

The present results point to the importance of attempting to change attitudes when introducing a new treatment approach, if it is not to meet with significant resistance. The induction of new members of a crisis team might also profitably focus on attitude change, particularly when a staff member is joining the team as part of a rotation scheme, rather than as a result of active preference for its style of work. Induction should also include explicit statements of the concepts and clinical strategies involved in crisis intervention, and of considerations which are relevant in deciding whether the approach is appropriate in a particular case (e.g. the distinction between crises and furores).

Therapeutic strategies

Evidence has been provided that the initial crisis intervention interview, as practised at Napsbury, is characterised by greater use of confrontation and less exploration by therapists than is the initial out-patient interview. However, the present investigation was essentially a descriptive study of current practice, and it was neither appropriate nor ethically desirable to match clients receiving crisis and out-patient interviews. The possibility remains, therefore, that the differences in therapeutic strategies observed between the two types of interview represent different reactions of the therapists to the two client groups concerned. Evaluation of the present results should also be tempered by the fact that no reliability check was carried out on the coding of strategies. This might have been particularly instructive in view of the fact that, although Hawton *et al* (1982) reported an acceptable level of inter-rater reliability for their coding system, reliability was poorest for codings of confrontation, which they found to be used infrequently in their crisis interviews.

Client constructions

Some, albeit slight, indication was provided that the construct systems of patients referred to the crisis service are less unidimensional than those of patients referred to the psychiatric out-patient clinic. As similar measures of looseness of construct organisation have been related to flexibility and to a greater likelihood of improvement in therapy (Fransella, 1972; Button, 1983; Winter, 1983), this finding lends support to the notion of crisis as a time of greater openness to change, and therefore susceptibility to therapeutic intervention. The tendency of the crisis patients not to construe their predicament so strongly in terms of defined presenting complaints or illness as did out-patients might also suggest a greater amenability to psychotherapeutically-orientated treatment. However, crisis patients also had less positive expectations of the outcome of treatment, or perhaps less motivation to change, and perceived less psychiatric disturbance in themselves than their significant others did in them. Therefore, in contrast to the untenable family relationships described by Scott (1973), the major discordance within the families of crisis patients appeared to concern the way in which the patient was perceived, rather than the way in which significant others were perceived. Nevertheless, the degree of this discordance suggests that family-orientated crisis intervention may have been a particularly appropriate approach to the problems presented by these families.

While these findings are in line with predictions, they require replication with a larger sample. The differentiation between referrals to the crisis team and to the out-patient clinic respectively may not have been an accurate reflection of the extent to which patients were in crisis, although therapists' ratings of the latter in the present study led to essentially similar results. In future research, a more precise indication of patients' degree of crisis might be obtained by the use of a life events measure.

A further shortcoming of the present study was that although the grid assessment was carried out within a week of the client's initial interview, when it was assumed that any crisis which had precipitated referral would still be operative, there was a possibility (and, indeed, this would be predicted in the case of crisis intervention) that the initial interview would already have had a significant effect on clients' construing.

The descriptive studies reported here will set the stage for prospective research comparing the treatment outcomes of, and examining changes in construing in, clients referred to the crisis team and those referred to a psychiatric facility which does not offer such a service.

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Appendix

Examples of case vignettes

Crisis case. A female patient seeks help because her husband is carrying on an affair with another woman. The patient is tearful and says that for the past month, since becoming aware of the affair, she has felt miserable all the time, and has been unable to sleep or to decide on any course of action.

Non-crisis case. A youth of 17 asks for help because he has become increasingly aware of unwelcome thoughts about male friends, and is afraid he may be homosexual.

Furore. Since her marriage broke up, a middle-aged woman has been unable to manage her financial affairs, and whenever she gets into financial difficulties she presents with symptoms of anxiety. She is now presenting again, at the time of a rates increase, complaining that she is unable to sleep and asking desperately for help.

Questions asked concerning each case vignette

1. How do you see this problem? Please place a tick on each dimension
   (a) medical/physical ----- psychological/social
   (b) individual ----- family/marital
   (c) crisis ----- non-crisis
   (d) appropriate response to situation ----- inappropriate response
2. What would be the most appropriate way of managing the patient if he or she is presenting with this problem? Please assume that all the alternatives below are available and rank them in order of appropriateness from 1 to 8:
   (a) admit to psychiatric ward
   (b) place on waiting list for psychiatric out-patient appointment
   (c) refer to crisis team
   (d) refer to non-psychiatric specialist health care/social service, e.g. family planning clinic; marriage guidance clinic; social services (please specify if possible)
   (e) send to other community facility, e.g. Citizens' Advice Bureau; solicitor; Salvation Army hostel; police (please specify if possible)
   (f) should be dealt with by reassurance and advice from GP
   (g) should be dealt with by discussion of emotional difficulties by GP
   (h) does not require professional help.
3. What would be the most appropriate form of therapy? Please assume that all the alternatives below are available and rank them in order of appropriateness from 1 to 8. If you do not know what is involved in a particular treatment approach, please use the definition in the attached glossary.
   (a) psychotropic medication/other physical treatment
   (b) long-term insight-oriented psychotherapy
   (c) brief insight-oriented psychotherapy
   (d) supportive psychotherapy/counselling
   (e) family or marital therapy
   (f) behaviour therapy
   (g) crisis intervention
   (h) no treatment required
4. Comments and other alternatives.

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Referrals to a Scottish Drug Dependence Unit
A Descriptive Study

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The social and demographic characteristics, drug history and current levels of drug taking, drug-related illnesses, and criminal records of a one year cohort of attenders at the Southern General Hospital are described. The findings are compared with those of previous studies at the same clinic; made when methadone was prescribed for a limited period in reducing dosage and when this policy was being replaced by one of the use opiate-free drugs to cover withdrawal.

The misuse of drugs, and in particular of opiates, has increased substantially in the past 5 years. The number of addicts notified to the Home Office in Britain in 1983 (5850) was 42% up on the figure for 1982, and although the 1984 and 1985 figures are not yet available, social indicators of abuse (i.e. the seizure of drugs, the number of drug offences and attendance at clinics) suggest that the figures are likely to show an increase of at least that figure (Home Office, 1985). Ditton (unpublished) predicts a prevalence of 30 500 heroin addicts for Scotland by 1987.

Illegal drugs constitute a multinational industry worth many millions of pounds. The value of drugs seized in 1984 by HM Customs and Excise exceeded the 1983 figure by over 60%. These drugs, mainly cannabis, heroin, amphetamines, and cocaine, were worth £102 million at street prices. Estimates of successful detection rates vary between 5% and 10%; this suggests that illegally imported drugs worth £1000–2000 million found their way into Britain in 1984. It is worth noting that vast quantities of barbiturates and other drugs originate inside the country, and these in turn add to profits made on the illicit market (Institute for the Study of Drug Dependence, 1985).

Since 1980 Glasgow, in common with other European cities; has faced an increase in the availability of heroin on the illegal market. The attention it has received from the mass media has resulted in fear, confusion, and misinformation, emphasising the need for empirical research.

The Scottish Office has responded to the problems by funding several projects in Glasgow, Edinburgh and elsewhere in Scotland. The data in this paper are from referrals to one of these new projects – the Drug Stopping Programme (DSP) at the Southern General Hospital in Glasgow. The DSP available to drug takers is a modification of that described by Drummond *et al* (1986). Replacement opiates are not prescribed; withdrawal is aided by brief reducing prescriptions of chlordiazepoxide and temazepam. In-patient care is offered only to those whose physical or mental condition is sufficiently serious to warrant it. Access to the programme is open; appointments are available by telephone, and the waiting period between contact and appointment is rarely more than one day. First contact assessments are carried out by clinic staff on a rota basis. The process of assessment continues throughout the participant's contact with the programme, using a problem-oriented form of record. Data reported in this paper are taken from first contact interviews.

Method and results

Referrals

During the period of study (Oct. 1984 to Oct. 1985) 279 people were either referred to the programme or booked themselves in. Of these, 216 kept their appointments. The default rate of 22% compares favourably with that for new out-patient appointments in the Department of Psychiatry as a whole. Complete data were obtained from 172 of those attending; reasons for failure to complete the initial interview included time pressure, lack of co-operation, inappropriate referral, and intoxication. Forty percent of the 172 were self-referred; 27% were referred by general practitioners, 20% by social work agencies, 10% by casualty departments, 2% by employers, and 1% by the courts.

Drug preference

There appears to be a trend towards increasing variability in drug-taking behaviour. Although almost all of those interviewed expressed a preference for one drug or drug type over all others, it is clear that their drug-taking was by no means restricted solely to that. They were likely to have at least experimented with the full range of illicitly available mood altering drugs. In particular, the use of cannabis was universally reported by our sample.

Using the main 'problem' drug or drug of choice as the basis for classification, 84% (N = 145) took opiates, (the vast majority heroin); 6% took amphetamine sulphate; cannabis and LSD were causing problems with 3% each; and cocaine, barbiturates, benzodiazepines, and poly-drug abuse accounted for 1% each.

The more detailed description and comparisons below are restricted to the 145 opiate drug takers. The mean age was 22.6 (s.d. = 6.3 years); the modal age was 20, and 51% of the patients were under 21. Seventy-two percent were male.

Drug-taking variables

The mean age at which opiate drugs were first taken was 18.9 years (s.d. = 3.4 years), with a modal age of 17. Seventy percent of the sample had their first experience of opiates between the ages of 13 and 20. Twenty-one percent were taking opiates daily within one week of first exposure; 34% (figures are cumulative) within one month; 50% within three months; 68% within six months; and 93% within the first year. The mean duration of drug-taking prior to contacting the programme was 3.8 years (mode = 2 years). Only 8% of the sample sought help during the first six months, and 19% within the first year, of their addiction.

Estimation of the quantity of heroin consumed is complicated by variations in purity and in the way it is sold. Forensic evidence indicates that the purity of street heroin in the West of Scotland can vary between 8% and 20%. The price has remained reasonably constant over the past 3–4 years at £80/g. It may, however, be sold in 'score deals' of £25, £10 or £5 bags, all of indeterminate quantity; someone who buys eight £10 bags may well end up with less than the person who buys one gram. Differences in the form in which heroin is produced are likely to reflect the availablity of ready cash and access to different levels of the distribution network.

Given these considerations, the amount consumed is best presented in terms of cost rather than weight. At the time of interview 4% of the sample reported spending an average of £5/day or less on heroin; 25% spent £10/day; 25% spent £20/day; 29% spent £40/day; 3% spent £60/day; 10% spent £80/day; and 2% spent between £100–160/day.

Patients were also asked what was the maximum amount they had taken on any one day. Forty-four percent had taken £20–40 worth; 24% had taken £40–80 worth; 15% had taken £80 worth; and 17% had taken £80–160 worth.

The most common method of administration was injection, 73% of the sample using this route by the time of interview. Fourteen percent inhaled and 1% only smoked. Twelve percent took their drugs orally; these were predominantly cases where pharmaceutical products such as codeine linctus, dipipanone, or buprenorphine were the favoured opiates. Drug-related illnesses, predominantly hepatitis B or abscesses at injection sites, had been experienced by 37% of the sample. Four cases had suffered fungal infection of the eye. There were no reports of drug-induced psychosis or other acute reactions; 8% showed evidence of previous psychiatric disorder.

Almost half the sample, 46%, reported one period of abstinence from opiates; 38% reported two or more, and 16% had never been abstinent. There was little evidence that those who had previously attempted to give up drug-taking through their own efforts had been successful; most such attempts had lasted no more than two or three days. However, the mean duration of periods of abstinence for the sample as a whole was 93 days, because drug-taking had been prevented by imprisonment.

Social and demographic variables

Given the average age of the sample, it is not surprising that 74% were single. Sixteen percent were married or co-habiting, and 10% were divorced or separated. The overwhelming majority (93%) left school at the age of 16 without any form of qualification. Indeed, 32% claim to have effectively left school, through truancy, by the age of 15. Six percent had obtained 'O' levels, and one individual had achieved university entrance but failed to complete the course. Eighty-two percent were unemployed, and had been so for a mean duration of 2 years; 16% were working, and 2% were still at school or employed on youth training schemes.

Eighty-nine percent of the sample lived in local authority housing, 10% in privately rented accommodation, and 1% were of no fixed abode. Sixty percent lived with their parents.

There is little evidence of a social support network available to the sample. Only 29% said that they had friends who were not opiate drug-takers; the remainder stated that all their circle of acquaintance had drug-taking habits similar to their own.

There is a clear association between drug-taking and criminal behaviour: 84% of the sample reported financing their drug-taking through crime, predominantly shoplifting and other forms of theft. There were no reports of crime involving violence against a person. Charges were pending against 65%, and 66% had previous convictions, not all related to drug-taking.

Comparison with other samples

In comparing this sample with those from the same unit in 1983 (Fraser & Leighton, 1984) and 1984 (Drummond *et al*, 1986), a number of interesting points come to light (Table I). The best comparison can be made with Fraser & Leighton's sample, since both their study and ours looked at characteristics of attenders (Drummond *et al* mainly investigated the replacement of the prescribing service by the opiate-free day programme currently in operation). The sample of Fraser & Leighton were prescribed methadone for a limited period, in reducing dosage. Fraser & Leighton studied all patients attending the unit at the time, and the dates of first contact thus spread over a number of years; they analysed the data for those whose first contact occurred after 1981 separately, however, and found no significant differences.

Both the mean age at first contact and the age range have decreased since Fraser & Leighton's study (Table I)

TABLE I

Comparisons between three cohorts of referrals to a drug dependency unit

	Fraser & Leighton (1984) (n=80)	*Drummond et al (1986) (n=63)*	*Present study (n=145)*
Attributes			
Self-referred	—	39%	40%
Age at First Contact: years			
Mean	25.1	21.8	22.6
s.d.	5.46	—	6.3
Range	19–55	—	15–35
Married	48%	—	16%
Single	45%	75%	74%
Divorced	7%	—	10%
Unemployed	70%	83%	82%
Employed	25%	17%	16%
In full time education or on a Youth Training Scheme	5%	0%	2%
Criminal convictions	69%	42%	66%
Current illegal activities	30%	27%	84%
Psychiatric history	27%	—	8%
Drug-taking variables			
Age at first use of drugs: years			
Mean	21.0	18.98	18.9
s.d.	4.25	—	3.4
Range	15–55	—	13–35
Mean duration of drug use before contact: years	3.89[1]	2.04[1]	3.8[2]
User friends	75%	—	71%
Never abstinent	44%	—	16%
Abstinent only once	35%	—	46%
Abstinent more than once	21%	—	38%
Hepatitis B	32%	38%	37%

1. Period before contact with any drug agency
2. Period before contact with the clinic where study was carried out

(although the drop is not statistically significant). The initial conclusion might be that drug-taking is becoming popular among younger individuals, due to the greater availability. However, this view may be simplistic. Drummond *et al* (1986) argue that the changes in service provision marked by the introduction of the DSP has made the unit more attractive and available to the younger drug-taker. This trend appears to have continued.

The marital status of both samples also differ. There are more single people in the present sample; this may be because the age group as a whole is younger, or it may have resulted from more chaotic living associated with illegal drug-taking. The number of unemployed persons in the sample is roughly similar, as is reported psychiatric history.

The proportions of patients in the two studies having had criminal convictions was similar. However, in contrast, a greater proportion of the present sample (84%, compared with 30% of Fraser & Leighton's sample) admitted to being engaged in illegal activity to finance their drug taking. A possible explanation for this difference may be that the individuals in the earlier sample found it difficult to admit to criminal activity, as medication was dependent on some measure of conformity.

The mean age for first use of drugs was similar across all three samples. There was an interesting difference in the percentage of patients who were never abstinent: this figure was 44% in Fraser & Leighton's sample, but only 16% in the present sample. Forty-six percent of the present sample were abstinent more than once, while in Fraser & Leighton's sample the figure was only 17%. This improvement may be attributable to health education advertisements encouraging people to attempt to stop using hard drugs. It may also be related to the expense of the habit; when people are paying for the drugs themselves, as in our sample, they are more likely to need to control how much they use.

Discussion

This paper describes the clientele of one of Glasgow's drug clinics; it may or may not be typical of others. The generalisations that can be made from this sample are limited by the fact that only a minority (5–10%) of drug-takers attend a drug dependence clinic (Ditton, 1981). The comparison of this sample with that of Fraser & Leighton (1984) shows these opiate takers to be remarkably similar demographically, although younger. It is possible that younger drug takers are coming to the clinic because staff at the unit are now full time specialist addiction counsellors, with an open appointment system.

The majority of the sample lived with their parents or with a partner. Most have no qualifications, and are unemployed. The level of crime reported is high, and even this may be an underestimate. Roughly one-third of patients gave a past history of hepatitis, which is comparable with figures found in other studies (Stimson & Ogbourne, 1970; Woodside, 1973; Fraser & Leighton, 1984; Drummond *et al*, 1986).

Although we have studied referrals to the DSP, rather than causal factors of drug addiction, some comments regarding the latter are worth making. Speculations about causes of heroin addiction include unemployment, peer pressure, and availability of drugs. In the present sample, 82% were unemployed at the time of first contact. Unfortunately, we do not have data regarding their employment status at or before the time of first use of drugs and unemployment is high in this sample's area anyway. Closer investigation is needed before any relationship can be determined.

Seventy-one percent of the sample reported that all of their friends used heroin. Again, this figure relates to the time of first contact and not to their situation at or before the time of first use. However, 84% reported that they first used drugs in the peer context (either at parties, in pubs or in the company of friends). Again, the peer pressure or social learning theory requires further empirical examination.

Finally, most patients reported that heroin was easily available to them. Perhaps this availability made it more likely that individuals would try the drug in the first place.

These and other factors associated with causation (such as personality factors or rebellion against authority) merit further detailed and long-term investigation.

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Comparison of Electrical Measurements on Constant Voltage and Constant Current ECT Machines

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Electrical measurements were made during treatment of two groups of patients, one group receiving treatment from a constant current ECT machine and the other from a constant voltage machine. The electrical impedance and energy were calculated and compared for the two groups. More energy was delivered by the constant voltage than by the constant current machine, but the spread of energy delivered was comparable. With a consistent technique for applying the electrodes there is little to choose between these machines, but the constant current machine may be preferred where consistency cannot be achieved.

The DHSS report on electro-convulsive therapy (ECT) recommends the introduction of constant current instruments for delivering the electrical energy required (Department of Health and Social Security, 1982). This recommendation is based on the fact that the more commonly used constant voltage device delivers a varying amount of current, depending on the electrical impedance between the electrodes (e.g. a person with half the average impedance would receive twice the average current). The electrical energy delivered by the constant current device would however still vary depending on the impedance encountered.

Two groups of patients chosen for ECT treatment were studied, one group receiving treatment from a conventional constant voltage machine and the other from a constant current apparatus. Electrical measurements were made during the treatment, and comparisons made of various electrical parameters. Assessments were also made to monitor the effectiveness of the treatment using routine clinical criteria, but a clinical trial of the two machines was not undertaken.

Method

Clinical

Twenty-two recently admitted in-patients suffering from acute depression were selected for ECT treatment on clinical grounds. The absence of organic lesions was confirmed where necessary by routine investigations (including skull X-rays and brain scans); nine patients received skull X-rays, and five subsequently had a brain scan. The results of these tests were negative. None of the patients were on any medication, and none had had cranial surgery or skull trauma; none of them had received a course of ECT in the previous three months.

The patients were randomly divided into two groups, without the knowledge of the referring clinician. Group A (2 males, 8 females, mean age = 53) received therapy with a constant voltage apparatus (Ectron Duopulse Mark 4) and Group B (1 male, 11 females; mean age = 45 years) with a constant current apparatus (Ectron Series 2A). The constant voltage device produces a biphasic truncated sine wave with an amplitude of approximately 170 V at a frequency of 50 Hz. The duration of the administration was 1.7 s (85 pulses). The constant current device produces a square wave with amplitude 850 mA, 1.25 ms pulse width, and a repetition frequency of 40 Hz (manufacturers' data). The duration of the administration was 4 s (160 pulses). A course of treatment typically consisted of six ECT administrations, but some had as few as three and one as many as ten.

For both groups bilateral ECT was administered under general anaesthesia (thiopentone) with a muscle relaxant (suscamethonium chloride). Two-handed electrodes were used, soaked in Ectronolyte (sodium bicarbonate and sodium sesquicarbonate) solution (one sachet dissolved in 285 ml of water). Treatment was given using the standard method of applying the electrodes across the temples. At the time of administration the presence or absence of bilateral convulsion was noted; when present, the duration of the convulsion, from the onset of the electrical energy to the last clonic twitch of the great toe, was measured. The administration was carried out by a registrar; over the period of study six individuals were involved.

Patients were reassessed clinically at three months by the referring clinician, who was not present during the administration. Treatment was considered to be successful if the patient could be discharged from the clinic.

Technical

The current and voltage pulses produced by the ECT machine were displayed on an oscilloscope at the time of administration to the patient (Weaver *et al*, 1974). The measuring circuit (Fig. 1) presented a load of 105 kΩ to

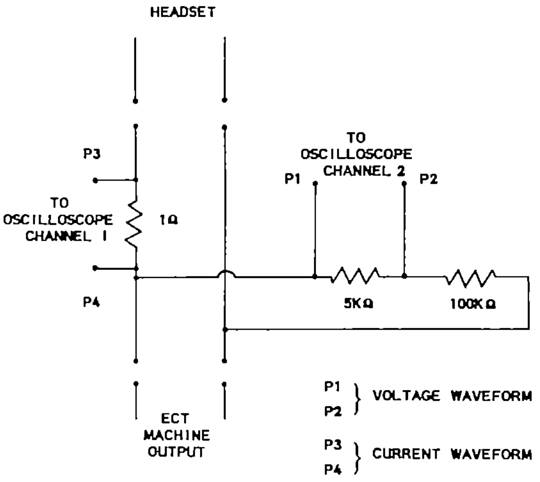

FIG. 1 Electrical circuit used for measuring the voltage and current waveforms.

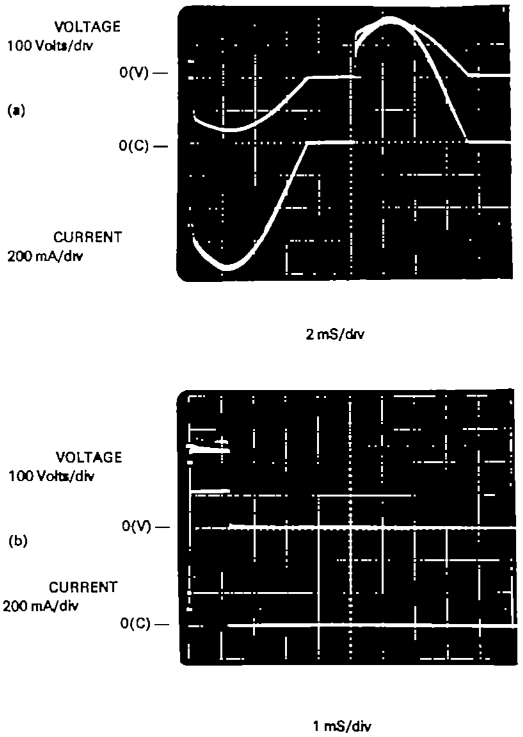

FIG. 2 (a) Cumulative recording (open-shutter photograph) in one subject of oscilloscope tracings (approximately 85) of voltage and current waveforms produced by the constant voltage ECT machine; (b) cumulative recording (open-shutter photograph) in one subject of oscilloscope tracings (approximately 160) of voltage and current waveforms produced by the constant ECT machine.

the ECT machine; this had an insignificant effect on the delivered power (for an inter-electrode resistance of 250Ω the effect is to reduce the resistance to 249.4Ω, which produces a 0.24% difference in the delivered power). A Polaroid camera was used in the open-shutter mode to photograph these pulses, and the resulting photograph (Fig. 2) represents the sum of the pulses produced during each administration. From these photographs the peak voltage (V_p) and peak current (I_p) were determined, and hence the impedance (V_p/I_p) between the electrodes could be calculated, along with the energy delivered. For the constant voltage waveform the energy was calculated using the formula $E = 0.25\ V_pI_p \times 1.7$, derived using the assumption that the sine wave was truncated at the peak value. For the constant current waveform the energy was calculated with the formula $E = 0.05\ V_pI_p \times 4$, derived using the assumption that rectangular pulses of duration 1.25 ms were occurring every 25 ms. The mean values and standard deviations for a series of measurements in each patient were calculated and used to find the mean and standard deviation of the group.

Results

Clinical

In Group A, out of 50 ECT administrations only two failed to produce a convulsion. Similarly, in Group B there were only two failures in 77 administrations. The mean duration of the convulsions in Group A was 32 s (s.d. = 8 s), and in Group B it was 32 s (s.d. = 4 s). The treatment as assessed at three months was judged to be clinically successful in seven out of ten patients in Group A and in eight out of twelve patients in Group B.

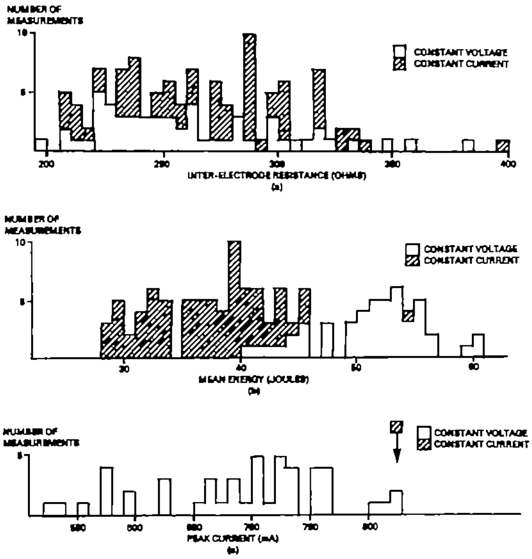

FIG. 3 Distribution of resistance, energy and current values for 50 constant voltage and 77 constant current administrations: (a) resistance; (b) energy; (c) current.

Technical

The photographs of the electrical pulses show that the electrical impedence presented to each machine was purely resistive, since there are no phase differences between the current and voltage pulses. These waveforms confirmed the general characteristics of each machine claimed by the manufacturer, and there was no evidence of additional short duration pulses (transients). The distributions of the resistance, energy, and current are shown in Fig. 3. The mean energy delivered to Group A was 51 J (s.d. =4 J), the mean resistance was 254Ω (s.d. = 31Ω), and the mean current was 700 mA (s.d. = 50 mA). For Group B the mean energy delivered was 37 J (s.d. = 3 J), and the mean resistance was 274Ω (s.d. = 22Ω); these values are significantly different from those for Group A ($P < 0.001$ and $P < 0.05$ respectively; Mann-Whitney U test).

Discussion

The clinical response to ECT in our study was comparable with that reported by other workers (Medical Research Council, 1965; Weschler *et al*, 1965), although no attempt was made to match their research criteria.

The wide variation in inter-electrode resistance reported by Gordon (1982) was not seen in our study. In the ten patients who received constant voltage therapy the minimum resistance was 200Ω and the largest resistance 320Ω, and in the case of twelve patients receiving constant current therapy the minimum resistance was 235Ω and the maximum resistance 380Ω. The slight difference (8%) observed between the mean values was probably due to systematic errors in the measurements of the voltage and current. The main source of the electrical resistance is the contact resistance between the electrodes and the skin of the scalp. Wide variations in resistance can be accounted for by variations in the application technique. Since the standard deviations in the measurement of resistance in individuals (mean across Group A = 29Ω, s.d. = 18Ω; mean across Group B = 34Ω, s.d. = 14Ω) is comparable with the standard deviation of the whole group 31Ω for group A, 22Ω for group B, it would appear that the variability is due to technique and not to variations in physiology. The small spread of the measurements found in our trial reflects the consistency of application of the electrodes by six different individuals. In addition, the concentration of the solution used to soak the electrodes was the same for each application.

The mean energy delivered by the constant voltage device is 38% higher than the mean energy delivered by the constant current device, but the range of energies delivered is comparable in the two cases. These results show that with consistent application of the electrodes, the advantages of the constant current device over the constant voltage device are not realised, in terms of variation of energy delivered. However, although most of the energy delivered by either device is dissipated in the electrodes, proportionately more energy is delivered to the brain by the constant voltage machine than by the constant current machine. Since this trial does not suggest any added advantage in this situation, it is probably desirable to reduce the energy delivered by the constant voltage machine by reducing either the voltage or the duration of the pulses.

The value of energy measurements in determining the convulsion threshold has been questioned (Gordon, 1982), with peak current measurements being favoured instead. Patients receiving treatment from the constant voltage machine experienced peak currents ranging from 520–830 mA (mean = 700 mA), compared with the constant current treatment which was all nominally at 825 mA. The two occasions when no convulsion was observed with the constant voltage machine involved patients on whom peak currents of 550 mA and 762 mA were

TABLE I
The effect of changes in inter-electrode resistance

Inter-electrode resistance: Ω	*Constant voltage*[1] *machine*		*Constant current*[2] *machine*	
	Peak current: mA	*Energy:* J	*Peak current:* mA	*Energy:* J
200	838	57	850	29
300	606	46	837	43
390	488	39	825	54
510	388	33	688	50
1000	220	20	388	31

1. Biphasic, 50 Hz, 1.7 s duration.
2. 40 Hz, 4 s duration.

measured. Two failures to produce a convulsion also occurred with the constant current machine; the only common feature of the four episodes was an indication that a slightly higher dose of muscle relaxant was administered, and this may have masked the presence of the convulsion. However, even if these applications are not excluded it would appear that an adequate peak current was present to stimulate a convulsion in 96% of constant voltage and 97% of constant current administrations.

The effect of changes in inter-electrode resistance (obtained by placing resistors of known value between the electrodes) can be seen in Table I. With the constant voltage machine, increasing resistance causes less energy to be delivered, whereas with the constant current machine more energy is delivered until the constant current characteristic cannot be maintained (above 450Ω). Consequently the latter machine is less dependent on variations in electrode application, since it will compensate for variations in the contact resistance of the electrodes.

In conclusion, with consistent techniques of electrode application there appears to be little advantage in either the constant voltage or constant current ECT machine, but it may be desirable to reduce the energy delivered by the constant voltage machine. Where consistency cannot be achieved, the constant current machine offers some advantages.

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Brief Reports

Varieties of Combat Stress Reaction
An Immunological Metaphor

The use of an immunological metaphor allows the incorporation of established theoretical ideas about trauma and stress in an integrative way which enables delineation and illustration of several varieties of combat stress reaction, including subtle forms of that condition which do not often appear in post-traumatic stress disorder literature.

Traumatic experience and the Israeli population are inseparable. The Holocaust, immigration, the eruption of five wars, as well as continuous military tension, all condensed into a short span of history, have unfortunately had a wide traumatic impact. The abundance of accumulated clinical data, as well as work with Holocaust survivors and psychological war casualties, has shown us that the post-traumatic experience has many expressions and takes many forms.

The concept of combat stress reaction (CSR), a sub-category of post-traumatic stress disorder (PTSD) (American Psychiatric Association, 1980), has suffered from the lack of a unifying explanatory model. Clinicians have tended to one of two theoretical concepts. The first uses Freud's (1921) idea of trauma, in this context to describe a massive emotional shock of such power that it breaks past the defence mechanism and floods the person with uncontrollable anxiety. This implies a highly specific individual vulnerability based on previous early experience, which persists in its power to immobilise afresh. The second and more recently elaborated major theoretical model is that based on the model of stress and coping (Cannon, 1929; Selye, 1956), which describes the accommodation of the organism to general stress. This biological stress model has been expanded to include psychological and social factors, such as occur in the battle situation (Lazarus, 1966; Titchner & Ross, 1974).

These two explanatory theories, while not contradictory, are not conceptually unified. Our ongoing experience with war casualties has revealed variations and nuances in the clinical expressions of CSR that required an approach using both models in combination. The immunological metaphor we have chosen, while not an explanatory model, runs like a thread between the two theories, exploiting the inherent nature of the immune defensive response: current adaptive responsivity based on previous exposure. Thus, the immune metaphor includes both explanatory models, without invalidating either, and facilitates further exploration of PTSD.

Characteristics of the immune system

The immune system is a defence system. It is reponsible for maintaining physical health, and in turn requires for its proper functioning overall integrity of other systems of the organism. To understand the immune metaphor, some of its unique operational modes (Kimball, 1983) must be made clear.

(a) The immune system has both general, innate defensive properties, independent of contact with a foreign substance (antigen), as well as acquired, specific protective responses that are gained through contact with foreign substances (antigen-dependent).
(b) It identifies and reacts to outside noxious elements.
(c) When confronted by an overwhelming, massive exposure to inimical elements, it may fail.
(d) It has a unique ability to memorise identifying features of the threatening element. Once sensitised, it may mobilise an enhanced response when rechallenged by even small amounts of the original threat. Thus, smaller and smaller challenges may produce larger and larger responses.
(e) The immune response may be either immediate or delayed.
(f) Once activated by a specific foreign substance, the response may later be cross-activated by less specific substances, thus changing from a highly specific response to a generalised reactivity.
(g) The immune response can be protective, but it may also miscarry and become self-destructive, creating a new form of illness.

Varieties of acute combat stress reaction

Immediate CSR

Immediate CSR occurs when a psychological system is overwhelmed by a massive, life-threatening situation. The defence capability, if present at all, is initially fragmentary, dissociated, and uncoordinated, and the response undifferentiated and amorphous. This is analogous to an overwhelming assault by a virulent infection, to which the body cannot mobilise an adequate immune response in time. This common battlefield entity is illustrated by the following case-history.

A 25 year-old tank crewman, after three days and nights of continuous action during which many of his comrades were killed, lost communication with his unit. He was discovered in a mute, disorientated state, wandering aimlessly in the desert, and was so overwhelmed with anxiety that he had to be evacuated by helicopter to a field hospital.

Acute CSR

The clinical picture of acute CSR is that defined by DSM–III under the category of acute PTSD. It involves such symptoms as: re-experiencing the traumatic event; numbing of responsiveness to, or reduced involvement with, the external world; and a variety of autonomic, dysphoric, or cognitive symptoms. Just as the clinical symptoms of an infection reflect the product of the invading and the defending forces, so the acute combat reaction may be seen as reflecting a duel between the anxiety-provoking, painful, and as yet unintegrated memories and emotions on the one hand, and the defensive efforts to block, seal off, or dampen them on the other. This rise in defensiveness may be reflected both in increased intrapsychic defensiveness and in behavioural-interpersonal responses such as irritability, decreased sociability, and withdrawal. It is this dynamic interplay of the anxiety and the attempt to overcome it which is so characteristic of acute CSR, and which determines the form of the ultimate outcome, whether towards resolution or towards chronicity.

The soldier described in the previous section became coherent after two days, and was then transferred to a rear-echelon psychiatric facility. His disorientation continued to improve, but he was troubled by nightmares, and by flashbacks of battle scenes during his waking hours. He was extremely irritable, hypersensitive to noise, and subject to bouts of crying, while at the same time reluctant to discuss his battle experiences. His complaints gradually abated, and by the end of five months he was sufficiently symptom-free to resume normal activities.

Missed immediate CSR

As in the immune system, the resistance to combat stress depends on the degree of previous preparedness (innoculation), and on the intactness of the surrounding support systems. As in most infections, the natural history of immediate CSR tends in most cases towards spontaneous remission. A certain proportion of these cases would therfore not be recognised or documented (Moses & Cohen, 1984), and as such could be defined as missed immediate CSRs. Whether the immediate condition remits, or persists to evolve into acute PTSD, the subject, like the immune system, becomes sensitised. He is thus permanently altered, harbouring the potential for a future enhanced response on re-exposure to a similar threatening stimulus; this lays the foundation for a possible delayed reaction.

Varieties of delayed combat stress reaction

On exposure to trauma, regardless of whether or not there is an acute response of the kinds mentioned above, there is always the possibility of a delayed response (Archibald & Tuddenham, 1965). This is analogous to delayed responses in the immune system; the use of the immune metaphor assists in discerning the delayed forms of PTSD.

Reactivation of combat reaction

Just as an exposure to a minimal amount of allergen in a sensitised patient may lead to an extreme reaction – even tranaphylactic collapse – so may soldiers who have suffered previously from frank, acute CSR collapse under minimal military exposure, years later.

A 25 year-old infantryman, diagnosed as suffering from acute CSR in the Yom Kippur War of 1973, returned to full functioning in civilian life, including family and work involvement. He managed to maintain his equilibrium until call-up, 11 years later, to the war of 1982. From the moment he received his mobilisation papers he became increasingly anxious, and finally collapsed on arrival at his unit, as it was preparing for the field.

Reactivation of missed combat reaction

This is a variation on the reactivation of CSR, mentioned above. Just as a patient who appears to develop a massive hypersensitivity reaction for the first time is found to have suffered subclinical, undiagnosed previous responses, so a soldier suffering from an unexpectedly severe combat reaction may be discovered to have undergone an antecedant, unnoticed CSR.

A 28 year-old soldier was evacuated with a full-blown CSR, which at first sight seemed to have been generated by a relatively minor battle stimulus: the chance sight of a casualty, witnessed from afar. In treatment it became clear that this disproportionate reaction actually originated from a trauma sustained in a previous war, when he suffered a severe CSR, which went undiagnosed at the time but which nonetheless, served to sensitise him.

Displaced combat stress reaction

Hypersensitivity, once established, may generalise and turn into cross-sensitivity, which implies development of the full-blown immune response, provoked by a wider range of less specific stimuli (Christenson *et al*, 1981). Similarly, a CSR may be provoked by a life event completely unrelated to war, occuring in a civilian setting.

A 31 year-old civil engineer developed a full-blown PTSD, following the death of his father. His symptoms consisted of guilt-laden dreams and flashbacks related to the tramatic war experience of six years previously, from which he had not suffered in the interim.

Our experience indicates that almost any life event which bears some relationship to any facet of the original trauma has the power to create a displaced CSR: e.g. loss of any kind, such as the death of a family member, divorce, road accidents, or dismissal from work.

Delayed combat stress reaction

The clinical picture of delayed CSR is, like the acute type, defined by DSM–III under the heading of PTSD, chronic or delayed. The symptoms are essentially the same as in acute CSR, except for the time element. The symptoms either endure for six months or more, or develop at least six months after the trauma (Archibald & Tuddenham, 1965).

The delayed CSR can be likened to an illness developing months or years after the exposure to the invading agent. The form of the illness is the product of the late expression of the bodily immune defences, which have become over-responsive and harmful in their own right. An example is tuberculosis, where the patient may present suddenly with a serious cavitating lung condition due to hypersensitivity to the bacillus, many years after first becoming infected

A 24 year-old soldier experienced many frightening combat situations, which evoked fear, anger, and grief at the loss of comrades. He continued his military function, although feeling "robot-like'. At the end of the war he was discharged normally and returned to civilian life, continuing university studies and renewing social contacts. Increasingly, he felt that he "wasn't the man he had been previously". He was surprised that he had mounting difficulty in concentration, unexplained depressive feeling, and was unable to enjoy life. Suddenly, without an apparent trigger, seven months after discharge, he developed sleeplessness and nightmares about friends who had died in the war; he also had flashback phenomena, and became apathetic and immobilised.

Retrospective combat stress reaction

In certain immunological illnesses, such as serum-sickness, a circulating foreign substance will develop enhanced antigenic properties, when the host later develops sufficient antibodies to cause a reaction. Similarly, there are soldiers who develop a changed attitude to previous war-related experience, so that re-evaluation of combat memories produces a stress reaction which was originally absent. This altered perception may be as a result of changed circumstances in their lives, particularly changes related to attachment and bonding, such as marriage or the birth of a child. These changes, which would be seen by life-cycle theorists such as Erikson or Maslow as healthy psychological growth and maturation, cause a more disturbing light to be shed on what was previously perceived simply as an adventurous event or a necessary unpleasant duty. The subject develops the capacity to imagine and to identify with the emotional consequences of killing and being killed, so that fragments of previous experience become retrospectively traumatic. This phenomenon has been noted in the Holocaust literature (Krystal, 1968), as in the case of the woman who, as a young girl, witnessed with relative emotional detachment the bashing to death of a baby plucked from its mother's arms by SS guards. Only after attaining motherhood herself, many years later, was the woman able by identification to experience the full horror of such an event; she then became severely anxious at the prospect of losing her own baby. New attachments may thus produce alteration in basic attitudes, so that veteran, married soldiers may now find it impossible to perform military functions that were once easy for them, and are statistically more at risk to develop CSR than their younger, single, comrades-in-arms (Solomon *et al*, 1984).

A 25 year-old soldier who experienced many frightening events as a member of an elite special-forces unit subsequently married and had a child. These new attachments in his life altered his perception of his previous close encounters with extreme danger, and he reported some anxiety at the thought of what he had been through in the past. He stated that the battle experience, previously perceived as adventures, had acquired a new frightening dimension, reported vague tension at the idea of being called up again, and was embarassed to discover that he was immobilised with anxiety. On rejoining his unit, he was suffering from a condition identical to immediate CSR, was unable to continue serving there, and was referred for psychiatric care.

Discussion

The clinical picture of CSR, and hence of PTSD, is richer than appears in the current literature. DSM–III has enabled broad categorisation of the nuclear syndrome of PTSD, while providing a valuable diagnostic tool (Solomon *et al*, 1984) for the identification of CSR. Our accumulated clinical experience through several wars reveals that the varieties of CSR are numerous and subtle, so that the immune system has proved to be a particularly useful metaphor in delineating and presenting them in a systematic way. This metaphor highlights the complexity of the interplay between stumulus and response, as well as the multiform clinical nature of combinations in which they appear. It reduces the relevance of the dispute over the importance of the source of the trauma – whether early or late, intrapsychic or interpersonal, personality-related or stimulus-related – and so contributes towards simplifying the paradigm that has troubled clinicians working in the field of trauma. Although essentially biomedical, it serves to incorporate psychosocial-aetiological factors in an integrative way. Moreover, the metaphor has important and direct ramifications, both in its preventive and therapeutic implications; while we have found it useful in discerning the varieties of CSR, we believe it is no less useful for civilian work, e.g. in the general hospital setting (Kutz *et al*, 1986).

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Heroin Abusers in Psychiatric Beds

A Glasgow Study

A retrospective study was carried out of all heroin abusers admitted to an acute psychiatric unit to examine their use of the facility of in-patient care. The compliance with treatment was low. Most patients discharged themselves or were discharged prematurely for using drugs while in the ward; only 21% were discharged as planned. The value of hospital admission in the management of opiate abusers may have to be reconsidered in view of the increasing prevalence of heroin abuse and the limited number of psychiatric beds.

The admission of opiate abusers to hospital for in-patient detoxification was once considered to be a mainstay in their management (Home Office, 1968), but is not now thought to be so important (Thorley & Plant, 1982). It may be a useful first step in a longer programme of rehabilitation involving either community projects or therapeutic communities (Department of Health and Social Security, 1984). In the London drug dependence clinics detoxification is usually carried out on a long-term out-patient basis (Ghodse, 1983). In Glasgow the recent rapid rise in heroin abuse (Ditton & Speirits, 1982) and the reluctance of psychiatrists to prescribe opiates on an out-patient basis has resulted in admission to acute psychiatric units continuing to be used as the main psychiatric response to the problem in many hospitals.

In theory, the benefits to be gained by admitting a heroin taker to hospital are

(a) assessment of physical health and of social and personality factors influencing the drug abuse
(b) management of the physical and psychological withdrawal symptoms in a supportive and controlled environment
(c) provision of 'time out' from the drug scene
(d) opportunity to plan changes in lifestyle
(e) introduction to a community-based project for continued rehabilitation.

The psychiatric service of Glasgow Eastern District, based at Duke Street Hospital, regularly admits heroin abusers to acute psychiatric beds. Initial screening is done on an out-patient basis by a senior psychiatrist to confirm the diagnosis, identify the level of abuse, and assess motivation. In-patient care is offered if appropriate, and patients are usually admitted within three weeks. The duration of in-patient care is flexible, being dependent on the patient's progress as assessed by staff.

Following admission, assessment of physical health is carried out and appropriate treatment given. If there is objective evidence of opiate withdrawal symptoms (such as pupillary dilatation, tachycardia or sweating), medication in the form of oral methadone, chlordiazepoxide or clonidine is prescribed. Random urine screening is undertaken frequently during the patient's stay to detect any surreptitious use of opiates in the ward. Patients are informed beforehand that their use of drugs in the ward will generally result in their discharge. During the day patients attend sessions of group psychotherapy and occupational therapy.

This study was carried out to examine the use of this facility of in-patient care by a group of opiate abusers and their progress.

Method

All heroin abusers who were admitted to the ward between 1 June 1983 and 31 May 1985 were included in the study. Their case records were examined to obtain basic epidemiological data, drug history and details of their progress in the ward.

Results

Over the two year period 56 heroin takers were admitted (41 men and 15 women). In most cases (84%) referral was made by general practitioners, but referrals were also made by other medical specialties (14%) and by social workers (2%). The mean age of the patients was 22.7 years (s.d. = 4.24 years, range = 14–36). Although most were single (79%), none lived alone.

Drug use

The mean reported duration of opiate abuse was 3.4 years (s.d. = 2.16 years) and all patients had been regularly injecting opiates intravenously before admission except for four who had been taking the drugs by insufflation. It was difficult to obtain precise information on the daily amount of opiates taken, as this was usually expressed as the amount of money spent on heroin per day. The mean reported amount spent was £37/day (s.d. = £25.5/day, range = £5–120/day); at the time of the study the patients reported the price of 'street' heroin to be £80–100/g, although its purity and potency varied widely.

Progress in the ward

Only 20% of the patients did not require medication to relieve withdrawal symptoms. Most patients (61%) received oral methadone given in reducing doses from 30 mg/day over eight days. A further 14% received chlordiazepoxide (initially 80 mg/day), and 5% were given clonidine (initially 0.2 mg/day), both these drugs being given orally.

Most patients (61%) either discharged themselves or left the ward without permission and did not return. A further 18% were discharged by medical staff after urine tests confirmed that they had been using opiates surreptitiously in the ward. Only 21% remained in the ward until their planned date of discharge. A total of 31 patients (55%) achieved a drug-free existence while in the ward in that they were neither receiving prescribed drugs nor using drugs surreptitiously, but many of these patients discharged themselves shortly after reaching this stage.

Those who remained in the ward until their planned date of discharge had (as expected) a longer mean duration of stay (32.7 days; s.d. = 17.2 days) than those who took their own discharge (10.2 days; s.d. = 10.5 days). Seven patients (12%) took their own discharge after only one day. Those who were discharged prematurely for using opiates while in the ward had a mean length of stay of 28.3 days (s.d. = 23.4 days).

Premature discharge was not associated with the non-prescription of medication; indeed, those who completed their stay were slightly less likely to have received medication (25% received no medication), and 90% of those who were discharged due to using drugs surreptitiously had initially been prescribed methadone.

Discussion

At this hospital one heroin abuser is admitted on average every two weeks; these patients are typical (in terms of age and sex) of heroin abusers attending other drug dependence clinics in Glasgow (Fraser & Leighton, 1984). More than half reach a drug-free state, however short-lived, while in the ward, but many of these patients discharge themselves shortly afterwards. Most patients do not remain in the ward until their planned date of discharge but discharge themselves after an average of ten days. A further number are discharged by medical staff for using opiate drugs surreptitiously while in the ward.

The prescription of medication to ameliorate withdrawal symptoms seems in anything to have a negative effect on satisfactory progress, although it is possible that the patients who required such medication were more severely dependent or less motivated and therefore less likely to remain in the ward. If is difficult, however, to quantify withdrawal symptoms, and the value of admission may lie in the 'time out' element rather than in pharmacological detoxification. In addition, the relatively long period of time after admission (28 days) before many patients started using opiates in the ward suggests that the longer-term craving for the drug, perhaps enhanced by boredom, is just as likely to produce relapse into using drugs again as the acute withdrawal symptoms. For this reason, much emphasis is placed on using the period of in-patient care to introduce the patient to a longer-term programme of rehabilitation in the community to prevent future relapse. Since most patients discharge themselves before this can be properly effected, much doubt is thus cast on the overall usefulness of admission.

The main shortcoming of the study is the lack of objective long-term follow-up information on the patients. We have had little success in arranging for these patients to attend the hospital for follow-up, which is perhaps a result of the policy here of not prescribing substitute drugs on an out-patient basis.

The significance of the frequent outcome of irregular self-discharge is also uncertain. It is possible that those who discharged themselves irregularly from the ward were just as likely to lead a drug-free existence thereafter as those who remained until their planned date of discharge. However, the often sudden, impulsive and desperate nature of the wish to leave hospital in these cases suggests that it probably indicates imminent relapse. Even if admission to hospital does not result in total abstinence thereafter from opiate use, though, it may have a significantly beneficial effect, perhaps by reducing the degree of dependence or by decelerating a process of physical and social deterioration.

The practice of admitting heroin abusers to psychiatric beds may not be a completely useless exercise, and currently accounts for a small proportion of psychiatric bed occupancy, but the poor compliance observed in this study suggests that this form of management now needs to be examined more critically in the current climate of limited psychiatric resources and an ever-increasing population of heroin abusers seeking psychiatric help.

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Capgras Syndrome: A Case Report from Zimbabwe

A case of Capgras syndrome in a black Zimbabwean patient is described. The syndrome occurs in the setting of a schizophrenic illness, but psychodynamic factors are readily identified despite the patient's different cultural background.

Early clinical descriptions by French authors (Magnan, 1893; Janet, 1903; Capgras & Reboul-Lachaux, 1923) referred to 'l'illusion des sosies' (the illusion of doubles), and it was not until 1929 that the term 'Capgras syndrome' was suggested (Levi-Valensi, 1929) in recognition of that author's contribution. For some years the syndrome was believed to be rare (Enoch & Trethowan, 1979), but recent reports indicate that it is much more common than was formerly suspected (Quinn, 1981). Berson (1983) reviewed the English literature, and found 133 cases. A report has also been received from Jamaica (Sims & White, 1973), and in Africa Collomb (1965) described the characteristic misidentification in one of his patients but did not identify the syndrome. We were unable to trace any other report of its occurrence in a black African patient.

Case report

A 27 year-old single, unemployed black male patient was admitted to Harare Hospital in June 1982 with a three-day history of violent and aggressive behaviour towards his father, and also of consuming large quantities of alcohol and stripping in public. On admission he told the staff angrily that his father was not his father but only a "caretaker", who was forcing him to visit various *ngangas* (traditional healers) with the intention of proving that the father was in fact the real father. He further alleged that the surburb of Mufakose, where the "caretaker" resided, was not real either but a carbon copy of the genuine town.

The patient's belief that the real father had been substituted by a "caretaker" began five years before admission, when the patient suddenly resigned from his job because "the boss was looking at him in a queer way". The following day, his father took him to discuss this with a priest. While the discussion was in progress the patient waited outside. He then observed that an impostor and not his father emerged from the church. He was certain that it was an impostor because of the differences which he noted: the real father was a kind, caring, religious and good person, whereas the "caretaker" was a fat, ugly, mean and strict person, who wanted to make the patient a slave in the house. The "caretaker" somehow knew the patient's thoughts and prevented him from taking a job. The patient further believed that the "caretaker" was bewitching him.

The father reported that from that time the patient had never again addressed him as "father" but invariably referred to him as "that man". The patient became argumentative, disrespectful and often broke household objects. Once he threw away the family radio because it was broadcasting news about him. He suspected that neighbours were plotting against him and that his family had poisoned his food. He often spoke to imaginary people.

Except for the delusional content, there was no evidence of thought disorder at the time of admission. Auditory hallucinations were present: the patient could hear the voices of his ancestral spirits comforting and commending him, and sometimes discussing him in the third person. The patient's account of himself was given with a moderate pressure of speech and with much circumstantial detail. His affect was angry and hostile. There was no evidence of either depression or elation. Consciousness was clear, and cognitive function normal. Physical and neurological examinations revealed no abnormality; investigations for syphilis, malaria, trypanosomiasis and cannabis intoxication proved negative. The electroencephologram was within normal limits.

While on the ward, the patient claimed that he could distinguish the "caretaker" from his father by his greater obesity, different voice, and facial expression. After one visit by his parents he insisted that the "caretaker" had a different watch and had hair like a European (i.e. straight, not tightly curled). His mother tried to convince him of his father's authenticity, but he retorted that the "caretaker" was trying to seduce her into marriage; he accused her of having divorced her real husband. Occasionally he wondered if his mother were an impostor as well, but this idea never assumed delusional strength.

Personal background

The patient was the eldest of 8 siblings. At the age of two he was sent to live with his grandparents in a rural area. During early childhood he lived a carefree life tending cattle, fishing in ponds and playing in mud pools. After the sudden death of his grandfather he returned to his parents at the age of eight years. His father was a hard-working factory worker in Harare, a religious man who was strict with his children. On his return home the patient had to start school, attend church daily and refrain from playing with dirty children in the neighbourhood. As the eldest of the family he was subjected to extra pressure and discipline to succeed academically. At the weekends he had to spend most of this time in social service connected with the church. His mother was a quiet, unassuming, dependent woman who never challenged her husband's opinions or instructions about bringing up the children.

In this atmosphere, the patient grew up to become a quiet, shy, lonely, obedient and studious adolescent. He could neither develop any peer group activities nor mix readily with girls; he had profound feelings of guilt over masturbation. After two unsuccessful attempts to pass his 'O' level examinations he left school at the age of 20 to become a painter's assistant. His father wanted him to study further, and repeatedly admonished him for failure. He continued to have difficulty mixing with people, and developed no relationships with women; he became a heavy drinker. After a year at his job he left because he was convinced that his colleagues were somehow against him.

Course and outcome

A diagnosis of paranoid schizophrenia was made, and treatment began with chlorprimazine (1000 mg/day). The symptoms gradually subsided over the next four weeks, and after six weeks in hospital the patient was discharged to the care of his parents. He was followed up as an out-patient for the next 18 months. He stopped addressing his father as "caretaker" or "that man" and once again called him father. He took a job at the same factory where his father worked, and expressed a feeling of shame that he had treated his father with disrespect during his illness.

Discussion

As our case illustrates, Capgras syndrome occurs in schizophrenia, but many authors have stressed the importance of organic factors (Weston & Whitlock, 1971; MacCallum, 1973; Hay *et al*, 1974; Hayman & Abrahams, 1977; Preskon & Reveley, 1978; Waghray, 1978; Shraberg & Weitzel, 1979; Alexander *et al*, 1979; Madakarisa & Hall, 1981) and some have seen it as an adaptational manoeuvre in old age (Goldfarb & Weiner, 1977).

It seems remarkable that such a specific syndrome should result from diverse pathological processes. The double is always a significant person in the life of the patient (who does not misidentify others), and is usually accused of not possessing the features the patient would like the original to possess. Such features strongly suggest the operation of psychodynamic factors, and it also seemed remarkable to us that such factors could be readily identified in our patient despite his non-European cultural background.

In Shona culture, feelings of love and respect towards one's father are accepted as normal; hostile feelings are expected to be suppressed from conscious expression.

A father is responsible for the moral upbringing of his son, and any disrespect shown towards one's father is regarded by society as a moral crime against the ancestral spirits. In our patient, these traditional values of filial respect were inculated by his grandparents (it is common to send a child who has been weaned – usually at about two years of age – to keep the grandparents company). These values were subsequently reinforced by a rigidly moral and religious upbringing. There is evidence that the father attempted to drive the boy academically which, together with peer group rejection, is a common cause of disturbance during adolescence (Buchan, 1971). It seems likely that our patient developed hostility towards his father and dealt with it by repression.

This intrapsychic stress, his difficulties in interpersonal relationships and his recourse to alcohol may have been precipitating factors for his psychotic illness.

Intense suspiciousness has often been observed prior to the onset of the Capgras delusion (Capgras & Reboul-Lachaux, 1923; Enoch & Trethowan, 1979) and our patient exhibited this kind of projective defence when he left work. With the development of the psychotic process and the failure of the early defences, the defence of splitting emerged. The patient's internal representation of his father was split into good and bad individuals (Arieti & Bemporard, 1974), and the bad individual was seen as an impostor. The message from our patient seemed to be quite clearly "Father must change". The motive appeared to be adaptational – an attempt to reconstruct the environment by replacing the current non-giving, unacceptable father by a warm, nurturant one.

Despite his regligious commitment, the patient's father had taken him to many *ngangas* in the hope of finding a cure. This accounted for the long delay between the onset of the illness and his being brought for treatment. The hospital was seen as a last resort when the patient became violent, which is probably the case with the majority of patients in this setting.

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Ganser Syndrome

A Case Report and Discussion

A case of Ganser syndrome occurring in an 11 year-old Indian boy is described, and the psychodynamics and nosological status of the syndrome discussed. Ganser syndrome should be regarded as a hysterical state, and should not be confused with the similar picture observed in organic brain syndromes and psychoses.

In 1897, Sigbert Ganser delivered a lecture on 'A peculiar hysterical state'. Later, after having examined twenty such patients, he refined his description of the clinical syndrome which now bears his name (MacDonald, 1977). It is also often called 'prison psychosis', 'syndrome of approximate answers' or 'hysterical pseudodementia' (Sim, 1974). The most important feature of this syndrome is that the patient gives incorrect and often ridiculous answers, despite having comprehended the question (this is termed '*vorbeireden*'). Whitlock (1967) regarded the condition as a psychotic one, occurring either after acute cerebral trauma or in the course of an acute psychotic illness. Many other authors agree with Ganser's original diagnosis of a special kind of hysterical dissociative state. The cases described by Ganser were all prisoners, but the syndrome is not confined to criminals (MacDonald, 1977). The condition is rare, especially in children.

Case report

An 11 year-old boy was referred for psychiatric evaluation with a five-day history of attacks of hysterical unconsciousness and periods of confusion during which he gave wrong answers and claimed amnesia for certain events. Fifteen days before admission he had experienced several fainting attacks; these had been attributed to evil spirits. At the onset of his presenting symptoms he suddenly started weeping loudly and complaining that he was not being given proper attention.

The patient lived in a village with his paternal grandparents, while his parents and two younger sisters lived in Bombay. The only educated member of the family was the father, who completed high school. The patient's mother tongue is Marathi, but he was sent to an English-speaking school as his grandparents had high expectations of him. He was initially regarded as a bright student, but his scholastic performance had deteriorated over the two years before admission.

The majority of the villagers were farmers and labourers, and placed little importance on education. Because the village had only a primary school which taught in Marathi, the patient attended a school in the neighbouring city. Since he was the only such student from the village, he was singled out from the village boys, and had no close friends among them. There was no-one who could help and guide him in his studies.

An English education is considered essential in India for upwards social mobility. The patient was thus under tremendous pressure to achieve good school results, despite the linguistic, scholastic and social difficulties that he had to face.

Throughout his childhood he demanded and received prompt attention from all his family. He had many food fads, and would have temper tantrums at the slightest provocation. He would always try to dominate his sisters and mother.

The immediate precipitating factor for his illness was his grandparents' refusal to send him on a school excursion. His fainting attacks followed the refusal, and his major symptoms started when his friends returned from the excursion.

The results of his physical and neurological examination were normal. On mental status examination he demonstrated *vorbeireden*. While counting to one hundred he omitted a few numbers; and answered that ten plus two equalled eleven. He initially claimed amnesia for his school's and village's names, but remembered them after a few minutes. He suddenly and dramatically developed hiccups, which stopped spontaneously when no attention was paid to them, and displayed *la belle indifference*.

After admission he showed a dramatic recovery, which was consolidated by giving suggestion during sodium pentothal interview. During psychotherapeutic interviews he stated ambitions to become a lawyer, a physician or an engineer. He drew a house, and said that in it lived two girls with their parents. Despite the obvious reference to his sisters and parents, he could not elaborate further. He accepted that he was finding it difficult to do his study in English. On intelligence testing his IQ was 107.

Discussion

This is the first case report of Ganser syndrome in an Indian child. Kanner (1972) reported a case of Ganser syndrome in a six year-old girl; he was able to find only one other report of the syndrome occurring in children.

Ganser specified four major features of this syndrome (Arieti & Bemporad, 1974): giving approximate answers, a clouding of consciousness, somatic or conversion symptoms, and hallucinations. Of these, the first symptom is essential. Our patient had the first three symptoms. Although headache is the commonest somatic symptom reported, in this case conversion symptoms (hiccups) and dissociative state (unconsciousness) were prominent features. The patient's personality development clearly showed hysterical traits, such as immaturity and demanding and manipulative behaviour. The syndrome has never been reported in a person of high intelligence; this patient, although regarded as bright by his parents, was of average intelligence.

Lehmann (1976) stated that in almost every case the patient finds himself in a confining situation. Such a situation was not found in this case. Although the patient was under stress, particularly as a result of approaching examinations, he did not view himself as trapped or confined.

Arieti & Bemporad (1974) suggested a simple psychodynamic mechanism for the syndrome: the patient wants to avoid an unpleasant situation and the burden of responsibility. This was the situation in this case: the patient was finding his schooling increasingly frustrating, his end-of-year examination was approaching, and he could not accept the possibility of failure. An illness with dramatic cognitive difficulties provided him with an excuse for subsequent poor performance. This is the secondary gain in this case. The presence of conversion symptoms, dissociative state, secondary gain, and response to suggestion favours the view that Ganser syndrome is a hysterical state and not a psychotic illness.

Whitlock (1967), in his review of the literature, concluded that the condition is a psychotic one. However, he himself accepted that the cases described in his paper and the majority of the cases described by other authors did not have even the majority of the symptoms described by Ganser in his original paper. It is advisable to accept Scott's (1965) suggestion that a differentiation should be made between Ganser syndrome and Ganser symptoms (approximate answers). Ganser symptoms are comparatively common, and are usually seen in acute functional or organic psychoses, while Ganser syndrome is clearly hysterical in nature.

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Folie à Deux in Down's Syndrome

A Case Report

A case of folie à deux in a 29 year-old man with Down's syndrome, whose mother had a paranoid psychosis, is described. Successful treatment of the mother's illness also resulted in full recovery of the son without the need for medication.

In their classic paper of folie à deux, Lasègue & Falret (1877) stressed that the first condition for its development in an individual is that "he be of low intelligence and "better disposed to passive docility than to independence". Tuke (1887) also suggested that induced psychosis is more likely in the "feeble-minded", and Gralnick (1942*a*) described folie à deux in two sisters, one of whom was mentally subnormal.

Various psychiatric abnormalities may be observed in Down's syndrome (Parsons *et al*, 1984), but there have been no reports of folie à deux in combination with this disorder. We report such a case here.

Case report

Mrs A., a 77 year-old woman, was first seen at her home at the request of her general practitioner. She complained that two young women who had escaped from prison were living in the rafters of her house; they had moved in six months previously, shortly after her husband was permanently admitted to hospital with a stroke. She claimed that they were particularly noisy at night, when they banged on her bedroom ceiling and window, threw insults at her and her 29 year-old son who lived with her, and clambered up and down drainpipe outside singing hymns and chanting "incy wincy spider, climbing up the spout" as they went. She believed the women to be in league with the next door neighbours, whom she thought broke into her kitchen at night to steal food and money, but were clever enough to leave no trace.

Mental state examination revealed a thin small suspicious woman who looked her age. She showed no depression of mood, disorientation or memory impairment, and displayed no insight into her delusional beliefs.

Her son had the physical features of Down's syndrome, and later chromosomal analysis confirmed trisomy 21. When interviewed separately he confirmed his mother's story and said that he sometimes heard the womens' voices independently from her; for example, they swore at him whenever he flushed the toilet at night, told him that his father was dead, and threatened to cut his throat if they caught him.

Mrs A. had married at the age of 27, and in addition to her son had three daughters, all of whom were married and lived away. She and her son were very close and mutually supportive, but he also enjoyed his daily attendance at a local training centre. He was socially competent, being continent and ambulant and having good self-help skills. He had no previous history of psychiatric illness or behavioural disturbance. Mrs A. also had no past personal or family history of mental illness, but was described by her family as having always been eccentric and domineering. Her psychosis appeared to start at about the time of her husband's hospitalisation, and in association with unexpected family bereavements and conflict with her neighbours over an aggressive dog. Following these events she became increasingly withdrawn and frightened, stopped her son from attending his training centre, and slept in the same bed as him for protection.

A diagnosis of paranoid psychosis was made in the mother and of folie à deux in her son. She was treated at home with trifluoperazine and community psychiatric nurse support, but refused to take her medication or agree to be physically investigated; however, she responded well to fluphenazine decanoate injections. Her son remained at home with his mother during this period, and his delusions and auditory hallucinations resolved completely without the need for treatment as her mental state improved. On later psychological testing he was found to be cheerful, sociable and co-operative, and was able to give the date and month but not the year of his birth. He was unable to read, but could name the letters of the alphabet, showed good colour and shape matching, and was able to cope with quantities up to four, however, he was unable to deal with more than two combined qualities or to seriate. At the age of 5 his IQ had been 52; his current IQ was assessed at 45. Speech therapy assessment using the Reynell Language Development Scales revealed that his comprehension was at the level of a 5 year-old, but that his expressive ability was that of a 7 year-old.

The results of subsequent physical examination and laboratory, radiological and neurophysiological investigations on Mrs A. were normal except for a low serum vitamin B_{12} level of 80 ng/l (normal range 180–960 ng/l); a Schilling test revealed an intrinsic factor defect, and she was started and maintained on intramuscular hydroxocobalamin. There has been no return of her psychosis despite the discontinuation of fluphenazine decanoate, and her son, who now lives in a group home and continues to attend his training centre, remains well.

Discussion

People with Down's syndrome are reputed to have a special talent for mime (Down, 1866), and imitation may be an aetiological factor in folie à deux (Gralnick, 1942*b*). However, we do not consider that the psychotic features observed in the son can be explained entirely on the basis of mimicry. His delusional thinking was well systematised, and even extended to unique symptomatology. Moreover, this case fulfils the diagnostic criteria of folie à deux suggested by Dewhurst & Todd (1956): positive evidence of intimate association; a high degree of similarity in the general motif and delusional content of the partner's psychosis; and the partners accept, support and share each other's delusional ideas. The case also has several other classical features: withdrawal from normal social interaction, dominance of the partner with the primary psychosis, and the sharing of the same bed. The reputed suggestibility associated with Down's syndrome may also be significant in our case, as highly suggestible individuals are said to acquire delusions more easily in folie à deux but also to shed them more readily (Dewhurst & Todd, 1956).

Acknowledgements

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Psychiatric Presentation of Creutzfeldt-Jakob Disease

A Case Report

A patient with Creutzfeldt-Jakob disease is described. Many alternative psychiatric diagnoses were considered, and the true situation only became apparent late in the clinical course.

The diagnosis of Creutzfeldt-Jakob disease is difficult, particularly in the early stages, because of the variability and complexity of its presentation. Often, therefore, no precise diagnosis or a wrong diagnosis is made during life (Matthews, 1983). Furthermore, the diagnosis remains essentially clinical, and the electroencephalogram (EEG) and computerised tomography (CT) scans, which are consistently abnormal in the later stages, are frequently normal for some considerable time after presentation (Kuritzky *et al*, 1980; Galvez & Cartier, 1984). Not surprisingly, psychiatric disturbances – which constitute the prodromal manifestations in 18–39% of cases (Will & Matthews, 1984; Brown *et al*, 1979) – are among the more difficult problems in differential diagnosis. The commonest early psychiatric symptoms of Creutzfeldt-Jakob disease include depression, emotional lability, asthenia, sleep disturbance and personality change (Will & Matthews, 1984). Such misleading pictures gain added importance in view of the precautions which ought to be observed over the handling of blood and other laboratory samples when the disease is suspected (Advisory Group on the Management of Patients with Spongiform Encephalopathy, 1981).

We describe in this report an unusual initial presentation which led to several psychiatric diagnoses being considered over the course of ten months before the features of Creutzfeldt-Jakob disease finally became apparent.

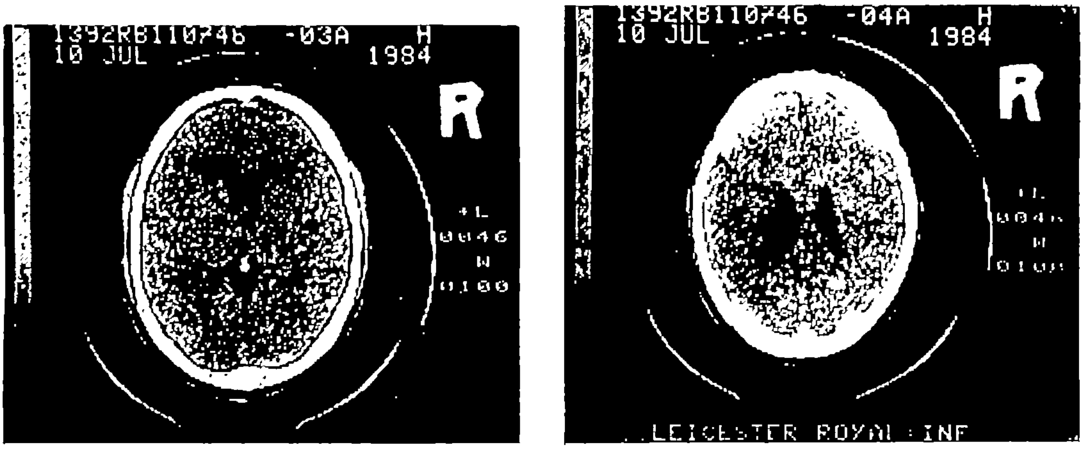
Case report

A 38 year-old antiques dealer was noted to be increasingly forgetful and disoriented from Christmas 1983. He had been a heavy drinker for many years, and particularly so in the preceeding 18 months, often consuming a bottle of spirits per day. He was also depressed because of growing tensions between himself and his second wife, centreing on his first wife's wish to send the two children of that marriage to live with him. There were episodes of his losing his way in his home town, and his business affairs had become increasingly chaotic.

When seen by a physician at the end of May 1984 he showed impaired memory and confabulation, but no focal neurological signs. A diagnosis of Korsakoff's psychosis was made, although depressive pseudodementia was considered a possibility. A brain isotope scan excluded the possibility of a cerebral tumour.

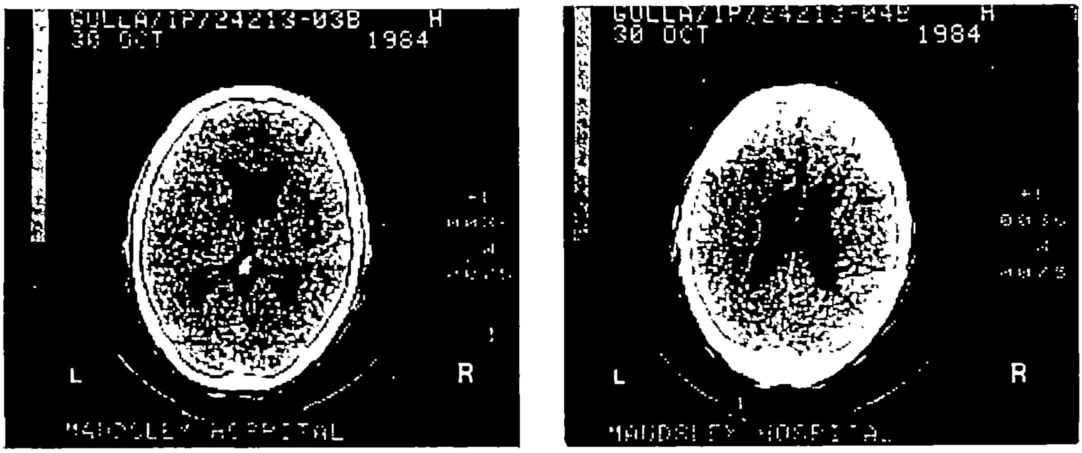
Despite discontinuation of drinking and a course of thiamine and antidepressants his symptoms worsened, necessitating admission to a psychiatric hospital in June 1984. On admission he was found to be disoriented and amnesic at times, but at others he was lucid and could give a good account of himself. No hallucinations or delusions were elicited. He was noted to be emotionally labile and gave approximate answers to questions, this leading to a diagnosis of hysterical pseudodementia or Ganser's syndrome. However, abreaction failed to clarify the picture further.

The following investigations were all normal: full blood count, syphilis serology, liver, thyroid and renal function tests, serum electrolytes, blood glucose, and serum B_{12} and folate. An EEG carried out on 7 August was difficult to interpret on account of restlessness and some involuntary



(a) (b)

FIG. 1 CT scan of patient taken in July 1984, 6 months after onset.



(a) (b)

FIG. 2 CT scan of patient taken in October 1984.

twitching of the limbs and head, but showed mixed theta and delta activity, the latter over the left temporoparietal region. These findings were attributed to a possible alcoholic encephalopathy.

In August 1984 he was observed in a neurological unit where he was found to be incoherent, agitated and with a short attention span. He could not give his name or that of his wife, and denied that he had children. He was capable of obeying only simple commands, and co-operated poorly with neurological examination. No abnormal neurological findings could be detected, however, apart from a pout reflex and shuffling gait. The CT was unremarkable (Fig. 1). Repetition of the EEG, again with difficulty, showed only minor and non-specific abnormalities.

During September he deteriorated markedly, becoming increasingly depressed and regressed in his behaviour. The favoured diagnosis remained Ganser's syndrome, possibly on the basis of a depressive psychotic illness. Refusal to eat and increasing dehydration led to placement on a treatment order; four ECTs were given over the course of ten days, but without improvement.

In October 1984 he was referred for a further diagnostic opinion, the diagnosis lying between alcohol-induced brain damage, Ganser's syndrome or some form of presenile dementia. At this stage he was perplexed, gazing vacantly, and spoke a slurred incoherent babble. Formal testing of cognitive functions was impossible because of his extreme distractibility. Neurological examination revealed gross apraxia, generalised myoclonic jerks, choreoathetoid movements and a lurching unsteady gait. There was generalised hyper-reflexia and down-going plantar responses. He was incontinent of urine and faeces.

Apart from a mildly elevated serum glutamic-oxaloacetic transaminase level, biochemical investigations, including serum copper and leucocyte enzyme studies (arylsulphatase A, β-galactosidase and hexosaminidase), were normal. CT scan now showed some definite enlargement of the ventricles (Fig. 2). The EEG showed two distinct pictures: when relaxed and relatively immobile, alpha rhythm was apparent bilaterally with little by way of slow activity; when more active and with the eyes open, however, there was evidence of gross diffuse cerebral dysfunction with little alpha and prominent slow or very slow wave forms maximal over the left frontoparietal regions. There were no definite spikes, sharp waves or regularly repetitive wave forms typical of Creutzfeldt-Jackob disease or subacute sclerosing panencephalitis. Lumbar puncture proved to be impossible on account of the patient's lack of co-operation. Further investigations by way of jejunal or rectal biopsy to exclude Whipple's or Kufs' disease were considered unwarranted in view of his deteriorating physical state.

He was returned to his base hospital with a probable diagnosis of Creutzfeldt-Jakob disease and treatment with amantadine (300 mg/day) was prescribed. Despite this he followed a steadily downhill course until his death in March 1985. During the last few months of life there were episodes of marked involuntary jerking movements, and also occasional tonic-clonic generalised convulsions.

Necropsy findings

The body was that of an emaciated man looking much older than his real age of 39 years. Extensive bed sores were present. The immediate cause of death was an inhalational bronchopneumonia; other internal organs were normal.

The brain weighed 1260 g. After fixation in formalin for 4 weeks, it gave the external impression of severe generalised atrophy with focal areas (frontal lobes and anterior left temporal lobe) of severe shrinkage atrophy. Large tissue blocks were taken of all cerebral lobes, the internal nuclei, the brainstem (in five places) and both cerebellar hemispheres. There was a profound spongiform encephalopathy with intense fibrous gliosis seen in the frontal, parietal, occipital and temporal lobes, in decreasing order of intensity. The internal cerebral nuclei, brainstem and spinal cord showed minor degenerative changes, chiefly gliosis, but no spongiform change. Minor spongiform change with gliosis was seen in the cerebellum.

In summary, the neuropathological findings confirmed the diagnosis of Creutzfeldt-Jakob disease and excluded other major causes of dementia such as Alzheimer's disease, Pick's disease, cerebrovascular disease, tumours and obstructive hydrocephalus. Unusual features were the marked focal atrophy, the only slight involvement of the cerebellum and the intensity of the fibrous gliosis. This last feature is in keeping with the fact that the course of the illness was longer than is commonly seen.

Discussion

Several confounding factors stood in the way of early diagnosis in this patient. Firstly, the history of excessive alcohol intake clouded appreciation of the significance of his early memory difficulties, leading to tentative diagnoses of Korsakoff's psychosis or other alcoholic encephalopathy. It remains possible that the presence of underlying brain pathology made him unduly susceptible to the effects of alcohol on memory, or conspired towards the escalation of his drinking prior to referral. Secondly, the difficulties in the marital situation could readily be construed as leading to depression or more directly to a state of pseudodementia. The variability of his cognitive impairment, not unusual in early Creutzfeldt-Jakob disease, reinforced this last impression, resulting in diagnoses of hysterical pseudodementia or Ganser's syndrome.

Of more general applicability were the difficulties raised by investigatory procedures. The finding of a normal CT scan well into the illness, and the inconsistent EEG changes, proved unhelpful in arriving at the correct diagnosis. Interestingly, both the later EEG and an earlier one showed abnormalities in the left temporal and frontal regions, correlating with the marked left temporal atrophy seen at autopsy.

The important general lesson must remain that a dementing process such as Creutzfeldt-Jakob disease needs to be seriously considered in any patient with progressive cognitive impairment, even though the clinical picture shows remarkable variability and the CT scan is normal. Effort directed towards careful serial EEGs and repetition of the scans could then be important in yielding clues to earlier diagnosis. A related point is the need to suspect such a process even in the presence of an alcoholic history when the course and clinical picture do not behave as would be expected in an alcoholic-related illness.

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Capgras Syndrome – An Unusual Case and Discussion of Psychodynamic Factors

A case of Capgras syndrome associated with extreme violent behaviour is described. The patient's psychodynamics support the belief that the Capgras delusion initially arises from an altered affective response towards one or more others and proceeds to intolerable ambivalent feelings. This ambivalence is neutralised through the imagined existence of doubles.

It is more than 60 years since Capgras & Reboul-Lachaux (1923) first reported the case of a patient with an 'illusion of doubles'. Their patient was a 53 year-old woman who had a delusional belief that people in her life had been replaced by doubles, and that she herself had a double. In an extensive review of the Capgras syndrome, Berson (1983) confirmed that the belief in the existence of doubles of others is frequently accompanied by a belief in the existence of a double of the self.

Originally the syndrome tended to be viewed as a single nosological entity, but it is now recognised that the central delusion is seldom seen in isolation. Merrin & Silberfarb (1976) reviewed 46 cases, 28 of which were diagnosed as schizophrenic. The majority of the 133 cases reviewed by Berson (1983) also had schizophrenia, particularly paranoid schizophrenia. The syndrome is also seen, however, in association with paranoid states and with both depression and mania.

Vié (1930) proposed that the Capgras syndrome was an organic psychosis. Many other authors have described the disorder in association with a wide range of organic brain disease; McCallum (1973) described five such cases. On the other hand, Kiriakos & Ananth (1980) examined 13 patients with Capgras syndrome, but found organic changes in only four.

Various authors have pointed out that the Capgras syndrome would be more appropriately named the Capgras symptom, i.e. a delusion of doubles. It certainly should not be regarded as a diagnostic entity in itself.

We describe here a patient with the Capgras syndrome associated with extreme violence. The relevance of some unusual features to the current psychodynamic theories of the Capgras delusion is discussed. It is not, however, suggested that psychodynamic factors actually cause a delusion; psychodynamic factors can account for the content of a delusion, but not its occurrence.

Case report

Mrs M was a 47 year-old divorcee, first seen by us when admitted to the forensic unit of a provincial psychiatric hospital in Canada. She was born in a small town, the oldest of four siblings. Her father worked as a motor mechanic and her mother as a housewife. There was no evidence of psychiatric illness in either branch of the family. The patient described her early childhood as relatively happy and uneventful until the age of eight, when her father started to drink heavily and to womanise. There was considerable disruption of her parents' relationship at this stage. Mrs M began to have nightmares, and at school was nervous and frightened of her teachers. Although her father continued drinking through her high school years, Mrs M appeared to be better adjusted, having a group of female friends and taking part in the usual adolescent pursuits, including dating. Educationally, she attained grade 12 and left school at the age of 19 to work as a typist and secretary.

At the age of 23 Mrs M had a six month courtship, during which she became pregnant. She had already experienced one psychotic breakdown by that time, and she stated that she married in order to avoid readmission to a psychiatric hospital. After her marriage Mrs M worked as a housewife. The couple's relationship was stormy; at the time of presentation Mrs M had been separated from her husband for five years and divorced for two years. She had a daughter and son by her husband, aged 23 and 16 respectively, and an illegitimate child who was born one year after her son and adopted away at birth.

Mrs M was first admitted to hospital at the age of 22, at the start of her illness. The onset was characterised by a conviction that her real father went off to Korea and that an almost identical double came into the house to take his place. She had 11 subsequent admissions to psychiatric hospitals, and had been an in-patient in another hospital for four continuous years prior to her transfer to our unit. Mrs M had been chronically deluded since her illness began and required a lot of psychiatric support, even when she was an out-patient. She had been consistently diagnosed as suffering from paranoid schizophrenia.

It was apparent from case-notes that Mrs M's psychopathology remained little altered throughout the years: the delusion of doubles had always been central. Her father, sisters, and husband, her two legitimate children, nursing staff, and other patients had all been replaced by doubles at one time or another. In the case of her son and daughter she reported the existence of multiple doubles who frequently interchanged at various times during their lives. She believed that she had the ability to distinguish between these various impostors by minor physical differences, such as height, skin texture and length of nose; this is typical of patients with the Capgras syndrome. Mrs M believed that some of the original people had disappeared permanently. She reported that her father had never returned from Korea and that her real daughter had been murdered at the age of seven. In contrast, most of the other doubles alternated with the original people they were impersonating, although sometimes the originals remained absent for long periods of time: "My son went off to explore the world when he was a toddler, only returning home a few years ago".

It has been suggested that the Capgras syndrome is only manifested towards people with whom the patient has a strong emotional attachment. In fact, in the case originally described by Capgras & Reboul-Lachaux (1923) this is not so, and our patient also attributed doubles to many people whom she knew only slightly. For example, she had only been on our unit for one month when she stated that one of the male nurses had a double, and she also believed that one of the authors had a double who had intentions of murdering her daughter. Mrs M also reported that on one occasion, ten years previously, she had seen a double of herself while in another psychiatric hospital. This experience only lasted a few moments and was not repeated, but she still believed in the existence of this 'self-double'.

A consistent and fascinating phenomenon was her delusion of part-replacement: she reported that she had been aware for some time that a number of her bodily organs had been removed during the night and replaced by those of other patients. This conviction started with her awareness of an altered internal sensation, and eventually she perceived this to be the result of replacement of her organs. She stated that this involved an operation through her vagina, thus enabling the organs to be exchanged without leaving a scar. Her vagina, uterus, ovaries, liver, heart, stomach, and brain had all been exchanged at some time or other. Occasionally messages arose from these organs, which often indicated their source of origin. Some months prior to her admission to our unit a voice came from her stomach saying that it was going to kill her son. She felt that this was not her own voice, and then suddenly realised that the stomach belonged to another patient and must have been exchanged for her own while she was sleeping.

Mrs M demonstrated the unpredictability of some chronic schizophrenic patients. She had been chronically deluded, despite high-dose depot injections of phenothiazines. Through the years she had intermittently expressed aggressive and homicidal threats towards her family and fellow patients in various hospitals. At home there were occasional episodes of violence; when her eldest son was two years old she injured him by throwing him against a wall, and on another occasion she attacked her sister with a knife. In hospital, aggressive behaviour had been an intermittent problem, although she had not inflicted serious injury on any occasion.

In the months before her admission to our unit, Mrs M had remained relatively well. Though still deluded, she appeared to be functioning at a socially acceptable level, displaying neither overtly aggressive or threatening behaviour. In the week prior to her transfer she became convinced that another patient on the ward (Mrs V) intended to murder her "daughter's double". Mrs M believed that it was Mrs V who had murdered the "original daughter" 16 years previously, and also believed (incorrectly) that Mrs V was about to be discharged from hospital. She came to the conclusion that the only way to save her daughter was to kill Mrs V. She ruminated on this for two days and then, while on a pass from the hospital, procured a large knife from a local shop, which she smuggled into the hospital. During the night she entered Mrs V's bedroom and, while the patient slept, she cut her throat, killing her outright.

At her trial Mrs M was found not guilty by reason of insanity. It was accepted that she felt the killing of this fellow-patient was justified and morally correct, on the basis of her delusional beliefs. She was ordered to be detained in a secure forensic unit.

Discussion

This case report sheds further light on psychodynamics of the Capgras syndrome. From our interviews with Mrs M it was clear that she had intense and ambivalent feelings towards a number of her family. She strongly resented her father's alcoholism and womanising, which disrupted the family stability. In her early twenties Mrs M experienced an early schizophrenic disintegration. It would appear that the intrapsychic regression that resulted allowed her to mobilise the primitive defence mechanism of splitting: the 'good father' was seen to be replaced by a double for whom she could more acceptably harbour aggressive feelings.

Such dynamics have been suggested previously to explain the Capgras delusion. Capgras himself, in relation to two cases, referred to feelings of strangeness (Capgras & Reboul-Lachaux, 1923; Capgras *et al*, 1925). Others (e.g. Berson, 1983) have expanded on this idea, suggesting that people become aware of changes in their usual affective responses to significant others and attribute these changes to changes in the other person. We might term this a 'projected depersonalisation'. Our patient demonstrated a similar phenomenon in relation to her somatic delusions. She reported initially experiencing a change in her internal sensations, and then assumed that these were not her own bodily senations: "There was a strange sensation from my vagina and I knew it was not mine, as mine had never felt like that. I then realized it had been exchanged during the night for somebody else's". The replacement of various internal organs appears to be in effect a partial Capgras delusion with respect to herself.

Mrs M also had hostile feelings towards her daughter. These may have been associated with her unexpected conception, which forced the patient into an unwanted marriage. By employing the defence mechanism of splitting, she was able to experience these feelings as being directed at an unrelated double. Presumably even this defence mechanism did not allay her anxiety, and she then projected her homicidal wishes onto a number of people, including her psychiatrist and Mrs V, all of whom she accused of wanting to murder her daughter. Mrs M then exhibited a gross reaction formation, destroying the person she perceived as wanting to harm her daughter. This had the incidental effect of sublimating her own homicidal impulses. Two pieces of evidence support this formulation. Firstly, Mrs M was in a state of calm on admission to our unit, only 12 hours after the homicide. She stated that there was no further threat to her children, even though she had believed that a number of other patients in her previous hospital had harboured similar feelings. Secondly, she had demonstrated projection of homicidal feelings previously, when she experienced the voice originating in her stomach which spoke of killing her son. On that occasion she denied ownership of the stomach and voice, ascribing both to another patient.

Conclusions

Apart from the presence of the Capgras syndrome, this patient resembles many chronic psychotic patients in long-stay hospital wards. It is worth bearing in mind that even chronic delusions can suddenly become invested with a high emotional charge, and that these patients can still act on their delusional beliefs. Chronicity reduces the likelihood of dangerousness, but it is not a guarantee against its occurrence.

This case supports the view that the Capgras syndrome is assocated with strongly held ambivalent feelings towards other people and is characterised by the use of the primitive defence mechanism of splitting, to defend against those feelings. Employment of splitting is probably secondary to an internal affective change, which is then projected onto the other person.

Acknowledgements

We thank Dr J. Lohrenz and Dr H. Merskey for their helpful comments and Gerry Stoner for her secretarial assistance.

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British Journal of Psychiatry (1987), **151**, 266

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Contents: Hostility and deliberate self-poisoning/Self-poisoning and suicide attempts by burning/No fixed abode/Late paraphrenia or the paraphrenias/Comparative trial of a new antidepressant/A comparative hospital survey of psychotropic drug prescribing/Compulsory detention of males of different ethnic groups/The psychological well-being of supporters of the demented elderly/Concept of mild endogenous depression/Tuberous sclerosis and the autistic syndrome/Delusional infestation in later life/Hysterical personality disorder/Capgras' syndrome in a patient with dementia/Trazodone-induced mania/Long-term psychiatric patients in the community/Erotomania and cerebral dysfunction/Italian psychiatry/Latitude and affective disorders.

Hostility and Deliberate Self-Poisoning

Sɪʀ: May I draw attention to another concept of 'hostility', additional to those covered by Farmer (*Journal*, May 1987, **150**, 609–614). This is the concept of irritability. Farmer supplies four descriptions of factors covering the construct of hostility: aggression, attitude of dislike, anger, and a predisposition to behave in a hostile or aggressive manner. The concept that we introduced (Snaith & Taylor, 1985) is that of irritability and, at our request, this was accepted as a new Medical Subject Heading, under the term 'Irritable mood', in the *Index Medicus*. In order that the term might be used in a standard way we supplied a definition: "Irritability is a feeling state characterised by reduced control over temper, which usually results in irascible verbal or behavioural outbursts, although the mood may be present without observed manifestation. It may be experienced as brief episodes in particular circumstances, or it may be prolonged and generalised. The experience of irritability is always unpleasant for the individual and overt manifestation lacks the cathartic effect of justified outbursts of anger".

Our interest in this state had been aroused by Pitt's (1968) observation of its frequency in the setting of post-natal depression, and our own research into mood disorder following childbirth led to the development of a brief self-assessment scale, the Irritability Depression and Anxiety (IDA) scale (Snaith *et al*, 1978). The article introducing that instrument described how we also had looked at the Buss-Durkee, the HDHQ, and other instruments but found that none fitted the state which we wished to delineate. The IDA scale has two irritability sub-scales (the outwardly-directed and the inwardly-directed irritability scales) and these, together with the depression and anxiety sub-scales, were published in the original article in order that they may be freely used – subject only to acknowledgement of copyright to *The British Journal of Psychiatry* (a convenient form with incorporated scoring device is available free of charge on application to myself). The first of our papers quoted above presents the validation of these sub-scales in four different studies and it was concluded that the outward-irritability scale is a homogeneous scale but that the inward-irritability scale is composed of two concepts; two of the four items composing it relate to suicidal self-harm and the other two items to a more general feeling of annoyance with oneself. If the IDA scale is to be used in research in the setting of suicide studies it is now advised that the inward-irritable scale is analysed by these two separate components.

Farmer has drawn attention to the diffuse concepts covered by different measuring instruments. We maintain that the IDA scale, with the modification just referred to, does provide a simple, acceptable set of measures and we hope that others will use it in suicide studies as well as all the other psychopathological and other states (such as post-natal depression, head injury, epilepsy, and drug withdrawal) in which the two aspects of irritability are a common and troublesome feature.

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Self-Poisoning and Suicide Attempts by Burning

Sir: The use of the term suicide 'epidemic' is fashionable. Both Farmer (*Journal*, May 1987, **150**, 609–614) and Zemishlany *et al* (*Journal*, May 1987, **150**, 704–706) use the term, in the former article to describe incidence trends in the general population, and in the latter to focus on the timing of three suicide attempts in a psychiatric hospital.

One should ask whether the 'epidemic model' of suicide is the appropriate concept for the phenomena being described. Strictly speaking, such a model would require the criteria of an epidemic, such as the identification of a 'host', a 'vector', and a 'mode of transmission' of a 'noxious agent', to be fulfilled.

Currently, there appears to be a resurgence of research interest in the 'Werther effect', referring to the phenomenon where newspaper publicity of suicides is apparently associated with an increase in suicide rates (Phillips, 1974). Phillips coined this term 200 years after Goethe's romantic novel *The Sorrows of the Young Werther* was banned in a number of centres in Europe for allegedly providing a model for imitative suicides. Some caution needs to be exercised in the labelling of increased suicide rates or of suicide clusters as "epidemics". Implied in the designation of a suicide "epidemic" based on imitation are preventative measures (such as the literary censorship that befell Goethe's novel), one being the suggestion that "educators, policy makers, and journalists may wish to consider ways of reducing public exposure to stories, both general and specific, about suicide (Phillips & Carstensen, 1986). While this recommendation may have superficial appeal, and may eventually be based on experimental evidence, the imitative propagation hypothesis to explain national suicide rate increases still awaits replicable proof.

Turning to suicide clusters, suggestible individuals, whether 'normal' young people or vulnerable adults in institutional settings such as hospitals or prisons, are more likely to be influenced by the basic psychological process of identification than by suggestion or imitation (Halasz, 1987) and the recommendation of "group sessions after a suicidal case in order to neutralise the identification with the act by suggestible patients" (Zemishlany *et al*) seems a clinical imperative.

However, this is a clinical recommendation, not in need of an epidemic model of explanation. Rather, it underlines the need for clinicians to recognise individual vulnerability in normal developmental phases, as occurs in childhood and adolescence, as well as in stress-induced regressed adult states brought about by illness, or dramatic changes in personal degrees of freedom.

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No Fixed Abode

Sir: We read with interest the recent report by Herzberg (*Journal*, May 1987, **150**, 621–627). We compared four groups of patients: urban fixed abode, urban 'no fixed abode' (NFA), and rural and semi-rural consecutive male psychiatric admissions, with 50 patients in each cell (O'Shea *et al*, 1983). This study also used retrospective case-note information.

Poor employment records and early parental separation were common in the NFA group; rural men generally live with others, the homeless tend to be single (NFA: married, 3; single, 40; separated, 6; divorced, 1; widowed, 0); age did not differentiate any of the groups; all groups shared 'chronic schizophrenia' and 'alcoholism' as the chief diagnostic categories; the strong impression was gained that alcohol-related difficulties were generally underestimated; patients in the NFA group were more likely to have legal records; standard diagnostic methods were hardly ever employed; physical pathology was common and widespread in all four groups; routine ancillary tests were not readily available; and most patients came from the lower socioeconomic levels of society.

Specifically, the physical findings among the NFA group were: eight men had chronic obstructive airways disease, mostly with acute exacerbations at the time of admission; three, one with a past history of cerebrovascular accident, were hypertensive, and all of these were on hypotensive drugs; the man with the stroke had a residual hemiparesis and epilepsy; two other cases had experienced alcohol-related convulsions; a fourth man's fits were found to be due to an inoperable glioma; one alcoholic was unable to walk because of peripheral neuropathy; one hypertensive patient had angina pectoris, another was diabetic, and a third required surgery for varicose leg veins; six men had anaemia of nutritional origin – one had undergone sub-total gastrectomy 30 years previously; one young patient had a marked strabismus; three men had had duodenal ulcers, two having received surgery for this; two had a history of hepatitis; and five had received anti-tuberculosis chemotherapy within five years of admission. Ten of the NFA patients were grossly obese.

The finding of a high physical morbidity among *both* urban groups is in keeping with other reported work (Feldman *et al*, 1974). The fact that all four groups shared this unenviable record is most likely related to the fact that they had psychiatric illnesses (Cutting, 1980; Sims, 1978; Sims & Prior, 1978).

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Late Paraphrenia or the Paraphrenias

SIR: Holden (*Journal*, May 1987, **150**, 635–639) perpetuates the confusion surrounding paranoid states in the elderly. A major contribution to this has to be the continuing reluctance to-diagnose schizophrenia arising for the first time in patients over the arbitrary age of 40 or 45.

Holden appears to be using the term 'late paraphrenia' in a very broad sense, including within the definition all cases of paranoia arising *de novo* after the 60th birthday. This is not in keeping with the clear definition of Kay & Roth (1961) he quotes.

I suspect that the 'organic' group could have been identified at the time of the initial contact, and it may be that the Gresham Ward Questionnaire is not sensitive enough to identify clinically important levels of poor cognitive function. It is interesting to note that the 'organic' group comprised 35% of the final sample, a figure not dissimilar to Post's "up to 30%". Paranoid symptoms are well known in patients suffering from organic brain syndromes (Ballinger *et al*, 1982) and the poor prognosis for organic brain syndromes has been known for some years (Roth, 1955; Christie, 1982; Blessed & Wilson, 1982).

I cannot agree with Holden that the differences between the concepts of "late paraphrenia" (Kay & Roth, 1961) and "persistent persecutory states of the elderly" (Post, 1966) have not been clearly resolved. The title of the Kay & Roth (1961) paper should leave one in no doubt as to their view of late paraphrenia. My own review of the term (Grahame, 1982) concluded that late paraphrenia is one of the schizophrenias, and this was supported by a later clinical study (Grahame, 1984).

Holden's final paragraph is, for me, the most confusing and I cannot see how the data presented support such a conclusion. Organicity is important in the genesis of 'persistent persecutory states of the elderly' but not of 'late paraphrenia', which in my view is as homogenous a clinical syndrome as any known to psychiatry. After all, there is no dispute about schizophreniform psychosis secondary to, say, trauma in young patients and the functional schizophrenias. The fact that some patients with late paraphrenia subsequently develop an organic brain syndrome should not necessarily lead one to say that the late paraphrenia was a symptom of an organic brain syndrome. Just over 40% of patients in the Blessed & Christie (1982) study had an organic brain syndrome, and they comment that their findings "again lend little support to the idea that functional illness in old age is the harbinger of senile or arteriosclerotic psychosis". Inspection of Table I of Blessed & Wilson (1982) shows what they consider late paraphrenia to be.

Finally, had Holden interviewed his patients using the Geriatric Mental Status Interview, he may well have found more patients reporting first rank symptoms which, in turn, may have had a significant effect on his final groupings.

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Comparative Trial of a New Antidepressant

SIR: There are 16 tricyclic and related antidepressants currently on the market. It seems likely that most psychiatrists use only a small number of them routinely. Before any more new antidepressants are added to this list, how insistent should we be in requiring that standardised methodologies be employed when testing their efficacy?

Levine *et al* (*Journal*, May 1987, **150**, 653–655) showed that fluoxetine was as effective an antidepressant as imipramine. However, their study did not include a placebo control to establish the relative efficacy of both of these drugs. The reason for this not uncommon omission is that imipramine is considered to be of proven efficacy and, therefore, a reliable benchmark against which new antidepressants can be measured. How justified is this assumption?

In two extensive reviews of the literature, tricyclic antidepressants were not found to be superior to placebo in 35% (Morris & Beck, 1974) and 41% (Thomson, 1982) of studies. Swallowing an inert placebo leads to a significant improvement in between 30% (Morris & Beck, 1974) and 50% (Medical Research Council, 1965) of those suffering from depression. Mild side-effects (which fluoxetine possesses) are associated with greater drug success, while zero or severe side-effects are associated with less success (Brune *et al*, 1962). The advantage of tricyclic antidepressants over placebo is significantly less when an active (atropine) placebo is used instead of an inert substance (Thomson, 1982). Thirty per cent of depressed patients do not respond to tricyclic antidepressants (Medical Research Council, 1965): in the Levine *et al* study this figure was approximately 50%.

Thus the universal and invariable efficacy of tricyclic antidepressants has yet to be demonstrated. Therefore, imipramine and other 'standard' antidepressants should not be used as the sole agent in comparative studies. Levine *et al* did not use a placebo control, so we cannot be sure whether the improvement in depressive symptoms was due to a specific psychotropic, or an active placebo, effect.

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Comparative Hospital Survey of Psychotropic Drug Prescribing

SIR: Muijen & Silverstone (*Journal*, April 1987, **150**, 501–504) showed that the hospital (hospital A) with the lowest prevalence of polypharmacy was the only one with an associated psychopharmacology unit. We have compared the authors' results with our acute in-patient population. Our data is obtained from a unit in our hospital, the population of which can be compared to that of the authors' hospital C. Ours is a teaching hospital, but has no association with any psychopharmacology unit.

Seventy-eight patients admitted in the past year were included in the study. All psychotropic drugs given on the seventh day of admission were recorded from case records; we presumed that a final diagnosis was reached and medication was started by one week after admission. The rest of our methodology is similar to that of the authors' study.

The results showed that more than one psychotropic drug was given to 49 patients (63%). This figure is close to the authors' figure for hospital A. More than two drugs were given to 23 patients (29%) and more than three drugs were given to 8 patients (10%). One patient received no drugs, one drug was given to 23 patients (29%), two drugs were given to 28 patients (36%) and three drugs were given to 18 patients (23%). Benzodiazepines were given to 22 patients (28%). More than one antipsychotic drug was given to 21 patients (27%), whereas antiparkinsonian drugs were given to 17 patients (22%). The last two figures are lower than the authors' hospital A figures. We conclude that drug prescription is more influenced by general training than by any association with a psychopharmacology unit.

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Compulsory Detention of Males of Different Ethnic Groups

Sir: We wish to comment on the article by McGovern & Cope (*Journal*, April 1987, **150**, 505–512).

Since 1976 the Mersey Region has had a secure unit, initially in the form of an interim facility and since 1983 as a regional secure unit. The unit accepts referrals from special hospitals, prisons, remand centres and other psychiatric hospitals. Our clinical impression was that admission rates for patients of West African and West Indian descent were in proportion to their numerical size in our catchment population. In the light of the above study we examined our data relating to admissions for a defined part of our catchment area, in which patients in the category defined above make up 6% of the population.

Since 1976 there have been 93 admissions to our unit from the defined area (78 male and 15 female): 14 patients were of West African or West Indian descent. Of the 93 admissions, 63 were offenders and 30 non-offenders. Patients of West African or West Indian descent were over-represented among offenders (12/63) but not among non-offenders (2/30).

Our figures support the findings of McGovern & Cope. We are unable to account for these differences, but can say that our clinical experience does not support the suggestion that patients of West African or West Indian descent are more disturbed, violent, or difficult to follow-up in the community. We are currently planning a survey in a larger sample including special hospital patients.

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The Psychological Well-Being of Supporters of the Demented Elderly

Sir: Two things puzzle me about the paper by Eagles *et al* (*Journal*, March 1987, **150**, 293–298). Firstly, it seems little short of miraculous that the 1980–1982 MSQ had 100% sensitivity, specificity, and predictive values with the 1983–1984 psychiatric assessment of the presence of dementia in the "dependants" of the first 80 supporters agreeing to the interview. It is not clear whether this represents a startling improvement in psychiatric epidemiology, or whether there is some more prosaic reason.

Secondly, why were the 'non-demented' elderly people in this survey being "supported" at all? It is likely from the way they were apparently recruited that they were having problems, but since the vast majority of 'non-demented' elderly people look after themselves perfectly well, there must have been something else going on here. This, rather than the explanations offered in the discussion, may explain why GHQ scores did not differentiate between the groups.

It is a pity that such elementary points of clarification could not be dealt with before the paper was published.

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Sir: Macdonald's puzzlement stems from his having misunderstood the methodology of our study. The MSQ carried out in 1980–82 allowed identification of a group of elderly patients who were living at home and were possibly demented, since their MSQ score was 8 or less at that time. These 205 subjects were matched for age and sex with 205 subjects scoring 9 or 10 on the MSQ. These subjects were then re-interviewed in 1983–84 with the aim of, among other things, assessing the follow-up validity of the MSQ. For the purposes of the present study, the 1980–82 MSQ was ignored and the ratings of the patients are those made during the psychiatrist's visit in 1983–84.

With regard to Macdonald's second point, the 'non-demented' people in this survey were not necessarily being "supported". We make it fairly clear that the criterion for their inclusion was that they were co-habiting with someone who was willing to complete our questionnaires, and that this co-habitee would bear the burden of support, if such support were necessary. I cannot understand Macdonald's contention about these patients that "from the way they were apparently recruited they were having problems". They were recruited solely on the basis that they were 'non-demented' elderly patients whose co-habiting relative was willing to answer questions about what problems, if any, they encountered in "supporting" the elderly person.

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Concept of Mild Endogenous Depression

SIR: Unfortunately, Snaith's essay (*Journal*, March 1987, **150**, 387–393) is representative of most discussions about the use of the term 'endogenous' depression, being just another example of defining words by other words at the same level of abstraction.

When multiple clinical items of depression are aggregated, the resulting scale of categories can be arranged as a hierarchy. The hierarchical arrangement provides a clear idea of what is contained in the total scale score of the individual items. The most simple model for evaluating hierarchical aggregations is the Rasch model, because items here will obtain equal weights if they fulfil the model (Bech, 1981). Applying Rasch models on the universe of items defined by the Melancholia Scale and the Newcastle Scales (Bech *et al*, 1983), we have found one dimension of severity of depression and two dimensions for the diagnostic type of depression (Bech & Allerup, 1987).

In total, 11 items constituted the dimension of severity, which in increasing hierarchical order are: difficulties in carrying out usual work; lack of interests and pleasure; reduced sleep; more apprehensive or irritable than usual; more emotionally introverted than usual; difficulties in concentration; more tired than usual; self-depreciation or guilt feeling; pronounced inertia in conversation; clear motor retardation; and suicidal thoughts or impulses.

In relation to the two diagnostic dimensions, we found that five items constituted endogenous depression: persistence of clinical picture; weight loss; early waking; worse in the morning; and distinct quality. Another five items constituted reactive depression: duration of current episode; somatic anxiety; character neurosis; reactivity of symptoms; and psychological stressors.

According to these dimensions we can classify patients into minor or major depression and diagnostically into endogenous or reactive depression.

The state of anhedonia described by Snaith is nothing more than mild endogenous depression. 'Anhedonia' is traditionally used to describe the borderline personality who suffers from a painful lack of vitality based on a narcisistic distance from other people. According to Vanggaard (1979): "Not infrequently this anhedonia is called 'depression', and this is erroneous. An anhedonic state is a far cry, phenomenologically and theoretically, from a depressive one, as different as the general personalities of persons harbouring such states, if the terms are properly understood and used".

From a psychopharmacological point of view anhedonia should be treated with neuroleptics, whereas mild endogenous depression should be treated with antidepressants. Patients who do not respond to antidepressants should be diagnostically reconsidered, as they might be borderline cases.

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Tuberous Sclerosis and the Autistic Syndrome

SIR: I was surprised to read that Lawlor & Maurer (*Journal*, March 1987, **150**, 396–397) believe tuberous sclerosis (TS) to be a rare cause of the autistic syndrome. Parents of children with TS have known otherwise for some years, and child psychiatrists, paediatricians and others have been made aware of it more recently. Lawlor & Maurer fail to point out that the autistic syndrome is closely linked to the early occurrence of *infantile spasms* (Kolvin *et al*, 1971, Hunt & Dennis, 1987) and, much less so, to other forms of epilepsy.

The point I wish to make is that TS is not as rare as was once thought. In the Oxford Region in 1984, the prevalence was 1 in 15 400 for children under five. Children with TS do present at departments of child psychiatry from time to time, and I support the conclusion of Lawlor & Maurer that a high index of suspicion should be maintained when confronted with an autistic child who has fits – but also, I would say, when assessing hyperkinetic, aggressive, and sleep disordered children with fits. Furthermore, in my experience, the diagnosis of TS has been made in children who show the behavioural disorders but who have never had a fit.

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Delusional Infestation in Late Life

SIR: Renvoize *et al* (*Journal*, March 1987, **150**, 403–405) provide a useful review of the literature, including the German literature, in reporting their interesting patient whose delusions of infestation occurred during the course of a dementing illness. We have been interested to find that delusions of infestation are seen quite commonly among the patients referred to our psychiatric services for the elderly and we recently reviewed seven cases that presented between 1983 and 1986. There were two men and five women. Their ages ranged from 69 to 76 – a little above the boundary of 'late middle age', the age-group from which patients are most frequently reported, but not very old (patients aged 75 and above constitute about three quarters of our referrals).

All seven were found to be depressed. None were seriously demented, although in three there was clear evidence of some organic cerebral impairment and in only one could we be sure that there was no such pathology. In two patients the delusions of infestation developed in the context of guilt at not maintaining cleanliness in the home after the death of a spouse. In three, previous neurotic preoccupation with cleanliness or 'phobia' of 'creepy-crawlies' had become delusional in the setting of severe depression, and in the last two the delusion was part of a systematic belief that the body was changed and degenerating.

In most of our patients the delusions appeared when powerful change of affect occurred with mild organic change. This is a potent combination in the genesis of delusions (Hay *et al,* 1974). Successful treatment of the mood disorder has led to resolution of the delusions of infestation in the six cases we have known longest. Thus, in this age group delusions of infestation may be less persistent and pernicious than those of the monosymptomatic psychoses that are reported more commonly in younger middle-aged patients. The presence of some organic impairment should not detract from the treatability of the condition through an appreciation of the mood disorder.

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Hysterical Personality Disorder

SIR: Thompson & Goldberg (*Journal*, February 1987, **150**, 241–245) question the validity, reliability, and utility of the diagnosis of hysterical personality disorder. Though such issues are properly raised about a vexing construct, I believe the authors' gloomy conclusions may not be wholly warranted.

Based on a retrospective review of case notes, Thompson & Goldberg observe that the diagnosis "is frequently made without the core features being present". Their finding may tell us less about the construct, hysterical personality disorder, than it does about diagnostic practice and documentation at Withington Hospital in 1975. Had the authors tabulated the "core features" present in the case notes of patients with another diagnosis we would be better able to judge where the problem lay.

This matter is of considerable importance because the authors fear that the diagnosis of hysterical personality disorder may be used "to label those patients who are perceived as hostile, difficult, and uncooperative, with the result that the doctor is distracted from recognising an underlying diagnosis". Prominent among such proposed underlying conditions is 'primary affective disorder', which the authors claim that McHugh and I found (Slavney & McHugh, 1974) in a sample of patients with the diagnosis of hysterical personality disorder. I feel constrained to point out that what we noted was not 'primary' affective disorder, but rather the frequent occurrence of depressed mood and self-injury, phenomena which we interpreted as the responses of self-dramatising and emotionally labile people to stressful circumstances (i.e. as 'secondary' to events such as romantic disappointment). What was 'underlying', then, was the personality disorder, the recognition of which a psychiatrist should not be distracted from by the presence of affective symptoms. The validation of the trait of self-dramatisation has yet to be accomplished, but there is some support (Slavney & Rich, 1980) for the existence of emotional lability as an actual attribute of patients who receive the diagnosis of hysterical personality disorder.

Finally, the authors observed low inter-rater reliability in the assessment of hysterical traits based on brief videotaped interviews. The diagnosis of personality disorders in the clinical setting, however, depends to a great extent on the description of patients by those who know them well, and recent developments in the standardised assessment of traits as rated by informants gives hope that diagnostic reliability can be improved. Using the scale devised by Mann *et al* (1981), for example, Ballinger (1987) has recently shown acceptable inter-rater reliability in the rating of traits (including hysterical ones) for a sample of patients with mental handicap. As well as this, there is evidence to suggest that judgements about the central trait in the hysterical cluster – self-dramatisation – are not greatly influenced by the sex of the rater or by the sex of the patient (Slavney & Chase, 1985).

Thompson & Goldberg call our attention to important matters, but not to lost causes.

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SIR: Our conclusions are far from gloomy. We draw attention to a group of patients whose response to illness, whether physical or psychiatric, is characterised by difficult, demanding, and aggressive behaviours. Our findings should encourage clinicians to properly examine such patients for underlying illness and not attribute their behaviours and symptoms exclusively to personality disorder. We contend that the recognition of such underlying illnesses in patients affords a greater opportunity for effective treatment than would be the case if they were to be managed simply as 'personality disorders'.

We concede that our data reflects the clinical practices at Withington Hospital, but we have worked in a variety of hospitals in the UK and would describe the practices at Withington Hospital as similar and certainly no worse than those in other hospitals. One author (DG) has also worked in several hospitals in the United States and observes that the particular clinical practices there are comparable. In Slavney & McHugh's (1974) paper comparing hysterical personality disorder with control patients, the only criteria that referred to the mental state and significantly distinguished between the two groups were the items "dramatic" and "change of therapist". The latter suggests those same behaviours that characterised our index group. Core features such as provocativeness, seductiveness, and lability of mood were not significantly different.

The theme which we consider central is the relative role of illness and personality. The evidence suggests that personality traits may be exagerated by major stress, and that may include an underlying physical or psychiatric illness. The personality disorder simply colours (or sometimes clouds) the presentation as seen by the doctor. To say otherwise ignores evidence amassed from follow-up studies in a variety of hospitals, almost all of which demonstrate a high level of unrecognised illness at the index episode (Slater, 1965; Reed, 1975).

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Capgras' Syndrome in a Patient with Dementia

SIR: I describe a further patient (Kumar, *Journal*, February 1987, **150**, 251), with the additional feature of prominent visual hallucinations.

Case report: An 86-year-old man was referred with a six-month history of believing his wife was replaced by an imposter every afternoon – during which time his real wife (78 years old) was soliciting young men in local pubs. At the same time he regularly saw up to four young women in the house. He varied between being annoyed by their intrusion and being friendly towards them when he believed they had come to help with the house-work. On his wife's 'return' he would express anger at her behaviour. After some weeks he began making sexual demands of his wife, stating that he was entitled to his 'share'. He was then distressed by his failure to achieve an erection. Since the couple's sex life has been in abeyance for years his wife made clear her fears for his physical health, which made him even angrier. He threatened her with a stick on a number of occasions. One night he tried to cut off her pyjamas with a pair of scissors, and this led to his referral.

His wife described a three-year gradual decline in memory. Premorbidly, he was said to be a self-contained suspicious man, particularly distrustful of women. There was no psychiatric history. Two months prior to the onset of this episode he suffered a chest infection during which he suspected his daughter of trying to poison him. These beliefs resolved completely following antibiotic treatment. On mental state examination, the features were delusional misidentification, delusions of jealousy, visual hallucinations, and impairment of short-term memory without impairment of consciousness. He had no insight, and expressed anger towards the "imposter". He was quite deaf, but otherwise physical examination was normal. Routine blood tests were normal. A diagnosis was made of a paranoid syndrome secondary to dementia, probably of Alzheimer type. He was treated with thioridazine, up to 100 mg/day, with resolution of the Capgras syndrome and visual hallucinations.

This patient is of interest for several reasons. Firstly, he illustrates Enoch's (1979) theory of ambivalence towards the loved object revealing itself in the Capgras syndrome, but differs in that the sexual demands followed the onset of the Capgras syndrome rather than that the rebuffed demands contributed to the development of the syndrome. One could speculate that a lesion in the right occipito-parietal area might result in both visual hallucinations and Capgras syndrome following the suggestion (Haymans & Abrams, 1977) that facial non-recognition causes the Capgras syndrome. However, I would agree with the observation (Weston & Whitlock, 1971) that the Capgras syndrome is the exact antithesis of facial non-recognition, there being no difficulty in the patient recognising the imposter as a replica of the person concerned. Sensory deprivation (Gluckman, 1968) has been mentioned as relevant; and this man's deafness, in conjunction with living close to a noisy girls' school, might be important aetiological factors.

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Trazodone-induced Mania

SIR: Knobler *et al* (*Journal*, December 1986, **149**, 787–789) report on three cases of trazodone-induced mania. Two of their patients had DSM–III mixed bipolar disorder, and the other had DSM–III recurrent major depression. I would like to report a case of trazodone-induced mania in a patient with a single episode of major depression.

Case report: A 58-year-old female widow met DSM–III criteria for major depression with melancholia (296.23) (single episode). She had suffered from depression for two years. Examination revealed depressed mood and affect, hopelessness, anhedonia, transient suicidal ideation, loss of energy and interests, loss of appetite and weight, and insomnia. She had no previous affective or other mental disorders. Her brother was a schizophrenic and committed suicide. Treatment (out-patient) with trazodone (50 mg t.i.d.) was started. In two or three days her mood was less depressed, and she felt better. But after one week of treatment, she became very talkative, quarrelsome, sleepless, hyperactive, and restless. She was spending money uncritically and frequently changed her clothes. She also thought that her grand-daughter had been poisoned by her son-in-law. She was admitted to hospital.

On admission she showed pressure of speech, psychomotor agitation, and mood-incongruent delusions. Trazodone was stopped, and treatment with haloperidol (1.5 mg t.i.d.) was started. After one day, her delusions completely disappeared and her mood was less elated. On the next day she was transferred to a general hospital because of paroxysmal tachycardia. There she was found to have hyperthyroidism and ischemic cardiomyopathy. She was seen after one month as an out-patient: her mood was stable and haloperidol treatment was stopped. After three weeks she was seen again, and she was in remission.

A causal connection between trazodone treatment and the manic state in this patient seems obvious. Her manic episode started only a week after trazodone was prescribed, and this was her first manic episode. She received trazodone without any other psychotropic medication. After the discontinuation of trazodone the manic episode remitted in less than ten days.

The mood-incongruent delusions in this patient are confusing. They were of short duration (about three days), but represent a difference between the patients described by Knobler *et al* and this patient. The patient's family history of schizophrenia may be relevant. The patient's hyperthyroidism could have some aetio-pathogenetic role in the depressive episode, but this is less likely in the sudden mood change to mania. Our patient was probably hyperthyroid for more than one year (according to the medical history and findings). She had had no mood up-swings until the trazodone treatment began. If the manic state was triggered by hyperthyroidism one would have expected its manifestation earlier.

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Long-Term Psychiatric Patients In The Community

Sir: Kathleen Jones and her co-workers (*Journal*, November 1986, **149,** 537–540) paint an optimistic picture of care in the community in York when documenting the fate of 50 long-term psychiatric patients discharged into the community. They suggest that similar surveys in other areas might have less favourable results.

We conducted a follow-up of all patients who had been in Banstead Hospital continuously for at least 2 years and were subsequently discharged into the community between 1970 and 1981. There were 25 such patients.

The majority had led surprisingly stable lives since discharge (average 6.25 years). Readmission rates to hospital were low. All but one patient remained out of hospital, and only four others had been readmitted for brief periods. Half the patients had lapsed contact with the psychiatric services, but of these, half still had regular contact with their GPs and received depot medication. One patient was unfortunately in prison for aiding and abetting rape, and four had died of natural causes at an advanced age.

We assessed the quality of life of the patients by interview with the patients and their carers using a semi-structured interview. The patients fell into two groups of roughly equal numbers. The first group was made up of those with few if any symptoms, a low dependence on psychiatric services, and an ability to lead active independent and generally contented lives. These were primarily schizophrenic patients living in high quality group homes. The second group consisted of those with some degree of symptomatology, receiving a higher degree of support from psychiatric services and having a tendency to live more passive, dependent, and somewhat discontented lives.

Our findings support the view that community care with appropriate resources is a viable option for selected long-stay hospital patients.

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Erotomania and Cerebral Dysfunction

Sir: *Case report:* Recently a 30-year-old right-handed patient presented with erotomania of the de Clérambault's type and somatic delusions. Two years ago he met a 16-year-old salesgirl, felt it was love at first sight, and thereafter believed that she communicated a mutual love by means of silent gestures, ringing in his ears, telepathic messages, chain letters, and cars driving past him; he believed that she attempted to make him jealous by having sexual relationships with countless men. He had previously held similar beliefs about two other women. Over the same period he had somatic delusions, experiencing a number of physical symptoms that he attributed to the influence of this woman.

The patient had depressed mood and a large number of neuro-vegetative symptoms when these delusions were at their height, but had recovered by the time he was seen. He had made a suicide attempt in the past, had a history of heavy alcohol abuse ending four years previously, and had a family history of affective disorder. On examination he was mildly elated with rapid, pressured speech; verbal fluency was poor, he was unable to do an alternating hand sequence, and he showed verbal-motor dissociation on the right with the Luria hand sequence. The EEG showed left temporal abnormalities. The symptoms abated slightly with pimozide; the patient refused lithium and carbamazepine.

Erotomania has been reported with frontal and left temporal lobe dysfunction with secondary mania (Signer & Cummings, in press); the latter has shown a particular association with delusions (Sherwin *et al*, 1982). The EEG showed left temporal, and the cognitive examination left frontal, dysfunction. With appropriate examination and investigation more patients with erotic delusions may be shown to have organic abnormalities requiring specific treatment.

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Italian Psychiatry

SIR: Most articles dealing with Italian psychiatry in the past five years have considered the public sector side of psychiatric intervention, while omitting any comment on private practice (Mosher, 1982; Becker, 1985; Jones and Poletti, 1985, 1986; Tansella, 1985).

The regulated, public sector is carefully analysed by the above authors, but it may be helpful to underline a trend towards a further splitting of psychiatric practice. In the public sector there is an area, i.e. acute control/therapy, which can be considered as strictly medical (it includes psychiatric wards in general hospitals and mental health centres); now, a second area is under development, i.e. long-term assistance/surveillance, which is to a great extent de-medicalised. This area includes day hospitals and criminal psychiatric hospitals.

In the acute control/therapy area psychiatrists see acute patients who respond to treatment. In the long-term surveillance/assistance area there are mental health workers, most of whom are psychologists and sociologists, who stress their rehabilitation role in relation to patients' (possible) readmission into society. There is a long-running dispute between the two groups about the relevance of medical or sociological attitudes towards patients with mental disorder. Psychologists (who use the language of psychoanalysis and anti-psychiatry) are trying to take over, particularly in those regions, like Veneto, where there is already a good level of long-term assistance. The gradual worsening of public health standards, and the fact that psychiatry is poorly regarded in medicine, do not allow a debate and a comparison of results on purely technical grounds.

Moreover, in the private de-regulated sector psychotherapies and psychopharmacological treatment have flourished in an uncontrolled way. Here too the field is splitting asunder: the former being the province mainly of psychologists, the latter of psychiatrists. Patients have great difficulty in finding reliable help in the private sector, (although, of course, they pay for services) as recent legal actions testify. While admission to private clinics is still limited (at least in Northern Italy), such interventions are likely to increase because of scant facilities for in-patients in the public sector. A recent analysis (CENSIS, in press) shows that among five clusters of public services only one is judged as being efficient, and this is limited to certain regions only (Toscana, Umbria, and some areas of Veneto).

The public sector is therefore supported by the private sector, according to that type of mixed economy which allowed the Italian economic miracle, but which for psychiatry continues to raise serious doubts about any future improvement in standards.

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Latitude and Affective Disorders

SIR: I am interested in the effect of latitude on the expression of the genetic trait for bipolar disorder. Inspection of a world atlas highlights the relatively few English speaking communities within the tropics. Having practiced in Mackay, Northern Queensland, for four years, I am sure that chronic mania is considerably commoner than in the temperate latitudes, e.g. London, where I previously trained and worked.

My impression is of a 'shift to mania' in people who have the genetic diathesis for bipolar illness. I see less chronic depression (although that must be a tenuous observation) and a striking increase in the severity and duration of mania. Hence, chronic mania and manic stupor are increased. I would be interested in comments on this from those in various different latitudes, especially colleagues in Hawaii, which is equivalent in latitude to Mackay but in the Northern hemisphere.

It may be helpful if I put an order of magnitude on the occurrence of chronic mania (my *ad hoc* criterion for chronic mania is a minimum duration of 6 months, but most of the patients have illnesses of at least a year's duration). I have seen a dozen cases in the first four months of this year (the catchment area I deal with has a population of approximately 100 000). Included in those dozen cases would be two of manic stupor, or at least partial stupor.

It would be interesting to know if anyone can elucidate the possible connection between this apparent increase in the incidence of mania in the tropics and the old expression, "going troppo".

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British Journal of Psychiatry (1987), **151**, 277–281

Book Reviews

Editor: Sidney Crown

The Heredity of Behavior Disorders in Adults and Children. By S. G. VANDENBURG, S. M. SINGER and D. L. PAULS. New York: Plenum. 1986. Pp 301. $35.00.

Reliable psychiatric diagnoses, new mathematical models and recombinant DNA technology have followed hard upon one another in the past decade to make psychiatric genetics not only respectable, but suddenly big business. Huntington's disease and narcolepsy have been tracked down on the genome and, even as this review is written, reports are emerging of the genes for manic depressive psychosis on chromosome 11, Tourette syndrome on chromosome 18 and Alzheimer's disease on chromosome 21. No-one could dispute the need for a contemporary textbook on psychiatric genetics.

The aims of this jointly-authored book are clearly set out after a foreword by Dr Elliott Gershon: these are to review the evidence for the importance of genetic factors in mental disorders, to show how this can further our understanding of their epidemiology, and at the same time to emphasise that genetic influences do not spell immutability. The text is written consistently well. However, in Chapter 2 ('The Diagnosis of Psychopathology') a small alarm bell sounds: they are evidently not entirely comfortable with the medical model. Such ambivalence is no bad thing in the right context, but it puts the authors of a book on genetics in a curious position. Take the title, for instance: what is meant by "behaviour disorders"? It is a term the authors choose not to define, despite their preoccupation (often justified) with operational definitions throughout the text. With a single exception (epilepsy), all the disorders they consider are better known as psychiatric disorders.

Chapter 3 reviews genetic causes of mental retardation and is used to introduce concepts in cellular genetics, such as mitosis and mosaicism. Chapter 4 is potentially the most important, and deals with the methodology of psychiatric genetics. The advantages and limitations of twin, family, and adoption studies are discussed capably, as is the importance of rigorously defining phenotypes and guarding against aetiological heterogeneity. The concepts of linkage and recombination and the use of restriction fragment length polymorphisms to detect linkage are explained clearly. Multifactorial models are rightly explained at length. This comprehensive section does miss one or two basic building blocks, nonetheless. The definition of fundamental terms of measurement in population genetics is, surely, mandatory in a text like this. Their inclusion might also have served to tighten up the authors' own use of these terms, which is occasionally lax (what are "lifetime rate" and "incidence of risk"?)

The genetics of epilepsy are tackled next. Here, the authors seem to be out of their depth. We read of what "medical experts believe", and are given unabashed citations from textbooks of general neurology. Chapter 6, on schizophrenia, sees the authors back on dry land, but with legs still shaky. A workmanlike account of genetic studies leaves largely unexplored the potential significance of "schizotypical" (*sic*) personality and schizophrenia spectrum disorder. In the authors' own words: "For a thorough review of the evidence for a genetic basis for schizophrenia, see Gottesman and Shields (1982)".

However, there follow good, clearly argued chapters on alcoholism, criminality, and a variety of childhood disorders. Those concerning specific reading disabilities, stuttering, and Tourette's syndrome are particularly good, reflecting the authors' own interests. Elsewhere there are sections on affective disorders and the neuroses. Alzheimer's disease warrants two poorly-informed pages.

In summary, this book is a brave attempt to hit a fast-moving target. There are few references more recent than 1983. Joint authorship ensures a uniformly readable style, but betrays some gaps in the authors' collective expertise. Might a book like this benefit from discussion of related issues such as genetic counselling and, since the orientation is towards behaviour disorders, consideration of the genetics of some aspects of normal behaviour, such as personality? University libraries would reasonably be expected to own a copy, but interested individuals might best be advised to wait; with a growth area like genetics, it is a fairly safe bet that there will soon be more than one book to choose from.

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The Nazi Doctors: A Study of Psychology of Evil. By ROBERT JAY LIFTON. London: MacMillan. 1986. Pp 561. £17.50.

Robert Lifton is justifiably renowned for the scholarly attention and ethical commitment he has given to the subject of man's confrontation with the extremes of human condition. His work on the Hiroshima survivor, the Vietnam war veteran, and the threat of massive genocide through the deployment of the nuclear bomb testify to his success. In turning to the Holocaust, and particularly to the contribution of the Nazi physician therein, Lifton has tackled, arguably, an episode unparalleled in history – the vigorous attempt to exterminate an entire people, based on racial ideology. Although we are provoked to anguish over the question of how a previously civilised state could resort to such barbarity, Lifton's chief purpose is to shed light on what motivated the Nazi physician to forgo his ethical and healing role and assume the role of killer.

Two principal themes emerge with brilliant clarity. The first is uncomfortably linked to the psychiatric profession. The eugenics movement in the early 20th century found an eager audience in Germany, and meshed neatly with Hitler's racial doctrines. It was the likes of the psychiatrist Alfred Hoche (in an influential book co-authored with the jurist Karl Binding) that provided a 'scientific' foundation upon which the Nazi regime could promulgate, as early as July 1933, a law permitting the sterilisation of the chronically mentally ill and the intellectually retarded.

'Mercy' killing of these 'lives unworthy of life' was the logical next step. Soon after the start of the war the gas chamber was installed in a number of psychiatric hospitals, and the psychiatrist came to assume the role of executioner. Over the next two years, he was to acquire an expertise and proficiency that resulted in about a quarter of a million deaths. Thus, when the 'Final Solution' was devised in 1942, the machinery of genocide was ready, and the physician was prepared for his task of selection on the ramps at Auschwitz and for the supervision of the grisly procedure in the gas chamber.

The medical profession may have unwittingly provided a scientifically plausible rationale for the decision to exterminate the Jewish 'sub-human' race, but how could physicians like Josef Mengele and Eduard Wirth (the chief doctor at Auschwitz) have allowed themselves to be recruited to the ranks of killer? Lifton offers us an explanation by resorting to the phenomenon of 'doubling': the deployment of a second 'Auschwitz' self – a complement to the previous self of ethical doctor, husband, and father. Since the two, he argues, were totally autonomous, all requirements for moral responsibility could be transferred to the Auschwitz self and in turn to the pervasive Nazi ethos with which that self was fully identified.

Lifton's propositions help to illuminate what seems to be inherently incomprehensible. If he is unable to offer a totally convincing explanation, then he is in good company; Hannah Arendt, Mary Midgley (in her excellent essay on Wickedness), Erich Fromm and others have all wrestled with the dimension of evil in man's behaviour, and only succeeded partially. Lifton's book deserves close scrutiny by psychiatrists. They will gain in at least two significant ways: by acquiring a greater understanding of the nature of human destructiveness, and by realising how vulnerable their profession is to gross unethical pursuits.

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Drugs, Ageing and Society: Social and Pharmacological Perspectives. By BRUCE BURNS and CHRIS PHILLIPSON. Beckenham: Croom Helm. 1986. Pp 180. £19.95.

On opening this book, one is surprised that in a stiff-backed volume of relatively high cost, poor quality paper and typewriter setting have been used. The title is puzzling and somewhat unfortunate: it *could* imply that the authors are examining drug addiction in the elderly, rather than medication. They claim to "critically review the use and marketing" of medication used in the elderly. This aim tends to become obscured by the presentation of much data, which at times is repetitive. It is unclear for whom this is written. The authors suggest it could interest geriatricians, sociologists, psychiatrists, and pharmacists. Is it really then necessary to define dementia, postural hypotension, or beta blockers, to name but a few?

Numerous references are quoted. These range from respected authors to the *Mail on Sunday* and *Everywoman's Doctors Book of 1934*. It is difficult to take such information seriously, and this tends to detract from the important questions addressed. The chapters of special interest to psychiatrists include critical comment on the high use of psychotropic medication in the elderly. However, this is not balanced by a discussion of the high incidence of psychiatric illness in old people. Also, "unacceptable behaviour" in the elderly in elderly people's homes *does* need to be controlled – such control is by no means necessarily a "punitive" measure.

Sadly, despite the amount of sound information presented in this book few conclusions are reached, and I remain as unclear about the inter-relationships between drugs, ageing, and society as I was before reading it.

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Grieving: A Handbook for Those Who Care. By RUTH BRIGHT. Missouri: MMB Music Inc. 1986. Pp 229. $19.50.

This book, written by a music therapist of 25 year's clinical experience, makes several claims, notably that (a) it is suitable for a wide readership, and (b) it provides a practical approach. The first claim is justified: practitioners and laymen can learn from this book, as can the bereaved themselves. The researcher should also find it inspiring, because (by her own admission) the author is essentially a pragmatist and many of her ideas require more formal scrutiny and testing. The rigorously-minded academic would be disappointed, therefore, if he were to expect this book to be a source of carefully-researched ideas and principles.

The second claim is also appropriate, because the author describes in some detail how she puts her ideas into practice. More specifically, for the uninitiated, there is a chapter on music therapy, and there are references to its use throughout the book. Although medicine and music have a long association, music therapy is still relatively unfamiliar to most clinicians.

It is an unpretentious book, written in clear prose. Maybe I am a Philistine, but I do not share Dr Kastenbaum's claim in the Foreword that 'musical sensitivity can be felt throughout this book in its rhythms, structure, and invitation to respond'.

The earlier chapters provide helpful descriptions of reactions to various forms of loss, including chronic disability, old age, loss of material possessions, and loss of role. There are also useful sections on the difficulties facing those who have to deal with the grieving and dying, including those individuals who are involved in the emergency services and rescue work.

In conclusion, I found this an interesting book written by an obviously sensitive and experienced practitioner whose professional philosophy includes the need to respond to individual differences among those who are grieving, and to adopt an eclectic and flexible approach to help them.

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Modern Psychoanalysis of the Schizophrenic Patient: Theory of the Technique (2nd edition). By Hyman Spotnitz. New York: The Human Sciences Press. 1985. Pp 337. Price not stated.

This is the second edition of a book which was required reading in the course of basic training at 'Modern Psychoanalytic' Training Institutes in the United States. Before reading it I was unaware of the Modern Psychoanalysis movement, and I was interested to learn that there are eight Training Centres in the United States, and that since 1976 they have been publishing their own journal, *Modern Psychoanalysis*.

The term used for this treatment method, however, I find confusing. Patients are seen once or twice per week, and the method seems to centre around ventilation of accumulated frustrations, with a number of tactical manoeuvres available to ensure this process occurs: "Any technique employed to deal with resistance is regarded as permissible." Examples are given, such as telling a patient who does not wish to talk to "Keep quiet", with the intention that a contrary-wise patient will then talk. This is clearly not psychoanalysis, and I was unhappy with Spotnitz's statement that his treatment approach is "basically Freud's theory and Freud's method of therapy." Also, he justifies the use of the term 'modern' with too many quotations from the early days of psychoanalysis, as if there have not been many developments in the past one hundred years.

The important issue of diagnosis was left unclear in my mind. Although a short section is devoted to reviewing diagnostic criteria in general, I could find no definite statement about the diagnostic criteria for the patients treated by this method. Clinical examples are mainly to be found in the last chapter of the book, where various therapeutic interventions and strategies are described. The patients came across to me as more integrated individuals than the schizophrenic patients I am accustomed to, and I wondered if this reflected a diagnostic difference. The book in general made more sense to me as a psychotherapeutic approach to treating patients with personality disorders and chronic neuroses.

On the more positive side, there is a chapter on the development of the therapeutic alliance which I found useful reading, and the reference list is comprehensive. There would also be little disagreement with the emphasis given to the therapist's capacity to cope with treating patients with narcissistic disorders.

On the whole, in spite of the exciting title, I failed to find this a particularly stimulating book.

Tom Murphy, *Consultant Psychiatrist, Royal Dundee Liff Hospital, Scotland*

Neuropsychological Rehabilitation. Edited by Manfred J. Meier, Arthur L. Benton and Leonard Diller. London: Churchill Livingstone. 1987. Pp 475. £45.00.

It is only in the past 10–15 years that neuropsychologists have become interested in rehabilitating the patients they have so assiduously studied to elucidate the relationships between brain and behaviour. This book provides a comprehensive account of these endeavours and what they have achieved so far. In general, the results are disappointing. The major emphasis is on careful and accurate assessment of disabilities, and the first part of the book is devoted to this topic. However, many of the chapters in the second part (representative research and application) are also largely concerned with assessment, with only a nod towards rehabilitation at the end. Many still believe that given a sufficiently careful assessment, the steps needed for rehabilitation are self-evident.

In my opinion the major contributions that neuropsychologists can make to the study of rehabilitation are firstly, to provide theories of the restoration of function, and secondly, to design experiments to show which types of rehabilitation are successful. It appears that, as yet, few attempts have been made to do either of these. As one contributor puts it "The description of the procedure is anecdotal and no data are presented". With regard to the lack of controlled trials, the all-too-frequent false moral is put forward that it is unethical to withhold treatment. This problem must be balanced by the lack of ethics in pinning hopes on procedures with no proven value.

The book is somewhat carelessly edited, in that there is much unassimilated overlap between different contributors. Also, some have reviewed their topic in detail while others have restricted themselves to their own specific interests. Posner and Rafal describe an interesting theory of attention, but are not yet able to come up with specific suggestions for rehabilitation. O'Conner and Cermak provide a useful account of the rehabilitation of memory disorders, and Anna Basso does the same for disorders of language. The last part of the book summarises what is actually being done in rehabilitation centres throughout the western world. Given all this activity I am sure there will be great advances in the neuropsychology of rehabilitation over the next ten years. This book has perhaps appeared prematurely.

CHRIS FRITH, *MRC Senior Scientist, Clinical Research Centre, Harro*

The Psychology of Child Firesetting: Detection and Intervention. By JESSICA GAYNOR and CHRIS HATCHER. New York: Brunner/Mazel. 1987. Pp 220. $25.00.

Arson is one of the commoner serious and dangerous crimes in this country and the USA, where, surprisingly, a third of all fire-setting is by children. Certainly the incidence in the UK of fire, in particular school fires, set by children and teenagers has drastically increased since my book *Fire Raisers* was published ten years ago. It is, nevertheless, a common observation that virtually all children play with matches. How then does this relate to serious fire crime? Any study on the subject is clearly of great importance. The current book by two psychologists is of particular interest because it reports a long programme of work in co-operation with the San Francisco Fire Department.

There are two main sections: detection and intervention. The former is based on a predictive model suggesting that 'fire interest' is channelled, because of various psychosocial determinants, into the 'fire safe' or the 'fire risk' categories. There are many useful tabulations provided in this section of the psychological and physical characteristics of fire-setters, but it is a pity that more case material is not provided.

The great variety of intervention processes, psychotherapeutic and behavioural, that can be employed are described, both for the individual and the family, as well as methods of patient selection. Of considerable interest are community intervention methods with schoolchildren in different age groups. The approaches vary in different parts of the United States. In one the cost-effectiveness was assessed, but on the whole follow-up information is limited. The final chapter suggests the appropriate roles of various agencies and professionals involved, including the physician. Much useful help in a difficult field is provided by this reasonably priced book, but it is clear that more remains to be done, particularly in the evaluation of intervention methods.

D. F. SCOTT, *Consultant Neurophysiologist, The London Hospital*

The Therapy of Pain (2nd ed.). Edited by MARK SWERDLOW. Lancaster: MTP Press. 1986. Pp 271. £32.95.

This is a second edition of a valuable textbook on pain management. It contains an update on the neurophysiological aspects of pain (Cervero) and there is a balanced and informative chapter on the role of the psychiatrist in the management of chronic pain (Pilowsky). Lipton and Swerdlow provide helpful and brief descriptions on the management of a pain relief centre and assessments of the pain patient. The chapter on pharmacological management of pain (Williams) provides practical advice for those interested in pain management. Unfortunately, however, there is an uncritical acceptance of treatments such as transcutaneous nerve stimulation, trigger point therapy, and biofeedback, none of which have been shown to be more valuable than the placebo in controlled studies. The chapter on non-invasive methods in pain relief accepts the value of these therapies without question (Mehta). The chapters on nerve blocking and neurosurgery for pain relief are well illustrated and helpful (Charlton and Hitchcock). Low backpain has been singled out for an independent chapter, and Porter's contribution provides sensible advice. The final chapter on oncological management is excellent (Saunders).

The quality of the book is good. I think it is a useful book for students and practitioners alike, but I would like to see more critical evaluation of therapies such as trigger point therapy, as there is a danger that they gain respectability without having been adequately tested.

CHARLOTTE FEINMANN, *Consultant Psychiatrist, Eastman Dental Hospital and Senior Lecturer, Middlesex Hospital and University College Hospital*

The Psychopharmacology and Treatment of Schizophrenia. Edited by P. B. BRADLEY and S. R. HIRSCH. Oxford: Oxford University Press. 1986. Pp 457. £37.50.

Although some reference is made in this book to the social methods of management of schizophrenia the emphasis of the volume as a whole is very much towards drug treatment. One might imagine that a book of this size devoted to this topic would be inclined to be repetitious and would be covering a good deal of well-ploughed ground. The editors are to be congratulated on the fact that this is not the case. They have incorporated some unusual chapters, bringing together topics which are not often discussed, and the chapters which deal with well-known areas of work generally do so in a careful and informative way.

The opening chapter, on peripheral biochemistry, elegantly draws together a number of areas of work which are relatively unfashionable among clinicians and other scientists but some of which tend to come to the attention of sufferers and their relatives. Clinicians who would like a convenient source of references in order that they may present concerned individuals with facts regarding zinc and magnesium metabolism, prostaglandins, and gluten sensitivity in relation to schizophrenia need to look no further. The chapter on the pharmacology of antipsychotic drugs is accurate, informative, and well referenced, as indeed are those on animal models, applied clinical pharmacology, and neurotransmitter hypotheses of schizophrenia. The case for the use of animal models in psychiatric disorders is made so persuasively that it must convince all but the most cynical of clinicians.

These chapters are followed by a series of competent and useful essays on clinical issues relating to the study of schizophrenia; these include diagnosis, the influence of social factors, and rating scales which may be used in schizophrenia. The chapter on the difficult issue of essential elements in the design of clinical trials is particularly well done. The thoughtful and well organised chapter on the evaluation of social functioning covers areas which are not often addressed in this careful style. The long chapters on the clinical treatment of schizophrenia and untoward effects of antipsychotic drugs cover the issues clearly and comprehensively, and the section on extrapyramidal movement disorders produced by antipsychotic drugs is very good indeed. In spite of the fact that this book concerns areas of work with which I am very familiar it was interesting and informative and I enjoyed it. It is a well-written account of the current state of knowledge of the psychopharmacology and drug treatment of schizophrenia and will be useful to trainees in psychiatry and to established clinicians.

EVE C. JOHNSTONE, *Member of the Scientific Staff and Honorary Consultant Psychiatrist, Northwick Park Hospital, Middlesex*

Can Schizophrenia be Localised in the Brain? Edited by NANCY ANDREASEN. Cambridge: Cambridge University Press. 1986. Pp 87. £15.00.

This small volume contains the papers presented at a symposium at the American Psychiatric Association Annual Meeting. In such symposia it is appropriate to make the best of one's data and to present one's case with force and conviction. The case is after all open to argument from the floor. The papers presented here must have been very successful in that situation, and Andreasen in particular develops her argument with considerable finesse.

As a written text, however, these papers have shortcomings. Most of them have been written to make conflicting cases in a way that could provoke discussion, and I am sure the authors would not claim that they have attempted a balanced or complete review of the relevant literature. This is a style which promotes lively controversy at a meeting and which may be appropriate enough in a paper published in a journal with an active correspondence column, but it is less suitable in book form. There is no opportunity for the more argumentative reader to ask how the authors take account of the work of X, Y, and Z, or in what way the data in some of the papers cited really support the case that is made.

The last paper, by A. M. and M. A. Reveley, differs from the others. It is written in a considered and understated style which most readers will find more familiar and certainly less abrasive. The fact that the provocative quality of the other papers raises so many questions in the reader's mind may, however, be a virtue in itself. This book may raise the interest of psychiatric trainees, but it will not give them a clear view of the current state of knowledge in the field.

EVE C. JOHNSTONE, *Member of the Scientific Staff and Honorary Consultant Psychiatrist, Northwick Park Hospital, Middlesex*

Expressed Emotion in Families. By JULLIAN LEFF and CHRISTINE VAUGHN. New York: Guilford Press. 1985. Pp 241. £19.95.

The scientific approach to the study of environmental influences on the course of mental disorders has seldom been applied with the rigour expounded in Leff and Vaughan's book on 'expressed emotion'. To date more than 10 published studies have replicated the finding that the quality of the social environment in which a person lives is the best single predictor of the short-term course of schizophrenia. George Brown's remarkable first person account of the development of the 'expressed emotion' concept from 1956 to the early 1970s provides a fascinating commentary on the manner in which an intuitive experience of sensing a tense household atmosphere was eventually quantified into a highly reliable measure. The surprise and scepticism he felt on finding that this factor proved such a robust prognostic indicator has been experienced by many since.

Brown's work has been refined in the past decade. Detailed accounts of these refinements are described in the remainder of the volume. The landmark 1976 study, the London and Californian replications, and early family intervention studies are included. Although much of this material has been published in scientific journals, this book provides many additional details that will undoubtedly stimulate at least another decade of 'expressed emotion' studies.

The significance of this body of work is such that this book should be essential reading for every mental health professional and student of the social sciences.

IAN R. H. FALLOON, *Consultant Physician (Mental Health), Buckingham Mental Health Service*

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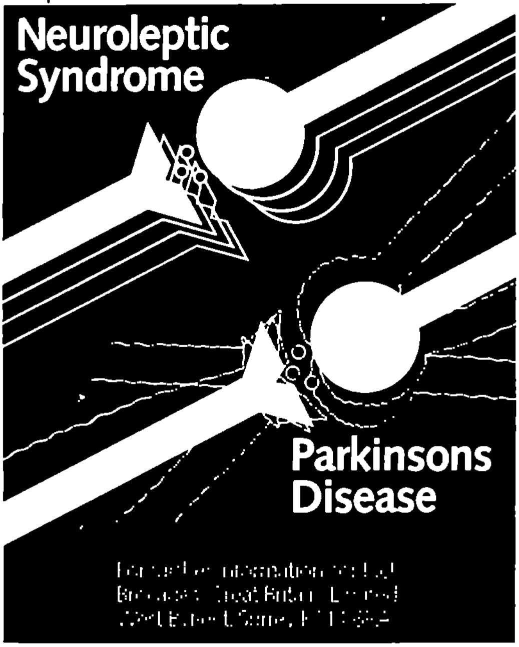

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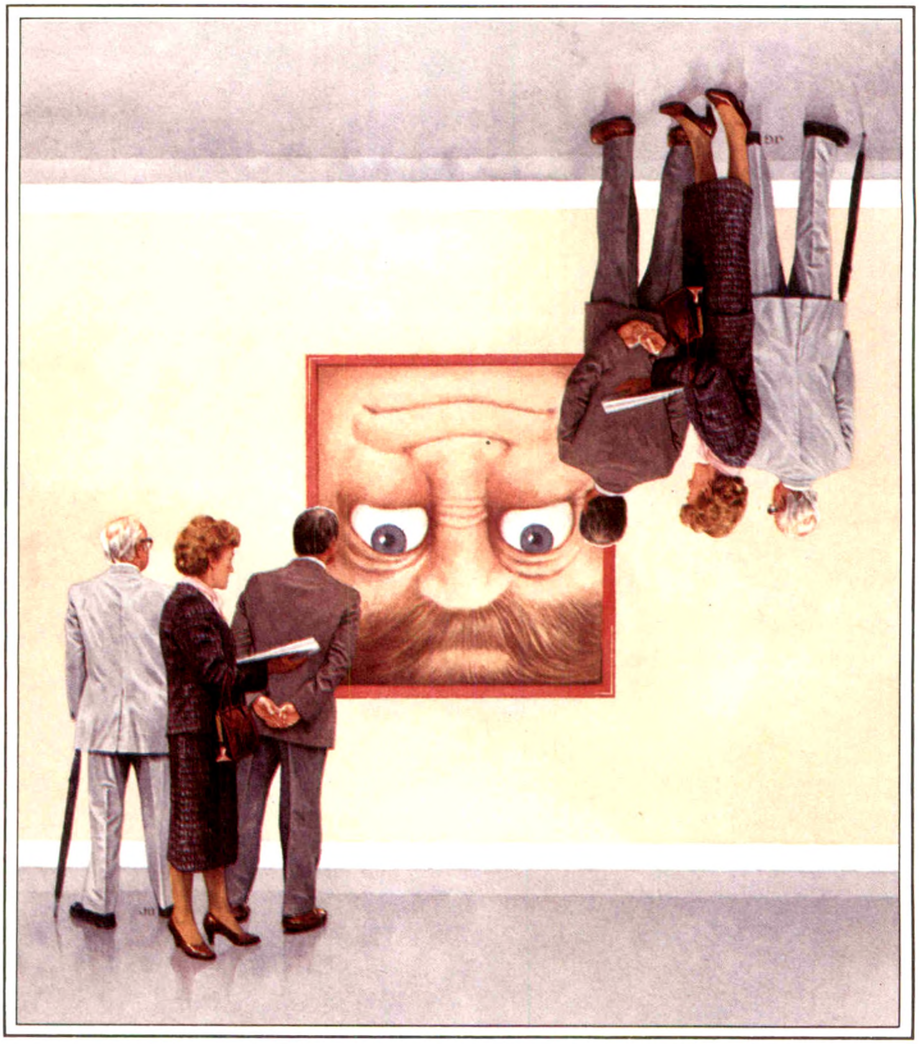

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Annotation

Psychotherapy Today

Further Consideration of the Essence of Psychotherapy*

STANLEY E. GREBEN

Despite swings between over-valuation and rejection of psychotherapy on the part of some psychiatrists, there remains a valid and necessary place for psychotherapy in psychiatry. The essential elements of that place are described.

Six years ago, I endeavoured to abstract what it is that matters and is effective in the practice of psychotherapy (Greben, 1981). There have since been important shifts in the attitudes of psychiatrists toward the value or lack thereof of psychotherapy. At the same time, many other professionals have become active in psychotherapeutic work. Members of the public, needing help for symptoms that discomfit them and diminish the quality of their lives, are often hard pressed to find useful help, both within and without medical facilities.

My awareness of this circumstance has been heightened by responses of both professional and lay people to views expressed in my book, *Love's Labor* (Greben, 1984). Patients and family members, some of whom are psychiatrists, expressed concern, dismay, even outrage, at ways in which they or others had been dealt with in psychotherapy. We need to address these concerns, if we intend psychotherapy to be a respectable component of our therapeutic armoury.

I intend to set out my current view of what should be the place and use of psychotherapy, from the vantage point of one who is a medical specialist in psychiatry and also a psychoanalyst, working in a teaching general hospital. I will also deal with issues that are important to other professionals in the health field, as well as to the consumers of our services. I hope to bring perspective to an area which is of great importance, not only to those of us who work in it, but to Western society in general.

In examining factors which I feel to be effective in bringing about change through psychotherapy (Greben, 1977), I began with the proposition that psychotherapy *does* lead to changes in patients, and that this has been amply demonstrated by numerous studies (Smith *et al*, 1980; Karasu, 1982), as well as by the observations of experienced clinicians. The challenge was to then demonstrate what are the reasons for the improvements that occur. As I set out in that instance with the relatively optimistic point of view that psychotherapy does work, I set out in this instance with the relatively optimistic view that much excellent psychotherapeutic work is taking place. At the same time, a considerable amount of work has been done that is not acceptable, and has not met justified expectations of those who have sought help.

Influences of society upon psychotherapy

As medical scientists, we prefer to think of the scientific part of our work as freestanding from popular attitudes in society, but of course it is not. Even 'hard' basic sciences are subject to the pull of current public attitudes. With psychotherapy, a number of external influences have played an important part in the directions which both our theory and practice have taken.

Freud developed psychoanalysis in Vienna at the turn of the century. Despite a milieu of relative psychological repression and denial, in a stratified society where surface and depth were so at odds, he was able to be candid and revealing about his feelings and findings. That candour was essential to the beginnings of a profession devoted to the uncovering and revelation of realistic truth. Freud's discoveries were of course consistent with the content of classical writings of centuries before: the authors of the Scriptures, Roman and Greek philosophers, Shakespeare, and Dickens clearly understood these principles. As Freud indicated, he became famous revealing "what every nursemaid knows".

The Second World War displaced European psychoanalysts, principally to England and the United States, and that which had been of mild influence came to be of much greater influence. They became, especially if their direct lineage could be drawn to Freud, a kind of guru. In those years, many people in Western society were uncertain about themselves, and about the form and direction which it was best for life to take. Scientists seemed to have important answers to the dilemmas that faced most people. Fear, ennui and insecurity abounded. The American child psychiatrist, Leo Kanner, alarmed by this phenomenon, wrote a book entitled *In Defence of Mothers* (Kanner, 1962).

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*Based on a lecture given to the Department of Psychiatry, University of Western Ontario, October 1986.

After the war, psychoanalysis dominated psychiatry, and also had a powerful general influence upon Western society. Much has since changed. Too much was expected of psychoanalysis by its practitioners, consumers and general adherents, and so disillusionment ensued. Many reasoned that helping people live more fruitfully and happily did not necessarily require years of formal training and personal psychoanalytic treatment. Anti-traditionalism thrived in all areas, and psychotherapy was no exception. Beginning among other places in Big Sur, California, everything old was rejected and everything new was attempted. Touching, holding, in addition to talking, was tried. All manner of theories were expounded and, for a time, cherished.

As we emerge from all that to a time when traditional and conservative values are coming once again to the fore, the work of Freud, Jung, Winnicott, Klein, Greenson and many others has not disappeared. Still, the influence of freer psychotherapy which demands more participation of the therapist has continued, and is likely to have a lasting effect upon the development of a general psychotherapy.

Another important and wholesome recent influence upon psychotherapy has been the view that people have a right to receive something evident and helpful when they spend time, money and energy seeking help. Numerous psychoanalysts took, in past years, the view: "I don't try to help people, I analyse them. Analysis is an art." Many people today do not accept that view. Correctly, professional practitioners are now being held to more account, both by those who provide financial support and by those who are the objects of that work.

Another societal influence upon the practice of psychotherapy arises out of the growing intention of people to live in more natural ways, avoiding the noxious effects of poisons and of disuse. Such 'holistic' approaches presume that that which is closer to nature and the experiences of our ancestors is most likely to suit us well, and do us the least harm. For all psychotherapists, an understanding of Oedipus and Psyche is insufficient. For the physician psychotherapist this carries particular implications, for he is expected to understand, not simply to dismiss, the significance for health of nutrition, exercise, meditation, hypnosis, yoga, and other special practices which have withstood, presumably for good reasons, the practical test of time. He must, for example, know the work of Benson (1974), so that he can assess his patient's search for techniques that will induce the relaxation response. All physicians must know the accumulated evidence of the benefit of a diet which is low in fat, simple sugars and salt, and high in complex carbohydrate and fibre. They must be equally cognisant of the multiple benefits of avoiding underuse of the body, recognising that the release of endorphins and epinephrine into the bloodstream has a profound effect upon mood and mentation and, aware of the dangers of substance abuse for the relief of the effects of stress and unhappiness, should encourage patients in the less dangerous habituation of regular and sustained athletic activities. Similarly, the psychotherapist who ignores the place of humour in becoming and continuing to feel well (Cousins, 1979) will be a very limited psychotherapist indeed. In short, we have ample proof that insular psychotherapies are inadequate and cannot succeed.

Another attitude has been growing in strength and popularity that must be paid heed to by psychiatrists. Considerable dissatisfaction is being expressed about the ways in which health care is provided. In part this arises because of a general deterioration in the quality of caring for others in everyday life. In part it relates to the cost of physicians' services, both physical and psychological. Complaints are now openly heard that physicians are more technicians and less care-givers. More demand is being made for humane, individual care of patients. This dissatisfaction, on the part of both physicians and patients, demands redress.

This trend has considerable significance for psychotherapists. Ten and twenty years ago I knew of patients in intensive psychotherapy or psychoanalysis whose therapists said literally nothing for months on end of several-times-weekly treatment. Today, when patients have come to feel more deserving of humane, active treatment, such neglect, even when benign, is less likely to be tolerated. This is a very good thing. The days when psychotherapists were gratefully attended and paid for just being present in the room are passing or passed. We are being expected, as are all professionals who are paid for their time and services, to produce something that will be useful and valuable to the patient.

One encouraging manifestation of these changed attitudes has been the establishment of a new international journal entitled *Humane Medicine*. Its stated purpose is: ". . . to provide a vehicle for communication among all those who have something to share about ministry to the whole person – to body, mind and spirit" (Oreopoulos, 1985). The provision of such a forum, bringing together the concerns of both health care professionals and their clients or patients, bespeaks a new attitude that has profound implications for the practice of psychotherapy. That attitude expects people to be treated well and respectfully, and to be given something valuable for the time, energy and money which they invest in the pursuit of health and comfort. A main goal of psychotherapy is the growth, maturation and psychological development of the patient, and these can take place only under the conditions that are currently being expected of us (Marmor, 1977).

Overall, those influences in society which currently pull us more into the mainstream, are likely to improve both our relations with our patients and the ways and degree to which we are able to be of help to them.

Influences of other professions upon psychotherapy in psychiatry

In recent years there have also been important relevant changes in the attitudes and activities of our fellow professionals, and in our attitudes toward them. Some psychiatrists adhere to the view that psychotherapy is a medical act only, and can properly be undertaken only by medical personnel. This is not a view that can be logically supported, since the largest part of what is done, most of the time, in psychotherapy is psychological, and has not to do with physical or medical matters. Others hold the opposite view: that psychotherapy is not a medical act, and that medical training is irrelevant to its performance. This view is also illogical, for much that presents as psychological disturbance originates in medical disease, and much that is treated solely by psychological means cries out for biochemical or other medical therapy.

A reasonable view would be that there is a place for psychotherapy to be practised by both medical and non-medical professionals, and that the advantages and limitations of the training and experience of each specific profession need to be recognised. From the vantage point of psychiatry, psychotherapy must continue to be taught, studied, and practised as an indigenous part of psychiatry (Katz, 1986). At the same time, it is appropriate that we find ways of co-operating with members of other professions, not just competing, in this area of our work.

In the history of psychotherapy, both as a science and as an art, a major role has been played by both physicians and non-medical workers, most obviously within psychoanalysis, but also in the field of psychotherapy research. It is healthy for psychotherapy that workers come to it from several professions, for in that way the effect of blind spots that exist in each profession is reduced. The territory of psychotherapy is large, and research by both physicians and others has produced fruitful results.

Influence of attitudes of psychiatrists and other medical psychotherapists upon psychotherapy

In recent years, some medical practitioners, including psychiatrists, have been prepared to eliminate psychotherapy as an indigenous part of psychiatry, and to delegate its practice to others. This tendency is against the entire history of the profession of medicine and of the specialty of psychiatry. There are training programmes for residents in psychiatry in which psychotherapy supervision for residents has been given over to a psychologist. There are hospitals in which suitability of a patient for psychotherapy is determined not by a psychiatrist, but again by a psychologist. This is not the major trend in psychiatry, but it should be strongly discouraged. For psychiatrists to relinquish the study, practice and teaching of psychotherapy is to be absolutely avoided, since doing so would lead them to be not medical psychologists, to use an old term, but behavioural physicians, and would justify the apprehensions, fears and even expectations of those who see medical practitioners as becoming more and more technicians, and less and less clinicians.

Another attitude which has hindered the development of the most useful general medical psychotherapy has arisen out of the use of our most important medical tool, the scientific method. This tool will always be the most reliable means of drawing trustworthy conclusions within any arena. Still, out of excessive respect for that method, we have tended to eliminate common-sense observations that mean so much in the learning that occurs in all areas of life. The psychotherapeutic process is both simple and complicated. In being scientific, we demand studies so rigorous, to prove the obvious, that no one is able to devise them. Who demands a study to prove whether a university education leads to change in students exposed to it? Further, we take hypotheses which had some value as such, and make them rigid dicta that are expected to stand forever unchanged. Freud altered his views, by and large, throughout his career. Many psychoanalysts who followed him took his tentative hypotheses as established gospel, and considered all efforts to change them as heretical. Theoreticians in psychotherapy have understandably sought to reduce the pathological factors present to the smallest possible number, seeing all human neurosis through the framework of one or another over-simplified theory. The Procrustean bed of Oedipus cannot begin to accommodate the complex sources of disease and behavioural disorder that confront psychotherapeutic clinicians. We must avoid excessive generalisation and undue simplification. If there is a universal rule about human behaviour and pain, it is that character is so complex and human interactions so idiosyncratic that useful psychotherapy demands an entirely individual approach to each patient. We must avoid automatism, and retain our individual styles in approaching our individual patients, with their highly individual discomforts and disorders.

We have often carried diagnosis to extremes whereby a popular current way of looking at a common difficulty makes us see something esoteric in that which is not. In the past two decades the unhappy and regrettable ways in which stresses and lacks in Western society have moulded people, especially young people, have made us designate disorders or syndromes which we then discover on all sides. For a time everyone is hysterical, then passive–aggressive, then narcissistic, then borderline, next whatever comes along. Different experts propose different approaches for dealing with those who demonstrate hallmark symptoms of these 'new' disorders. It is doubtful that those approaches contribute much that is new, and they may divert the therapist from his most useful stance – that of an interested, enquiring, empathic, informed and open-minded clinician.

The struggle in psychiatry with the tendency to divide the person into body and mind has been a long and difficult one (Eisenberg, 1986). As a physician training in psychiatry in the 1950s I was exposed to two quite opposite views in regard to serious depressive disorders. Clinicians who were either psychoanalysts or else psychiatrists who felt that the only fundamental treatment was psychoanalytically oriented psychotherapy believed that severe depression was entirely a psychological matter, despite the fact that vegetative signs and behavioural symptoms of all seriously depressed patients were the same. The obvious need for organic (that is, biochemical and neurophysiological) explanations of the signs and symptoms was not seized upon. We used electroshock when the severity of the disorder demanded it, but with regret and often with disparaging references to 'the little black box'. Other clinicians, understanding that the depressed state must have biochemical underpinnings, prescribed biological treatments only. They considered it misguided and contra-indicated to 'waste time' talking to patients who suffered from such biological disorders, even when the cause was seen to be 'reactive' rather than genetic and familial, that is, 'constitutional'.

There has been a considerable improvement in this dichotomous situation. Today many psychiatrists are prepared to offer both biological and psychotherapeutic treatments to such patients. They hold that serious depressive disorder can arise in various ways. There may be a strong constitutional imperative or, at least, vulnerability, or it may result from the effects of "the slings and arrows of outrageous fortune". Whatever the aetiology, it is a situation in which biochemical change has occurred in the brain. To shorten the duration of pain, to forestall the possibility of violent behaviour, biological treatments (ordinarily chemical) are often or usually indicated. At the same time, some appropriate psychotherapy is equally indicated to deal with numerous factors: low self-esteem, the psychosocial causes that contributed to the development of the disorder, and, most important, the qualities of character that helped create the problem and threaten to do so again if untreated.

Such a multidimensional approach to psychiatry sees the presence of a psychotherapeutic component of treatment as not only acceptable, but essential. Psychotherapy must be recognised today as the potent tool that it is, one that does not exist in a vacuum: a flexible, variable, individually tailored treatment that takes its place as one amongst important equal companion treatments.

The essence of psychotherapy within psychiatry

What then is an appropriate psychotherapy for psychiatrists today? Often in the past, psychiatric psychotherapists have claimed to be super subspecialists, and have disparaged other than their own small area of special interest, be it psychoanalysis, family therapy, group therapy, behaviour therapy, or cognitive therapy. As I have pointed out elsewhere (Greben, 1984), it appears that most practitioners are more flexible and use more varying approaches than they imply publicly. Still, many therapists have restricted themselves to one technique only and this is as unsatisfactory as when a general psychiatrist is familiar with only one antidepressant or one major tranquilliser.

The general public has often been mystified by our language and our excessive contractions, but so have educated people in a variety of fields and professions. Somehow, our ways of understanding people must be less different than they have thus far been from the ways in which people have always been seen. We cannot improve upon Shakespeare and Dickens and Tolstoy, in terms of how a person is to be understood. That is, we must in the end return to everyday language and everyday formulations. For when we cannot be understood by intelligent people outside our own profession, then in truth we are not being understood by one another, and that in turn means that we are not being understood by ourselves.

What, then, are the implications of all this for psychotherapy in psychiatry? I would say that the following principles obtain:

- There can be no psychiatry without psychotherapy. In one form or another, psychotherapy is an essential treatment method, among numerous others, that are the tools of psychiatry.
- There is no single superior psychotherapy. More intensive work is better for some patients and worse for others.
- A psychiatrist needs to be competent in the use of psychotherapy and must be equally competent in the use of biological treatments, which currently largely means medications. If his training is deficient in this area, then he must have a colleague to whom he can refer his patients for such consideration.
- There is no reason to hesitate to combine biological treatments with psychotherapy, since the former in many instances will shorten the painful period for the patient, lessen the danger of suicide, make admission to hospital less often necessary, and render the patient better able to engage in the psychotherapeutic undertaking.
- A deep interest in psychotherapy does not mean that a psychiatrist should become distant or alienated from his medical colleagues. He should see himself as first a medical person, then a specialist in mental and emotional disorders and then a person with a particular interest in psychotherapy.
- Each of the various ways of applying psychotherapeutic principles has its own merit and value. Psychiatrists should be aware of the value of a therapist's meeting with more than one person – a married couple, part of or a whole family, or groups that have any of a variety of things in common.
- At the heart of all psychotherapy is the need of people in crisis to be heard, understood and helped. Therapists should be what patients need them to be: empathic, interested, concerned, reliable, respectful, and resourceful. They need to work hard at what they do, and not leave all the effort to the patient.
- The patient needs to be understood. The therapist needs to understand. On neither side is it necessary for that understanding to be perfect. The imperfection of the therapist will in the end help the patient to deal with the tendency toward idealisation of the therapist.
- In deeper psychotherapeutic work, attention to and discussion of what happens between the two participants is essential if the greatest possible degree of change is to eventuate.
- In addition to addressing symptoms, should they choose to do deeper work, psychiatrists will deal with

the underlying modes of behaviour and ways of relating to others which are best described by the word 'character'. The analysis of all aspects of character gives deeper forms of psychotherapy their greatest interest, and provides both therapist and patient with their greatest reward.

Isaac Bashevis Singer (1985), commenting upon a writer's work, said: "We always love to discuss and reveal character because human character is to us the greatest puzzle. No matter how much you know a human being you don't know him enough. Discussing character constitutes a supreme form of entertainment."

In a comparable way, attempting to understand the characters of our patients is the most challenging and entertaining aspect of the work of psychotherapists. To be humane, real people engaged in the work of psychotherapy, whether frequent and deep, or occasional and superficial, we must know and keep in mind the character of the patient, as well as his and our own humaneness. If we manage this, we will succeed in making and keeping psychotherapy, both today and tomorrow, the useful, important, even essential part of psychiatry that it must always be.

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Pathology, Phenomenology and the Dopamine Hypothesis of Schizophrenia

P. J. McKENNA

The dopamine hypothesis of schizophrenia implies that positive schizophrenic symptoms should be understandable by reference to brain structures receiving a dopamine innervation, or in terms of the functional role of dopamine itself. The basal ganglia, ventral striatum, septo-hippocampal system, and prefrontal cortex, sites of mesotelencephalic dopamine innervation, are examined and it is argued that their dysfunction could form the basis of particular schizophrenic symptom classes. The postulated involvement of dopamine in reinforcement processes might further assist such interpretations. This type of analysis can be extended to other categories of schizophrenic psychopathology.

The leading view on the pathogenesis of schizophrenia, that a functional dopamine excess underlies some symptoms of the disorder, originated in observations that dopamine agonist drugs could induce a schizophrenia-like state, while therapeutically effective neuroleptic drugs were invariably dopamine antagonists (Carlsson & Lindqvist, 1963; Van Rossum, 1966; Randrup & Munkvad, 1967). The dopamine hypothesis gained ground with the systematic exploration of these effects (Snyder *et al*, 1974; Carlsson, 1977; Johnstone *et al*, 1978), which also indicated an involvement only in the productive, positive symptoms and not in those of the negative symptom defect state. A subsequent search for indices of dopamine excess in schizophrenic brains (see Mackay *et al*, 1982) has recently culminated in the demonstration of increased receptor binding in drug-free (Crawley *et al*, 1986) and drug-naive (Wong *et al*, 1986) patients. If confirmed, these findings strongly imply that some schizophrenic symptoms should be understandable in terms of structural and functional aspects of brain dopamine projections.

Structurally, the ascending dopamine pathways arise from the partly continuous midbrain A8, A9 and A10 cell groups, show a complex pattern of overlap in their terminal fields, and are currently considered to represent a single 'mesotelencephalic' system (Bjorklund & Lindvall, 1984). Two gross subdivisions are nevertheless retained: in the rat a 'mesostriatal' branch projects to the basal ganglia and some affiliated structures. The other, 'mesolimbocortical', branch innervates restricted parts of the limbic system and also reaches cortical areas corresponding to the prefrontal and anterior cingulate cortex of higher mammals. A similar pattern of innervation appears to be followed in primates and man, with the possibility of additional significant projections to parts of the temporal lobe cortex (Brown & Goldman, 1977; Bjorklund *et al*, 1978; Farley *et al*, 1978; Scatton & Zivkovic, 1984).

Functionally, the mesotelencephalic dopamine system has invariably been considered to exercise some diffuse modulatory function distinct from classical neurotransmission (Hornykiewicz, 1976; Bjorklund & Lindvall, 1984; Bloom, 1984). The further characterisation of this function, however, has involved the discipline of experimental psychology. Following Olds & Milner's (1954) discovery of the rewarding properties of electrical brain stimulation, it soon became established that catecholamine systems were crucially involved (see Crow *et al*, 1972; Wise, 1978). At present it seems clear that dopamine, either alone (Mason, 1984) or in co-operation with other transmitter systems (Crow & Deakin, 1985), mediates the reinforcing effects of brain stimulation, and it may govern at least some aspects of reinforcement under natural conditions as well (Wise, 1982; Beninger, 1983; Mason, 1984). This evidence has survived numerous attempts at refutation and requires accommodation in any theory of dopamine's behavioural role (Deakin, 1983).

The structural interpretation of schizophrenic symptoms began with Kleist (see Fish, 1957; Kleist, 1960) who, long before the discovery of dopamine, argued for the presence of signs of basal ganglia and frontal lobe pathology. Several subsequent authors (e.g. Stevens, 1973, 1977; Crow *et al*, 1977; Iversen & Fray, 1982) have attempted to relate schizophrenic symptoms to these and other sites of mesotelencephalic dopamine innervation. The complementary view, of schizophrenia as a disorder of reinforcement, dates from Stein & Wise's (1971) proposal of progressive damage to a central reward system. This approach, applied specifically to positive symptoms, has been adopted by Crow and co-workers (Crow *et al*, 1977; Crow, 1979) and elaborated further by Miller (1981, 1984). But, while some aspects of the dopamine hypothesis have been the subject of detailed investigation (e.g. see Joyce, 1983; Creese *et al*, 1983; Mason, 1984), its application to the phenomenology of schizophrenia remains uneven, haphazard and unintegrated with developments in neuroanatomy, neuropsychology and learning theory. This review, therefore, summarises current knowledge on dopamine-innervated brain structures, examines ways in which their pathology might be related to schizophrenic symptoms, and argues that in each case independent findings support the conclusions reached.

The basal ganglia

Animal experimental findings

The largest site of forebrain dopamine innervation is the basal ganglia. The mesotelencephalic dopamine system provides dense fibre distributions to the caudate nucleus and putamen (jointly termed the striatum), and there are additional sparser projections to other basal ganglia core structures, the globus pallidus and subthalamic nucleus (Bjorklund & Lindvall, 1984).

The major input to the basal ganglia is the entire cerebral neocortex; this projects in an orderly way upon the striatum, and a large through route, the striopallidal pathway, connects this with the premotor regions of the frontal lobe (Graybiel & Ragsdale, 1979; Nauta & Domesick, 1984). The obvious anatomical inference, that the basal ganglia have some motor function, is supported by considerable physiological evidence (Marsden, 1982). At the same time, certain inadequacies in such a view are recognised: both lesion (Teuber, 1976; Stern, 1983; Divac, 1984) and electrical recording (DeLong *et al*, 1984; Evarts & Wise, 1984; Rolls, 1984) studies associate the basal ganglia as much with the learning and performance of various behavioural tasks, as with the simple motor control of these. Overall theories of basal ganglia function have tended to be inexplicit, or have pointed to a role in the specifically volitional aspects of motor behaviour (Villablanca & Marcus, 1975; Divac & Oberg, 1979). According to Marsden (1982, 1984), the basal ganglia may ultimately be responsible for "the automatic execution of learnt motor plans".

Dopamine has complex effects in the basal ganglia, but overall has been argued to facilitate the passage of information round the striopallidal circuit (Groves, 1983). Behaviourally, severe dopamine depletion induces a profound hypokinesia (see Joyce, 1983; Mason, 1984) which characteristically is temporarily abolished by intense adverse stimulation (for instance, immersing rats in freezing water or placing them among cats!). Dopamine agonist drug stimulation, on the other hand, induces a general locomotor hyperkinesia which quickly gives way to the phenomenon of stereotypy, where there is an increasing repetition of one or a few responses from the normal repertoire of the animal (Iversen, 1977): the normal variability of behaviour gradually diminishes and is replaced by the incessant performance of simple acts like sniffing, biting or rearing.

The conceptualisation of dopamine as reinforcement is by no means incompatible with such findings. A number of authors (Iversen & Koob, 1977; Wauquier, 1979; Miller, 1981) have noted similarities between behaviour termed 'voluntary' and that referred to as 'instrumental' in learning theory, and have suggested that the basal ganglia might function generally as an effector system for instrumental learning and performance. Instrumental behaviour is of course governed by reinforcement, and recent formulations stess not only the familiar trial-to-trial 'learning' effects of reinforcement, but also its moment-to-moment 'performance' functions. Reinforcers are considered able to energise responding directed towards themselves, resulting in the occurrence of relatively unstructured approach responses (*vide infra*) or of already learnt formal instrumental acts (Bolles, 1975; Gray, 1975; Bindra, 1978) – this latter function perhaps being not far removed from a facilitation of Marsden's (1982, 1984) "automatic execution of learnt motor plans".

Human basal ganglia pathology

The cardinal signs of disturbed basal ganglia function in man are Parkinsonism and the involuntary movement disorders. In addition, a variety of basal ganglia disease states are accompanied by cognitive impairments; this echoes animal findings, though the extent to which the basal ganglia rather than other structures are involved remains controversial (Marsden, 1980; Cummings & Benson, 1984).

In Parkinsonism, the loss and slowing of voluntary movement is thought to reflect a relatively pure basal ganglia hypofunction, with features like tremor and rigidity being attributable to the abnormal release of function in distant brain sites (Marsden, 1984). Parkinsonism is typically the result of reduced striatal dopamine availability, and there is no dispute that it is analogous to animal hypokinetic syndrome that follows dopamine depletion.

The involuntary movement disorders consist principally of chorea, athetosis and dystonia. Despite considerable phenomenological differences, these are usually considered to be the closest human counterpart to the animal syndrome of stereotypy. As with stereotypy, striatal dopamine excess is commonly implicated in the genesis of involuntary movements and has been argued to be the common denominator of choreo-athetosis in a number of disease states (Klawans & Weiner, 1976; Calne, 1982).

Basal ganglia dysfunction in schizophrenia?

Irrespective of any basal ganglia role in cognitive functioning, the dopamine hypothesis clearly predicts that among the clinical manifestations of schizophrenia, involuntary movements should be seen. Controversy over this issue predates the discovery of dopamine by decades. In his original description of catatonia, Kahlbaum (1874) recorded abnormal movements which so resembled those of basal ganglia disease that later authors (Marsden *et al*, 1975) felt obliged to explain them. Kraepelin (1913) noted the occurrence of "peculiar, sprawling, irregular, choreiform, outspreading movements" in several of his patients, these being recorded in detail by Farran-Ridge (1926). Bleuler (1911), on the other hand, considered that true choreo-athetosis was rarely, if ever, seen in schizophrenia.

Contemporary surveys of psychiatric populations have revealed the presence of tardive dyskinesia-like movements in up to 14% of non-geriatric patients never exposed to neuroleptic treatment (Jones & Hunter, 1969; Crane, 1973); in two of these the frequency approached that seen in treated patients (Demars, 1966; Brandon *et al*, 1971). These studies, however, all failed to specify a schizophrenic diagnosis, to control for age, or to exclude organic disease.

Two recent detailed investigations may be more decisive. Owens *et al* (1982) examined 411 chronically hospitalised patients fulfilling research diagnostic criteria for schizophrenia, 47 of whom appeared never to have been exposed to neuroleptic treatment. All patients were assessed for movement disorder twice, using rating instruments, and under blind conditions. At least half showed evidence of tardive dyskinesia-like involuntary movements, and the authors were unable to demonstrate significant differences between treated and untreated patients in the prevalence, severity or pattern of these.

Rogers (1985) examined 100 patients admitted to chronic care prior to the availability of neuroleptic drugs, at least 92 of whom were diagnosed as suffering from schizophrenia. In order not to pre-empt classification as neurological or psychiatric, motor abnormalities were recorded descriptively, in non-prejudicial categories. Amidst many more elaborate phenomena, simple chorea- and athetosis-like disorders were found in 50–75% of patients, including eight who had never received neuroleptic treatment. Furthermore, in nearly 40%, the presence of similar abnormalities had been recorded before any use of neuroleptics:

> For 38 there were more detailed descriptions of various movements which could be brief, random or continuous: having frequent tic-like movements of mouth, cheeks and head; numerous "schizophrenic" tics; their head occasionally jerking back or moving suddenly from one side to another; keep twitching their shoulders, hands and arms; having constant movements of hands fingers and feet . . . or sustained, spasmodic movements: continually squirming about in bed; exhibiting a strange, writhing, snake-like movement of the limbs combined with grimaces which strongly resembled a choreo-athetosis; insane mannerisms such as contorting their body; or insane athetoid movements.

Individually, it is possible to dispute these findings, but taken together they make it difficult to resist the conclusion that involuntary movements indistinguishable from those of basal ganglia disease sometimes form part of the untreated symptom picture of schizophrenia. Such abnormalities appear generally minor, are associated principally with chronic disease, and are probably promoted by neuroleptic treatment (Owens, 1985), though this is by no means a prerequisite. Finally, their occurrence appears inextricably bound up with volitional motor disturbances of an altogether more complex nature, a theme which is developed further in the next section.

The ventral striatum

Animal experimental findings

The mesotelencephalic dopamine system provides projections to two subcortical structures, the nucleus accumbens and olfactory turbercle (Bjorklund & Lindvall, 1984), which until recently were of obscure affiliation. Over the last decade, however, it has become clear on a number of grounds that they should be regarded as part of the neighbouring caudato-putamen (Heimer & Wilson, 1975; Swanson & Cowan, 1975); along with the ventromedial sector of this structure, they are now considered to form a distinct basal ganglia subregion, the ventral striatum.

The ventral striatum receives its principal inputs from limbic system sites, notably the hippocampus and amygdala (Heimer *et al*, 1982). Its subsequent projections parallel and overlap with the classical striopallidal pathway at every step to reach ultimately frontal regions anterior to the premotor cortex

(Heimer *et al*, 1982; Graybiel, 1984). Functional information derives almost exclusively from studies employing selective striatal dopamine alterations. In rats, ventral striatal dopamine depletion has no overall hypokinetic effect, but alters locomotor patterns, impairs exploration and investigation of novel stimuli, and leads to various other difficulties suggesting impaired co-ordination of different classes of behaviour (Koob *et al*, 1978; Robbins & Koob, 1980; Iversen & Fray, 1982; Wallace *et al*, 1983). Conversely, dopamine agonist stimulation results in a state of locomotor hyperactivity relatively uncontaminated by stereotypies, and it has been argued that there is a complex partitioning of such functions between dorsal and ventral striatal sectors (see Joyce, 1983; Mason, 1984). In primates, in addition to hyperactivity, searching, startle responses and the repetitive adoption of inappropriate postures are seen (Dill *et al*, 1979).

Such findings have been virtually unanimously interpreted as indicating a ventral striatal involvement in the motor expression of emotional and motivational processes (Iversen, 1980, Mogenson *et al*, 1980; Iversen & Fray, 1982). The ventral striatum, these authors argue, controls the motor aspects, particularly the head, whole body and locomotor movements, of functions which mediate the animal's arousal by, orientation towards, and investigation of significant stimuli in the environment. This is the behaviour which invariably precedes the organisation of specific motor responses to such stimuli.

As has been noted (Crow, 1973; Iversen & Fray, 1982) such concepts are essentially similar to those of incentive and approach in learning theory. In various theoretical formulations (Mowrer, 1960; Bolles, 1975; Gray, 1975; Bindra, 1978), stimuli associated with reward acquire the ability to induce a state of motivational arousal, which in turn generates the flexible, relatively unstructured responses of approach. These serve to guide the animal towards the motivational stimulus and give way to formal instrumental responses as this is reached. According to Gray (1975), approach behaviour depends ultimately on a positive feedback mechanism and this, he argues, is mediated by reinforcement.

Human ventral striatal pathology

The ventral striatum has been linked to human disease states only in a preliminary way, as expected to phenomena in some way related to those of basal ganglia disease. A hypofunction has been implicated in some of the more subtle akinetic abnormalities in Parkinson's disease (Young & Penney, 1984). Among hyperfunctional phenomena, tics show an intimate association with basal ganglia disorders (Jung & Hassler, 1960; Weingarten, 1968), and various authors (Fahn, 1982; Nauta, 1982; Young & Penney, 1984) have speculated on the involvement of the ventral striatum in their pathogenesis.

In addition, some basal ganglia disease states appear to be marked by other hyperfunctional phenomena related to, but going beyond, those of chorea, athetosis and dystonia. In encephalitis lethargica, Von Economo (1931) described intricate, elaborate, often grotesque paroxysms of motor activity accompanying the insidious development of Parkinsonism. Wilson (1940) also drew attention to "more leisurely and complex movements, distinct from chorea, which recall those of tic but are slower, more purpose-like and even more compound". With l-dopa therapy, a massive potentiation of this type of abnormality was observed to take place (Sacks, 1983). In the contemporary condition of tardive dyskinesia, numerous authors (Jones & Hunter, 1969; Mackay, 1982; Fahn, 1984; Lees, 1985) have described movements which are repetitive or rhythmical, rather than choreo-athetoid in nature. One widely used rating scale for the disorder (Simpson *et al*, 1979) even includes items for 'grimacing', 'caressing or rubbing face or hair' and 'crossing and uncrossing legs' – phenomena classed uneasily alongside the random, shifting, fragmentary movements of choreo-athetosis.

Ventral striatal dysfunction in schizophrenia?

The ventral striatum, by virtue of the dopamine hypothesis, has been repeatedly incriminated as a site of dysfunction in schizophrenia (Stevens, 1973; Crow *et al*, 1977; Matthysse, 1981; Iversen & Fray, 1982). At the same time, the resemblance between the elaborate, integrated motor phenomena that could reflect its dysfunction, and some of the catatonic symptoms of schizophrenia, has regularly struck phenomenologically minded authors (Farran-Ridge, 1926; Von Economo, 1931; Sacks, 1983; Sims, 1987). Combining these preclinical and clinical findings, therefore, it may be possible to arrive at interpretations of some fundamental catatonic phenomena.

Anatomically, the ventral striatum receives a selectively limbic afferentation, its connections overlap with those of the basal ganglia proper, and it is considered to mediate the motor expression of emotional and motivational processes. Contributing to a basal ganglia movement disorder, it might thus lend otherwise unremarkable involuntary movements a certain emotional toning, a motivational quality not ordinarily seen. In schizophrenia just such a symptom is occasionally encountered as parakinesia (Kleist, 1960), described by Fish (1957) as follows:

> Here the leading symptoms consist of fragmentary movements which resemble those of chorea, athetosis and the tics. Although they look like involuntary movements they can always be imitated and are partly reminiscent of expressive and intentional movements.

Functional studies suggest that the ventral striatum is concerned with the integration and strategic co-ordination of motor behaviour rather than the elaboration of individual responses. This has been considered clinically relevant to the occurrence of tics and other complex motor abnormalities. A catatonic symptom, mannerisms, however, has rather similar characteristics. Bleuler (1911) in fact stated that in many cases mannerisms could not be distinguished from tics. With the complex tics seen in l-dopa treatment of post-encephalitic Parkinsonism (Sacks, 1983) and in Gilles de la Tourette's syndrome (Marsden *et al*, 1983; Lees, 1985), which may also have a basis in dopamine excess (Devinsky, 1983), the differentiation from mannerisms becomes especially problematic. According to Rogers (1985), the two abnormalities may be phenomenologically one and the same.

But ultimately, it may be that the basal ganglia and ventral striatum govern different aspects of a single superordinate function, the learning and performance of instrumental behaviour under the influence of reinforcement. In animals, adventitious reinforcement results in a syndrome of 'superstitious' behaviour, where instrumental acts, often stylised or incomplete, are performed repetitively (Matthysse, 1981). In man, a dopamine excess affecting the two striatal sectors in a balanced way might give rise to a comparable phenomenon, with the appearance of largely normal-appearing goal-directed acts made abnormal by virtue of repetition. But this of course is a description of a catatonic symptom central to schizophrenia, which, years before the investigation of dopaminergic drug effects in animals, had already been given the name stereotypy (Bleuler, 1911; Kraepelin, 1913).

More detailed examination of dopaminergic drug actions provides independent support for a relative rather than absolute distinction between involuntary movements and catatonic symptoms. In rats, the behavioural response is stereotyped head, limb or whole body movements (Iversen, 1977); more elementary movements are not seen or are obtained only with difficulty (Waddington *et al*, 1983). In cats and dogs, a greater variety of phenomena begin to appear, and in monkeys the abnormalities range from simple tongue and lip movements, through nodding, bowing and rocking, to activities like opening paws in succession or following a fixed route repeatedly (Randrup & Munkvad, 1967; Ellinwood *et al*, 1973; Schiorring, 1981). The equivalent human syndrome, seen in stimulant-drug abusers, encompasses choreo-athetosis, simple repetitive movements, incessant tidying, grooming and mechanical activities, even the writing and destroying of hundreds of fraudulent cheques (Rylander, 1972; Lundh & Tunving, 1981; Schiorring, 1981; Lees, 1985). In short, as the evolutionary scale is ascended a central syndrome of stereotypy progressively diversifies, with involuntary movements at one end of the spectrum and, at the other, phenomena indistinguishable from schizophrenic stereotypies.

The septo-hippocampal system

Animal experimental findings

Prominent limbic projections of the mesotelencephalic dopamine system include those to the entorhinal cortex and lateral septal area (Bjorklund & Lindvall, 1984). It is clear, however, that these are components of a well defined structural complex with the hippocampus as its core, the septo-hippocampal system (Swanson, 1978). The hippocampus itself may also receive a dopamine innervation, though only indirect evidence exists for this (Bjorklund & Lindvall, 1984).

The septo-hippocampal system receives important inputs from sensory association cortex, the prefrontal cortex and the amygdala; its outputs reach the cingulate gyrus, the ventral striatum and the hypothalamus among other sites; its circuitry otherwise features a number of loop systems (Swanson, 1978; Gray, 1982; Graybiel, 1984). A huge body of experimental data has revealed no simple motor, sensory, learning or emotional functions, but has pointed instead to important modulations of behaviour at a fairly sophisticated level (Grossman, 1978; O'Keefe & Nadel, 1978; Gray, 1982).

The diverse theoretical approaches to septo-hippocampal function have recently been reconciled and integrated by Gray (1982): he proposes that the septo-hippocampal system acts quite generally to compare 'actual' with 'expected' in an animal's environment. On a moment-to-moment basis, the system receives 'actual' sense data via its sensory association area inputs, and matches these with 'expected'; that is, predictions based on previous experience generated in the cingulate and prefrontal cortex. When 'actual' and 'expected' coincide, the system functions passively, permitting continued elaboration of the current motor plan and passing on information that a match has taken place for storage elsewhere. But if, because of environmental change, 'actual' and 'expected' no longer agree, the septo-hippocampal system takes active control of behaviour: on-going behaviour is inhibited, arousal increases, and alternative exploratory and investigative strategies are instituted. This active state of behavioural inhibition constitutes an animal version of the human experience of anxiety.

The ascending monoamine systems provide a further important class of inputs to the septo-hippocampal system, and almost certainly exert significant modulatory effects (Gray, 1982; Deakin, 1983). In the context of Gray's theory, noradrenaline and serotonin serve to label incoming stimuli as 'important' or 'important, associated with punishment', enhance environmental checking, and bias septo-hippocampal operation towards behavioural inhibition. Dopamine (Gray, personal communication) is liable to have somewhat different effects. It may also act to label incoming stimuli as 'important' (perhaps as 'important, associated with reward') but, in line with its general reinforcement affiliations, it should antagonise mismatch detection and behavioural inhibition and promote matching and the passive facilitation of behaviour.

It is no accident that Gray's theory is constructed around the premise that animals form and verify predictions. This in fact represents the most important recent development in learning theory. It has been established beyond reasonable doubt that animals model, predict, or otherwise compute expectancies in procedures including habituation (Gray, 1975), classical conditioning (Dickinson, 1980) and instrumental learning (Mackintosh, 1983; Toates, 1986). In the latter two procedures the computations performed revolve principally around reinforcement; in particular, the degree to which stimuli and responses are predictive of reward and punishment.

Human septo-hippocampal pathology

Damage to components to the septo-hippocampal system in man figures prominently in the long term, largely anterograde amnesia seen in Korsakoff's psychosis (Victor *et al*, 1971) and following bilateral medial temporal lobectomy (Milner, 1970; Butters & Cermak, 1975). While this finding is unexpected on the basis of animal work, equally the amnesic syndrome is a far from straightforward phenomenon.

The outstanding deficit appears to be in retrieval, rather than the encoding and storage of information (Weizkrantz, 1978; Baddeley, 1982). As retrieval almost certainly involves systematic checking and evaluation (Baddeley, 1982) and is marked in the amnesic syndrome by interfering effects reminiscent of those seen after animal hippocampal lesions, a degree of reconciliation between the animal and human data can be achieved (Weizkrantz, 1978; Winocur, 1982; Gray, 1982).

The amnesic syndrome is also surprisingly circumscribed: affected patients show near-normal learning and retention in a variety of perceptual and motor tasks, their access to vocabulary and other classes of 'semantic' information remains intact, and the memory defects appear to be largely restricted to personal, autobiographical, 'episodic' events (Baddeley, 1982; Cohen, 1984). Some recent theorising emphasises the reciprocal interactions that must take place between 'episodic' and 'semantic' memory (Tulving, 1983). It has also been suggested that the amnesic syndrome may be best understood not simply in terms of memory, but as a disruption of certain processes in the formation, maintenance and expression of 'declarative' knowledge (Hirsh & Krajden, 1982; Squire, 1983; Cohen, 1984) – the propositional 'knowing that', stored factually, as opposed to the procedural 'knowing how', stored as instructions.

Septo-hippocampal dysfunction in schizophrenia?

The possibility that a specific disturbance of septo-hippocampal function may take place in schizophrenia is raised by its dopamine innervation and reinforced by recent neuropathological findings (Scheibel & Kovelman, 1981; Bogerts *et al*, 1985; Brown *et al*, 1986). Furthermore, a key aspect of Gray's (1982) theory is its application to human psychiatric disorders, not only anxiety but also depression and obsessional states. The question thus arises of how a dopamine excess might affect septo-hippocampal function, and what particular schizophrenic symptoms this might form a basis for.

Within the framework of Gray's (1982) theory, it is reasonable to suppose that dopamine, like other monoamine inputs, serves to label incoming stimuli as 'important', perhaps specifically as 'important, associated with reward'. A dopamine excess would then cause neutral stimuli to become erroneously identified in this way. But according to phenomenologists (Schneider, 1949; Jaspers, 1959; Sims, 1987), the abnormal attribution of significance to neutral events is at the heart of at least some classes of delusion. In the view of Jaspers (1959):

> A basic feature of the first experience of delusional meaning is "the establishment of an unfounded reference."

> Significance appears unaccountably, suddenly intruding into the psychic life. Later the identical experience of significance is repeated, though in other contexts. The trail is blazed and the preparedness for the significant experience then permeates almost all perceived contents.

This line of argument can be taken further. According to contemporary theory, animals model events, make predictions, compute expectancies; the verification of these may take place in the septo-hippocampal system; in man, this function becomes one of formation, maintenance and expression of 'declarative' knowledge. The unmistakable implication is that there exists a specific neuronal machinery for belief, a machinery which, furthermore, is liable to be undermined by dopamine excess: in Gray's theory (Gray, personal communication) dopamine may bias septo-hippocampal function towards its passive, checking mode of operation, where predictions are confirmed as matched. Miller (1984) similarly argues that dopamine acts to "set the threshold for inductive inference", an excess permitting associations which would normally be rejected as coincidental to become established in memory. In other words, the plausible effects of a septo-hippocampal dopamine excess might be not only to induce an erroneous attribution of significance – or delusional meaning – but also to promote the generation of abnormal inductive inferences – or delusional beliefs.

Independent considerations also argue for a septo-hippocampal role in delusion formation. The single 'positive' symptom of Korsakoff's psychosis, confabulation, may share aspects of its phenomenology with delusion. As noted by Kraepelin (1909), confabulations are held with "rock-like certainty"; sometimes they are adhered to tenaciously for long periods (Jaspers, 1959; Talland, 1961); their content may be grandiose, persecutory or even fantastic (Kraepelin, 1909; Talland, 1961; Berlyne, 1972). At times, in fact, it may be difficult to decide whether the false statement of a Korsakoff patient is a confabulation or a delusion (Cutting, 1978). Finally, the occurrence of isolated delusions in Korsakoff's psychosis is well documented (Cutting, 1978); conversely, occasionally seen in schizophrenia is the striking phenomenon of delusional confabulation (Kraepelin, 1913).

The prefrontal cortex

Animal experimental findings

The prefrontal cortex is commonly defined as the area of frontal lobe anterior to the premotor regions which has reciprocal connections with the mediodorsal thalamic nucleus (Nauta, 1971). Across a range of animal species, however, the mediodorsal projection field coincides with the major site of neocortical dopamine innervation (Bannon & Roth, 1983; Bjorklund & Lindvall, 1984), suggesting that the prefrontal cortex is a specific target of the mesotelencephalic dopamine system.

The prefrontal cortex is a site of ultimate convergence for cortical sensory association projections in all modalites (Jones & Powell, 1970) and also has close connections with the limbic system, basal ganglia, ventral striatum and premotor cortex (Nauta, 1971; Goldman-Rakic, 1984; Nauta & Domesick, 1984). Functionally, while extensive prefrontal lesions are followed by apathy, emotional changes and over-responsiveness to stimuli (Fuster, 1980), more detailed lesion and electrical recording studies (Warren, 1964; Mishkin, 1964; Fuster, 1980) have indicated a particular role in the elaboration of behaviours requiring a high level of cognition. There is evidence of specialisations of function within different prefrontal sectors, but the common theme of these may be involvement in tasks requiring choice from a repertoire of recurring familiar alternatives whose relevance to reward changes from trial to trial (Rosenkilde, 1979; Fuster, 1980).

The various general theories of prefrontal cortex function (see Stuss & Benson, 1985) all show tantalising similarities and point to some executive role involving the formation, monitoring and revision of behavioural strategies or central response sets. The formulation drawing most heavily on animal work, however, is that of Fuster (1980): he argues that the prefrontal cortex has a superordinate function of giving temporal structure to complex, goal-directed behaviour. By means of three subsidiary functions, the prospective anticipation of events (a proposal similar to that in Gray's (1982) theory), the retrospective holding of information, and the attentional control of interference, the prefrontal cortex assembles complex schemas for keeping behavioural sequences on course to their goals. These schemas are, above all, active and intentive in nature; there is a continuous analysis, updating and referring back of information to the overall plan to determine the individual responses most suitable at each step.

The dopamine projection to the prefrontal cortex has complex electrophysiological properties (Bannon & Roth, 1983; Glowinski *et al*, 1984). At a behavioural level, its effect appears to be essentially facilitatory: Brozoski *et al* (1979) found that dopamine depletion in a particular region of monkey prefrontal cortex reproduced the typical impairments seen after destructive lesions. The effect was specific to dopamine and could be reversed by systemic administration of dopamine agonists.

Even here, there are affinities with learning theory concepts. Central to instrumental learning is the relationship between stimuli and responses – the way in which presentation of a particular stimulus comes to elicit performance of a particular response. Although originally considered simple, a great deal of subsequent work has made it clear that this process must involve selective attention, expectancy analysis, and the formation of 'representations' between stimulus, response and reinforcer (Mackintosh, 1975, 1983). But these are just the functions which have, quite independently, been attributed to the prefrontal cortex; this has been noted and preliminary attempts at integration have been made (Pribram, 1973; Gray, 1982; Mesulam, 1986).

Human prefrontal cortex pathology

The consequences of prefrontal damage in man are well known as the frontal lobe syndrome. The psychomotor, emotional and social aspects of the clinical picture have been described in detail and have clear counterparts in the animal lesion syndrome (Lishman, 1978; Fuster, 1980; Stuss & Benson, 1985). At the same time, a group of cognitive abnormalities are recognised: thinking becomes concrete, narrowed in scope and lacking in inventiveness. Simple, familiar tasks continue to be peformed without difficulty, but behaviour requiring planning, and the active pursuit of prospective goals can no longer be sustained. If attempted, significant information is disregarded in a characteristic way (Luria, 1973; Fuster, 1980; Stuss & Benson, 1985). On formal testing, in procedures like the Wisconsin Card Sorting Test (Milner, 1963) frontal lobe patients learn the initial sorting principle without difficulty, but fail to adjust their strategy when this is unpredictably changed, despite continuous feedback that they are in error.

As in animals, the human prefrontal cortex shows evidence of regional specialisation of function (Lishman, 1978; Milner & Petrides, 1984; Stuss & Benson, 1985). In addition, the dominant-sided prefrontal cortex has particular verbal functions; patients with lesions here show reduced fluency and are unable to generate normal numbers of words in given semantic categories (Milner & Petrides, 1984). A subregion of dominant prefrontal cortex appears to have an even more specialised language function: lesions just anterior to Broca's area give rise to 'central', 'frontal dynamic' or 'transcortical motor' aphasia (Goldstein, 1948; Luria, 1970; Benson, 1979), characterised at its purest by impoverishment of vocabulary, reduction in the length and complexity of sentences, and difficulties initiating and sustaining narrative speech.

Prefrontal cortex dysfunction in schizophrenia?

The question of abnormal prefrontal function in schizophrenia was raised by Kraepelin (1913), Bleuler (1911) and Kleist (Fish, 1957; Kleist, 1960), has been re-opened in contemporary work (Oades, 1982; Levin, 1984; Cutting, 1985; Muller, 1985), and is recently the recipient of notable experimental verification (Weinberger *et al*, 1986). The way such a dysfunction would be reflected clinically, however – in particular, the positive symptoms a prefrontal dopamine excess might give rise to – has attracted relatively little attention, though amongst other things conceptual, verbal, and linguistic phenomena might be anticipated. A class of positive symptoms with such characteristics is of course recognised in schizophrenia as formal thought disorder.

In animals an important, perhaps superordinate, prefrontal cortex role is the formation, monitoring and revision of cognitive schemas for goal-directed behaviour. In man, there are involvements in analogous aspects of conceptual thinking. Dopamine has facilitatory effects (Brozoski *et al*, 1979) and an excess might therefore induce an abnormal heightening of this function. Explanations of the discourse abnormality in schizophrenic thought show intriguing similarities: the loss of direction by "any unifying concept of purpose or goal" (Bleuler, 1911), and the "inability to restrict, eliminate and focus on the task in hand" (Cameron, 1944) are reminiscent of an overfacilitated generation of goal-directed conceptual schemas. The abnormality of 'derailment', closely related to 'loss of goal' (Andreasen, 1979), could reflect an increase in the moment-to-moment monitoring and revision of these.

More substantially, it is clear that the dominant prefrontal cortex has particular verbal functions, and a subregion specifically linguistic functions. If a dopamine excess disturbed function here, clear-cut semantic and dysphasic abnormalities might be anticipated to follow. A well documented element of schizophrenic thought disorder is the tendency to use imprecise approximations to words (Cameron, 1944; Andreasen, 1979): a glove is called a "handshoe", a pen a "paperskate", a patient stated he "had menu three times a day". The occurrence of specific dysphasic signs in schizophrenia also appears well established (Morice, 1986). In addition to agrammatism, a feature of Broca's aphasia, Kleist (1960) described paragrammatism where speech remained fluent but contained multiple grammatical misuses. Paragrammatism and another well known schizophrenic phenomenon, neologism, are also seen in jargon aphasia (Butterworth, 1985; Howard, 1985), whose general similarities to thought disorder have attracted attention (see Rochester & Martin, 1979; Brown, 1981). But the relevant phenomena remain complex, and their categorisation as linguistic or conceptual remains controversial. In the words of Weinstein (1981):

> Speech contains ornate phraseology, pedantic redundancies and 'high sounding' malaproprisms. When one patient was asked what was wrong with him, he replied with "Well it has been suggested that there were certain oddities and restrictions, technically the activities of the student body, so to speak." Even when speech has improved and the patient no longer has neologisms, he tends to make unusual word choices. For example, one man commented on the 'congestion' of his speech, and another recalled having done a lot of 'verbal gambling' with words. When tested for ability to generate rhymes, alliterations, synonyms, antonyms and homonyms, patients produce strings of impressive sounding but inappropriate words. A woman 'rhymed' the following: "ovoid, ovum, spectrum, parallax, quadrangle, trigonometry, algebra." Some subjects speak officialese. The use of alliterations and assonances and an exaggerated rhythm may impart a poetic quality.

– a passage which is not, in fact, a description of schizophrenic thought disorder but was instead drawn from an account of the clinical features of jargon aphasia.

The available independent evidence tends to follow the theme developed here. Perhaps the best established abnormality in schizophrenic thought is overinclusiveness (Payne, 1973). Consistent with a prefrontal dysfunction, overinclusiveness has been considered to represent the inverse of concrete thinking (Reed, 1970; Schwartz, 1982); it is typically demonstrated using sorting tasks (Payne, 1973); in a recent review (Schwartz, 1982), it was concluded that its basis could lie in 'pigeon-holing' – or the adoption of central response sets. At a verbal level, in contrast to the inability to generate semantic category words seen with dominant prefrontal lesions (Milner & Petrides, 1984), schizophrenic patients show increased word usage and choose distantly related words (Payne, 1973). Finally, severe schizophrenic thought disorder is not always distinguishable from dysphasia (Faber *et al*, 1983), and while it does not equate with known regional syndromes, it has recently been argued to be consistent with disorder in an 'executive' speech centre – located in the dominant prefrontal cortex (Morice, 1986).

Conclusion

By examination of the effects of dopamine on different brain systems, and with the assistance of its conceptualised role as reinforcement, some interpretations of the extraordinary clinical picture of schizophrenia can be attempted. The relevant symptoms, however, are far from exhaustive, and if such an approach has validity it should have broader applications. A few tentative steps in this direction can be outlined as follows.

Some versions of the dopamine hypothesis propose that an episodic dopamine excess may be superimposed on a more fundamental dopamine deficiency state (Chouinard & Jones, 1978; Mackay, 1980), this manifesting itself in the negative or defect state symptoms of schizophrenia. The commonplace simultaneous presence of positive and negative symptoms might appear incompatible with such a view, but has precedent in that tardive dyskinesia and Parkinsonism can simultaneously co-exist (Gerlach, 1979). Deficiency in a transmitter whose overall role is one of facilitation of voluntary, motivated or instrumental behaviour is of course in keeping with the important negative symptom of lack of volition (Andreasen & Olsen, 1982). More specifically, dopamine depletion in the prefrontal cortex has lesion-like effects (Brozoski *et al*, 1979) and the resemblances between the frontal lobe syndrome and the schizophrenic defect state have been noticed from the beginning of the century (Bleuler, 1911; Kraepelin, 1913; Kleist, 1960). But most provocatively, the prefrontal cortex contains a distinct speech area, lesions of which produce a loss of syntactic complexity (Morice, 1986) and "a reduction of spontaneous speech, a curtailment of the amount and the range of narrative expression" (Fuster, 1980), abnormalities tantalisingly similar to the negative schizophrenic symptom of poverty of speech.

The mesotelencephalic dopamine system also provides projections to nuclei of the amygdala, the piriform cortex and the bed nucleus of the stria terminalis (Bjorklund & Lindvall, 1984), implying a significant modulation of the amygdaloid complex (Ben-Ari, 1981). Anatomically, the amygdaloid complex is an interface between sensory association cortex and the hypothalamus, it has functions in the co-ordination of emotional reactions to perceptual stimuli, and, of relevance to learning theory, it may process the affective qualities of reinforcing events (Kapp & Kesner, 1981; Mishkin & Aggleton, 1981). There is thus no doubt about its involvement in emotional processes, and the scope for application to the bizarre and incongruous mood accompaniments of active schizophrenic episodes is obvious. Furthermore, if the net result of an amygdaloid dopamine deficiency was to impair the co-ordination of emotional responses to perceived events, this might not be an unreasonable model for the negative symptom of affective flattening.

Finally, no examination of schizophrenic psychopathology would be complete without some statement on the baffling and perhaps most peculiarly schizophrenic symptom of all. One site of mesotelencephalic dopamine innervation so far given little consideration is the anterior cingulate cortex (Bjorklund & Lindvall, 1984). In primates, this appears to play a leading role in the voluntary initiation of vocalisation (Jurgens & Pratt, 1979), and has recently also been shown (Muller-Preuss *et al*, 1980) to have direct anatomical links with a region of the superior temporal gyrus known to be concerned with the recognition of species-specific phonation. These authors speculate that this connection between vocalisation-initiating and auditory-decoding areas might be important for vocal learning, or could, on a moment-to-moment basis enable the animal to 'subtract' its own on-going or intended vocalisations from the rest of the acoustic information reaching it. Depending on attentional needs, either self-produced sounds or the environmental background could then be screened out. If an analogous anatomical arrangement existed in man, if it were devoted to the particular species-specific vocalisations of speech, if its 'subtractive' function were deranged by a dopamine excess, might it not be verbal auditory hallucinations that would be the result?

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Unravelling the Relationship between Genetic and Environmental Risk Factors in Psychiatric Disorders

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Murray *et al* (1985) have proposed a method for using biological markers and information about family history to reduce the heterogeneity in a disease such as schizophrenia. They propose that families which are heavily loaded with illness are most likely to be segregating for a major locus and therefore should be used for studies of genetic marker or other biological traits that are thought to be related to a genetic etiology. They propose that patients without a family history (sporadics) of an illness should be investigated for hypothesised environmental components since they are the cases where environmental factors are most likely to play a large role. They give an example from their own data on Ventricular Brain Ratios (VBR) in a sample of schizophrenic twins (Reveley *et al*, 1984) where schizophrenic twins without any family history (FH−) of a major psychosis had significantly larger ventricles than did schizophrenic twins with a family history (FH+). They conclude that while there is a genetic determinant to ventricular size within the normal range, large ventricles reflect an environmental aetiology that is more important in sporadic cases than in those with a familial pattern to the illness. Subsequently, Reveley & Chitkara (1985) found that singleton schizophrenic patients who were FH− had a significantly larger VBR than did controls while FH+ patients had a mean midway between FH− patients and controls. While this is an attractive hypothesis for the structural brain changes seen in some schizophrenic patients, results of other studies are not consistent with these findings (Nasrallah *et al*, 1983; Schulsinger *et al*, 1984; Owens *et al*, 1985; DeLisi *et al*, 1986). For example, in our own data (DeLisi *et al*, 1986), 'familial' schizophrenics had larger ventricles than did controls and risk factors thought to be environmental (head injuries and birth complications) were found to be present in that sample.

The classification of patients into FH+ and FH− groups has been used in a number of other investigations of biological traits in psychiatric patients. For example, Sedvall *et al* (1980) found that FH+ schizophrenics had higher CSF 5-HIAA and HVA than did FH− patients. However, these were preliminary findings, the number of FH+ patients was very small, and the study has not been replicated. Baron & Levitt (1980) found that FH+ schizophrenics had significantly lower MAO activity than controls and FH− patients did not differ from controls. Here again, the sample was small and other investigators have not confirmed the finding (Belmaker *et al*, 1977; Duncavage *et al*, 1982; Maj *et al*, 1984).

Some of the problems with this methodology have previously been raised by us (DeLisi *et al*, 1985). These include: (1) the fact that this comparison assumes that a substantial number of cases have a non-familial aetiology; (2) some patients may not have enough available relatives to classify confidently as FH+ or FH−; (3) The definition of what constitutes FH+ is somewhat arbitrary; and (4) some FH− patients will be of the genetic type. In this paper, we discuss these criticisms in more detail and try to determine which strategies are most likely to be powerful in identifying risk factors that can separate psychiatric patients into more homogeneous groups.

Problems of dividing patients into sporadic and familial cases

Existence of a non-familial aetiology

It is well accepted that environmental factors do play a role in the development of schizophrenia. It is also clear that the symptoms of schizophrenia can be caused by drugs and other known environmental agents. However, we do not know the extent to which other *unknown* environmental factors cause schizophrenia. By dividing patients into FH+ and FH− groups, one assumes that the environmental factors of interest to the investigators are playing a *much* larger aetiological role in the FH− patients.

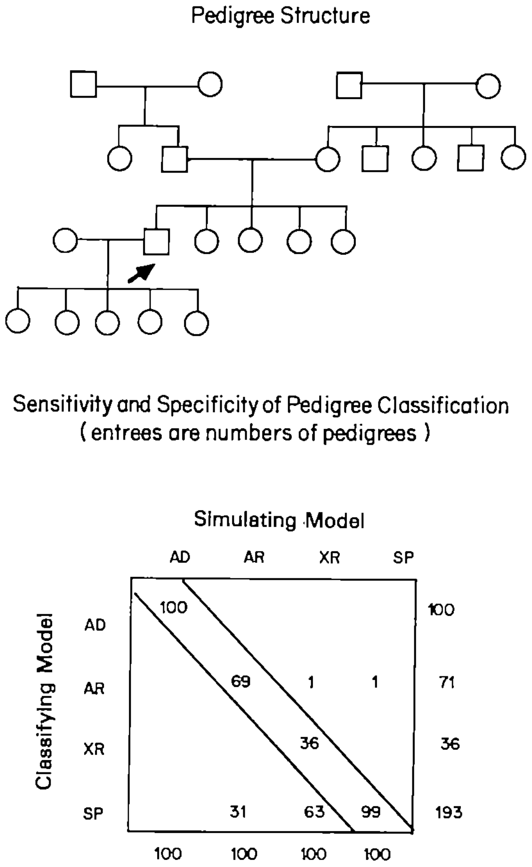
Sample size of relatives and definitions of FH+

If the family is very small, or information is only available on a few relatives, then it may not be possible to classify the family history type of the patient confidently. In addition, assumptions must be made about the disorders in relatives which should be considered as positive (i.e. genetically related to the disorder in question). It might seem reasonable to consider a spectrum of major psychiatric disorders in the relatives as a positive family history. However, some disorders may be common in the population and if many relatives were examined, it would not be unexpected to find additional cases. In such situations, it might be useful to classify patients taking the family size and population prevalences into account to determine if each family has significantly more cases than expected based on population frequencies. Furthermore, one must also consider how many relatives have not passed the age of risk at the time of data collection. Thus, it would also be necessary to know the age of onset distributions. Fortunately, data on population prevalences from recent epidemiological studies of psychiatric disorders are available (e.g. see Robins *et al*, 1984) although data on the prevalence of some of the milder disorders are not. For example, the population prevalence of schizotypal personality disorder, which is hypothesised to be part of the schizophrenia spectrum, is not known.

Heterogeneity of FH− group

Assuming that patients have enough relatives to be classified as FH+ or FH−, familial and sporadic groups, this classification is inherently heterogeneous because certain genetic mechanisms are expected to give rise to significant numbers of families that appear to be sporadic. A recent simulation study by Beaty & Boughman (1986) illustrates the effects of genetic heterogeneity for a disorder on the accuracy of subdividing a sample of families according to the most likely subtypes. They used retinitis pigmentosa as an example of a genetic illness with known heterogeneity. For the simulation, they chose a variety of pedigree sizes and structures ranging from small nuclear families to moderate size pedigrees of five generations and 20–30 individuals. For each pedigree, starting with one affected individual as the proband, they simulated 100 replicates each of four different models of inheritance (autosomal dominant, autosomal recessive, X-linked recessive, and sporadic). They then classified each simulated pedigree into one of the four categories based on which model had the highest relative likelihood in that pedigree.

They found that generally, families classified into the genetic types were homogeneous. For example, most of the families classified as autosomal dominant were correctly classified; that is, they had been simulated with the autosomal dominant model. The pedigrees classified as autosomal recessive were somewhat more heterogeneous. However, pedigrees classified as sporadic were the most heterogeneous group. On the whole, about half of those pedigrees classified as sporadic had been simulated with recessive inheritance. This was even true for pedigrees that were relatively informative (i.e. four generations and about 20 individuals). An example of typical results from this study is shown in Fig. 1. The pedigree used for this example has four generations and relatively large sibships. The 4×4 table gives the results of classification for the 100 pedigrees of this structure simulated for each of the four models. The numbers in the table indicate the sensitivity, i.e. the proportion of each simulated type correctly classified, and the specificity, i.e. the proportion of each classified type that were the correct type. Thus one can see that if the inheritance of the disease is autosomal dominant, both sensitivity and specificity are 100%; however, for the sporadic model, sensitivity is nearly 100% but specificity is only 51%. That is, only 99/193 pedigrees classified as sporadic had been simulated with the sporadic model. The remainder were pedigrees simulated with recessive inheritance misclassified as sporadic.


Pedigree Structure

Sensitivity and Specificity of Pedigree Classification (entrees are numbers of pedigrees)

FIG. 1. Example of simulation results from study of Beaty & Boughman (1986). One hundred pedigrees of this structure were simulated for each of the following models of inheritance: Autosomal Dominant (AD), Autosomal Recessive (AR), X-linked Recessive (XR), and Sporadic (SP). Each of the 400 pedigrees were classified according to the most likely mode of inheritance. The entrees in the 4×4 table are the results of simulated *vs* classified categories, the columns being the simulated type and the rows being the classified type. Measures of *sensitivity* are given by the diagonal/column total. Measures of *specificity* are given by diagonal/row total. For example, the AD model has 100% sensitivity and specificity whereas the SP model has 99/100 (99%) sensitivity and 99/193 (51%) specificity.

In our own previous simulation studies (Goldin *et al*, 1984), we were interested in trying to determine the accuracy of parameter estimates and hypothesis testing for different modes of inheritance. We also found that when the transmission was recessive or intermediate (i.e. heterozygote penetrance was 5–15%), the genetic parameters could not be reliably estimated and the major locus could not be consistently detected. We also found that many pedigrees simulated under these models looked sporadic (i.e. no other individuals were affected aside from the proband) which was likely to be the reason that the correct mode of inheritance could not be determined. For any real disorder, the heterogeneity of the group labelled sporadic will depend on the actual proportion of different types of families in the population and the size of the families being studied. Thus, it appears that if a recessive subtype of illness exists, many patients who have that subtype will come from families with no other affected relatives and would be classified as sporadic. The group called familial will only be heterogeneous to the extent that there exist different genetic types of illness.

This heterogeneity of familial and sporadic subgroups has consequences for biological risk factor studies. If there are some environmental factors in illness that play a much larger role in sporadic illness than in genetic illness, these can be detectable in FH+ *vs* FH− studies. However, large samples of patients may be required to detect such factors consistently since the sporadic subgroup will be a mixture of different aetiologies. On the other hand, biological traits that reflect genetic defects may be detectable in studies of familial patients.

Alternative approaches to clarifying the role of risk factors

We would argue that simply classifying patients into familial *vs* sporadic groups is a strategy that lacks power. Clearly the power could be increased if the familial risk could be quantified more exactly than simply as positive or as negative. One could then correlate a biological trait in a patient with the degree of risk in his family. Unfortunately, this may not be practical for most studies because family sizes are not usually large enough for accurate estimates of risk to be made. Another approach could be to classify patients into categories based on biological traits and then compare the morbid risks in relatives of the different subgroups. This strategy has proved useful in detecting clinical heterogeneity related to risk of illness in relatives in large family studies of affective disorders (Gershon *et al*, 1982; Leckman *et al*, 1983). Here again, relatively large numbers of families are needed in order to be able to estimate the risk in subgroups. In addition, for biological traits, it would be necessary to create some cut-off values in order to classify the patient. Often, cut-off values for a continuous trait are arbitrary and do not have any biological meaning.

Conclusion

Reveley *et al* (1985) argue that schizophrenia is analogous to coronary heart disease (CHD) where a rare monogenic form (familial hypercholesterolemia) exists in addition to the common form. Both share some of the same environmental risk factors, but these factors will play a much larger role in the common (non-familial) form. This may be analogous to schizophrenia. However, the ability to detect differences by comparing 'familial' and 'common' forms will depend on the role played by the risk factors in each group. For example, cholesterol levels might not differentiate familial CHD from non-familial since it may be a marker for a common final pathway leading to CHD in both types of patients. In fact, the rigorous study of the genetically determined receptor defects leading to familial hypercholesterolemia has provided a model for the mechanism behind dietary induced hypercholesterolemia. (It is thought high dietary cholesterol suppresses the number of liver LDL receptors which results in high serum cholesterol, which is the same end result obtained with decreased receptor numbers due to genetic defects.) With respect to schizophrenia, markers that directly reflect gene defects will contribute information about genetic subtypes of illness. However, markers that reflect environmental factors may not be detected by simply comparing sporadic and familial patients. Focusing studies on patients that are most likely to have a genetic form of the illness may clarify the underlying biological mechanisms and thus also define the roles played by environmental factors.

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The Familial/Sporadic Distinction as a Strategy in Schizophrenia Research

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The rationale and limitations of discriminating between cases of schizophrenia with and without a family history are reviewed. It is concluded from the evidence available that, by identifying subgroups of greater aetiological homogeneity, the strategy can be a useful starting point for research into likely causes.

Research into cause in schizophrenia has lately been aided by standardised methods of interview and diagnosis and the arrival of a variety of techniques to measure, *in vivo*, aspects of cerebral morphology and function. As yet no clear answers have emerged, although it seems that such answers may now be within the scope of the combined efforts of epidemiological and biological psychiatry. Unfortunately, the search for causes is often hampered by inadequate reference to two important principles, one concerning the nature of schizophrenia and one the method of the search itself.

The first principle is that aetiological research must adhere strictly to the concept of schizophrenia as a *syndrome* defined by a pattern of symptoms and signs, without reference to the presence or absence of any presumed cause. This is the case in ICD-9 (WHO, 1978) but confusion arises if, as in the Research Diagnostic Criteria (Spitzer *et al*, 1975) and DSM-III (American Psychiatric Association, 1980), the diagnosis is explicitly disallowed when organic cerebral disease is known to be present. The relegation of those schizophrenias which arise in the context of cerebral disease to the status of "schizophrenia-like psychosis", or, in DSM-III terms, "organic mental disorder", leads to insurmountable circularity. Where is the sense in searching for the organic substrate of a disorder which is deemed only legitimately to exist in the absence of such a substrate? The problem has been thrown into sharp relief by the advent of the new brain imaging techniques, since the frequency with which schizophrenia is found to be accompanied by cerebral abnormalities depends on the sensitivity of the instrument used to look for them. To insist on excluding cases where the syndrome seems secondary to organic brain disease is to risk throwing out the aetiological baby with the nosological bathwater.

It is our view that schizophrenia is best viewed as the final common pathway of a range of different processes. This notion is not new (Mapother, 1937), but it brings us to the second general principle: the need to employ the right sort of hypothesis. If a cause is being sought, then testable hypotheses concerning the possible nature of that cause should form the basis of the search. Paradoxically, it is the very sophistication of many of the latest tools in neurochemistry and brain imaging that has led to the neglect of plausible hypotheses. The temptation is to use a novel technique as an end in itself, rather than to exploit it in testing a prediction. Computerised tomography (CT) is a case in point where, with a few exceptions (Owens *et al*, 1985), research has proceeded with little theoretical direction. The result is that we now know that a good proportion of schizophrenic patients show radiological appearances which differ, at least quantitatively, from normal, but we have little idea of why this should be so.

CT in schizophrenia

The significance of the CT scan research in schizophrenia should not, however, be underestimated. Variations between studies in selection and characteristics of patients and controls, and in the measurement techniques used, has meant that a consensus has been difficult to reach (Reveley, 1985; Smith & Iacono, 1986). Nonetheless, the weight of evidence points to lateral ventricular enlargement and cortical sulcal widening, in particular, being unexpectedly frequent in schizophrenia; these changes bear no relationship to the duration or type of treatment (Weinberger *et al*, 1979; Andreasen *et al*, 1982; Nasrallah *et al*, 1983; Kling *et al*, 1983; Williams *et al*, 1985; Owens *et al*, 1985) and are present at the start of the illness (Weinberger & Wyatt, 1982; Nyback *et al*, 1982; Schulz *et al*, 1983; Turner *et al*, 1986). The abnormalities speak, therefore, of cause rather than effect and can reasonably be assumed to antedate the illness. This being so, the CT research has yielded one of the few well-replicated findings which have a direct bearing on the aetiology of schizophrenia. Other such findings include the related observation that some cases of schizophrenia appear to be symptomatic of coarse brain disease, often environmentally mediated (Davison & Bagley, 1969), and the long-established evidence that relatives of schizophrenic patients have an increased risk of developing the illness themselves (Gottesman & Shields, 1982; Murray *et al*, 1986).

It is upon these few substantiated facts that testable hypotheses about underlying mechanisms should be based. As several mechanisms may be important, a fruitful starting point in their elucidation is to examine how these different findings interact. For example, is a family history of the disorder more or less commonly found in those schizophrenias secondary to coarse brain disease? Do patients with radiological abnormalities report past cerebral insults more or less frequently than those with normal scans? Consideration of the available evidence has led us to propose a strategy for future research (Murray *et al*, 1985) by which groups are identified in whom one or other of these aetiological indicators appears to predominate. We believe this to be a useful strategy, but it has found critics (DeLisi *et al*, 1985; Farmer *et al*, 1985; Eaves *et al*, 1986) and it is therefore timely to review not only the rationale behind it, but also its potentials and shortcomings in schizophrenia research.

Subdividing schizophrenia

Schizophrenia presents in a variety of different ways and leads to a variety of outcomes. This variation has resulted in many attempts to identify subgroups showing greater homogeneity. Traditionally, these attempts have been made using phenomenological criteria as in the classical Kraepelinian subtypes (WHO, 1978) and, more recently, the paranoid and non-paranoid types of Tsuang *et al* (1974), or those types with and without nuclear symptoms (Harrow & Quinlan, 1977), altered mood (Pope & Lipinski, 1978), or negative symptoms (Andreasen & Olsen, 1982). In addition, validity has been claimed for a number of subdivisions based on non-phenomenological variables, such as age of onset (Kay & Roth, 1961), premorbid personality (Strauss *et al*, 1977), neurological signs (Quitkin *et al* 1976), response to drug treatment (reviewed by Murray & Murphy, 1978) and prognosis (Langfeldt, 1956). Experience of syndromes in general medicine (e.g. cor pulmonale or epilepsy) suggests that subdivisions based solely on symptomatic or demographic data may have face validity but have little directly to say about the origins of the syndrome; in contrast, a single disease may present in a bewildering variety of symptomatological patterns (e.g. syphilis or diabetes).

The thrust of our argument is that the most rational basis for a valid subdivision of schizophrenia, and the one which is most likely to generate homogeneous groups for the purposes of fruitful research, is that based upon likely aetiological indicators rather than phenomenology. The main candidates are therefore indicators of genetic transmission and indicators of cerebral pathology.

Experimental subdivisions of schizophrenia according to the presence or absence of a family history have been used before (Shur, 1982; Baron *et al*, 1982; Kendler & Hays, 1982), as have subdivisions based on the presence or absence of structural pathology. Of the latter, Kleist's (1960) speculative classification remains the most complex, but Crow (1980) has suggested an aetiological classification into types 1 and 2 which has been influential despite remaining essentially untested. Several studies have examined the clinical correlates of lateral ventricular enlargement on the CT scan. In terms of illness features, only impaired neuropsychological test performance (Golden *et al*, 1980; Donnelly *et al*, 1980; Adams *et al*, 1984; Owens *et al*, 1985) and, perhaps, negative symptoms (Andreasen *et al*, 1982; Williams *et al*, 1985; Owens *et al*, 1985; Pearlson *et al*, 1985) stand as well-replicated associations with increased ventricular/brain ratio (VBR) in schizophrenia.

It is our interpretation of the evidence regarding the relationship between the presence of cerebral abnormality and the presence of a manifest family history in schizophrenia which lies behind our proposal. We contend that structural cerebral abnormality is statistically more common in those schizophrenic patients without an obvious family history. The evidence for an inverse relationship between these two factors is disparate but, we believe, strong.

Family history and cerebral abnormality

Where schizophrenia arises in the context of pre-existing cerebral disorder, a family history of schizophrenia is rare. This has been shown for the cases which follow cerebral trauma (Achte *et al*, 1969) and epilepsy (Slater *et al*, 1963). In their definitive review, Davison & Bagley (1969) concluded that schizophrenias with evidence of concurrent cerebral disease "usually occur in patients without genetic loading for schizophrenia". Kendler & Hays (1982) demonstrated that schizophrenic patients with a family history of schizophrenia were significantly

TABLE I

Published studies comparing CT findings in schizophrenia with and without family history (FH)

Reference	*Number in schizophrenic group*	*Positive FH*		*Control group*	*Measure*	*Reported significance of difference FH+ v. FH−*
		Definition	*Number*			
Campbell *et al*, 1979	35	Major functional psychosis in first-degree relative	18	None	Radiologist's rating	NS
Nasrallah *et al*, 1983	55	Schizophrenia in first-degree relative	8	Head-injury patients with 'normal scans'	VBR	$P<0.05$ larger in FH+
Reveley *et al*, 1984	21[1]	Major psychiatric illness in first/second degree relatives	7	Healthy volunteer twins	Total ventricular volume	$P=0.016$ larger in FH−
Oxenstierna *et al*, 1984	20	Schizophrenia in first/second-degree relatives	8	None	Radiologist's rating of cerebral atrophy	$P=0.05$ larger in FH−
Owens *et al*, 1985	112	'Family history' of schizophrenia	–[2]	Neurotic patients	VBR	$P<0.002$ for curvilinear relationship
Pearlson *et al*, 1985	19	Unspecified	6	Healthy volunteers	VBR	NS
Reveley & Chitkara, 1985	51	Major psychiatric illness in first/second-degree relatives	26	Healthy volunteers	VBR	Trend towards larger VBR in FH−
Farmer *et al*, 1985	39	Schizophrenia in first-degree relative	8	None	VBR	NS
Cazzullo *et al*, 1985	119	Schizophrenia in first-degree relative	15	Head-injury patients	VBR	$P<0.05$ larger in FH−
Turner *et al*, 1986	30	Schizophrenia in first/second-degree relative	–[2]	Healthy volunteers	VBR	$P=0.02$ larger in FH−

1. Twins.
2. Not stated.

less likely to show an abnormal EEG than patients without a family history. Case reports of unsuspected discrete abnormalities on CT scan (Reveley & Reveley, 1983; Lewis & Mezey, 1985) suggest that a lack of familial predisposition characterises such patients.

Ten studies have now examined systematically family history in relation to CT scan abnormalities, particularly ventricular enlargement, in schizophrenia (Table I). Three studies (Campbell *et al*, 1979; Pearlson *et al*, 1985; Farmer *et al*, 1985) with small sample sizes found no relationship while the study of Nasrallah *et al* (1983) stands alone in reporting a *higher* rate of schizophrenia in the relatives of patients with large, compared with those with normal, ventricles. Five studies have found an inverse correlation between ventricular size and positive family history. In addition, Owens *et al* (1985) found an intriguing curvilinear relationship between "definite family history of schizophrenia" and VBR in 112 patients. The authors concluded that "a positive family history of schizophrenia is associated with normal ventricular size and that in those patients at extremes of ventricular size a family history is less common". Perhaps extremely small ventricles should also be regarded as pathological, as has been the practice in some post-mortem, pneumoencephalographic and CT studies in schizophrenia (Wildi *et al*, 1967; Gross *et al*, 1982).

The weight of evidence, therefore, suggests that in unselected populations of schizophrenic patients an *inverse* relationship exists between the presence of cerebral pathology inferred from clinical, radiological or post-mortem findings, and the presence of a manifest family history. This cerebral pathology is seen as the consequence of environmentally mediated processes. A causal continuum exists between these environmental factors at one pole and genetic factors (which manifest familial expression) at the other. This reciprocal relationship resembles the multifactorial model (Reich *et al*, 1972) of schizophrenia, with one important difference. The multifactorial model conceives of a liability to the disorder which is normally distributed throughout the population and is the sum of numerous genetic and environmental factors each of individually small, but additive effects. We would contend that the radiological data, for instance, imply that the environmental contribution often comprises single, relatively large, events. The possible nature of such events has been reviewed (Murray *et al*, 1985).

This is the rationale behind our proposal. The classification of cases into those *with* and those *without* a manifest family history will yield subgroups which, although not totally discrete, will be of substantially greater aetiological homogeneity. This will increase the chance of future research successfully identifying those genetic and environmental factors which are important in the cause of schizophrenia. Subdivision into familial and sporadic subtypes has been used fruitfully in other disorders whose epidemiology is not dissimilar from that of schizophrenia, such as epilepsy (O'Donohue, 1985).

We must emphasise that the familial/sporadic distinction is intended to be a pragmatic first step and is not, in itself, explanatory. Its value is heuristic, allowing the identification of groups of patients in which hypotheses can more readily be tested. Failure to appreciate that the strategy offers a means to test models, rather than being a model in itself, is part of the reason it has drawn criticism. Nevertheless, these, and more serious, criticisms should be examined.

Criticisms of the strategy

The first criticism is a theoretical one: "this proposal assumes, without sufficient evidence, that a significant proportion of schizophrenic patients have a non-familial aetiology" (DeLisi *et al*, 1985). In other words, it is wrong to suppose that non-genetic factors play a part in more than an insignificant fraction of schizophrenics. This *ex cathedra* statement is itself made with no evidence to support it. The plain fact is that all purely genetic models have been found lacking and none is sufficient to explain the way that schizophrenia is distributed within and between families. Furthermore, the genetic contribution to schizophrenia may be one of the few established aetiological facts, but its significance should not be exaggerated. It is of a similar order to that in medical conditions such as diabetes or hypertension (Kendler, 1983) and no one would suggest abandoning the search for non-familial causes in those conditions.

DeLisi *et al* (1986) have recently published in full the findings of the study which they previously quoted (DeLisi *et al*, 1985) in objection to the familial-sporadic strategy. We disagreed with their assertions then (Reveley *et al*, 1985) and inspection of their data substantiates our scepticism. Measures of lateral ventricular size were made on CT scan in 12 schizophrenic probands known to have at least one schizophrenic sibling. All the available siblings, of whom 12 had schizophrenia and 10 did not, were then scanned. Ventricular size was significantly greater in the schizophrenic patients than in their well siblings, thus replicating the results of Weinberger *et al* (1981) and Reveley *et al* (1982). The authors concluded that "increased ventricular size could represent an inherited vulnerability toward schizophrenia". This conclusion draws attention away from the truly interesting finding in their sample: a highly significant association, regardless of diagnosis, between ventricular enlargement and previous obstetric and childhood environment insults. In fact, all eight of the schizophrenic patients whose ventricles were larger than one standard deviation of a control mean had a history of either childhood head injury, birth complications, or intracranial infection.

Surely, then, to suggest from these findings that ventricular enlargement in schizophrenia represents an inherited vulnerability is mistaken. The conclusion can only be that pathological ventricular enlargement is a result of environmental rather than genetic influences. We do not dispute that environmental factors may also be of aetiological importance in some patients with a genetic predisposition. To do so would be misguided. Our point is that environmental factors assume greater importance in patients *without* a family history and that, if a search for these factors is to bear fruit, it should be directed primarily at such patients. The study of Schulsinger *et al* (1984) also confined itself to patients with a family history of schizophrenia. Their conclusion, in line with ours, was that environmental insults, in particular birth injury, predicted both large ventricles and schizophrenia and that these insults were of "direct aetiological significance".

Two further criticisms have been made that point to practical limitations of the strategy. The first is that some patients who actually have a genetic predisposition may be falsely classified as sporadic. The likelihood of this is highest if the patient's family has few relatives at risk, either due to small numbers or young age. Recently, Eaves *et al* (1986) made an elegant statistical exploration of the usefulness of the familial-sporadic distinction in aetiological research. They used a power analysis based on a series of more or less likely assumptions about the proportions of liability attributable to heritable and environmental factors. Their results suggested that, unless twins were used, large sample sizes would be needed before the familial-sporadic strategy could be expected to detect aetiological heterogeneity. However, as the authors went on to note, the fact remains that several studies of more modest size exist which have successfully demonstrated indicators of heterogeneity using the familial-sporadic approach. One explanation for the empirical success of the strategy despite the pessimistic projections of Eaves *et al* is that the aetiological model underlying their power analyses was mistaken. They assumed the polygenic-multifactorial model, in which environmental (as well as genetic) factors are of individually small, albeit additive, effect. As stated earlier we are dubious about this assumption, believing rather that some observations are more parsimoniously explained by the existence of environmental risk factors of relatively *large* individual effect. If this were so, it would serve to increase substantially the power of the familial-sporadic strategy and perhaps explain why it has already proven fruitful. The power could be increased further by discriminating the two groups subsumed under the sporadic heading: one group in whom positive radiological or historical evidence exists for past cerebral insult and a remaining group identified by exclusion, with manifest evidence neither of cerebral insult nor of familial risk. Nevertheless, possible artefacts arising from small family sizes cannot be ignored and may account for the failure of some studies to find an inverse relationship between family history and CT abnormalities (Campbell *et al*, 1979).

The second practical criticism is that there is no agreed definition for what constitutes a "positive family history" and that any definition is likely to be arbitrary. To some extent, this is so. Yet theoretical and practical considerations can guide the choice of such a definition, in terms of which disorders to include, in which relatives, and how the ascertainment should be made. From a theoretical standpoint, the disorders which are important in relatives are those for which good evidence exists of a genetic relationship with schizophrenia. Obviously, this will include schizophrenia itself, as well as schizoaffective disorders (Tsuang, 1979; Kendler *et al*, 1985). Furthermore, non-schizophrenic psychoses may occur more frequently than expected in the families of schizophrenic probands (Odegaard, 1972; Scharfetter & Nusperli, 1980). Evidence also exists that certain varieties of personality disorder, specifically schizotypal, are genetically related to schizophrenia (Kety, 1980; Baron *et al*, 1985), but these present problems of practical ascertainment. There is no doubt that direct family interview is the best method of detecting illness in relatives (Thompson, 1982), but family history data properly obtained from a related informant can be of adequate reliability (Andreasen *et al*, 1986). The reliability of such information is a function both of the severity of the disorder and the closeness of the relative, although schizotypal and related spectrum disorders have been assessed using the family history method (Baron *et al*, 1985). Empirical precedent varies as to whether to restrict consideration to illness in parents and siblings (Stephens *et al*, 1975), or all first-degree relatives (Scharfetter & Nusperli, 1980), or to include second-degree relatives (Owens *et al*, 1985; Reveley *et al*, 1984).

This lack of agreement makes it all the more important that researchers intending to use the variable of positive family history make explicit their defining criteria. It is hoped that forthcoming investigations of the impact that different definitions of this variable might have on the phenomenology, course and biological attributes of schizophrenia will enable more sensible selection from the differing definitions used to date (e.g. Table I). In the meantime, the dogmatic recommendation of any one definition cannot be justified. It is likely that the most restrictive definitions of positive family history would identify probands with the highest degrees of genetic liability. In this way, a 'narrow' definition might be the occurrence of schizophrenia requiring hospital admission in a first degree relative–parent, sibling, or child. A 'broad' definition, on the other hand, would be that including any psychiatric disorder, excluding uncomplicated drug or alcohol abuse, requiring the admission of a first or second degree relative to a psychiatric unit. Completed suicide would also be included. Broad definitions would reduce the chance of false negatives but inflate the danger of false positives and perhaps might best be used to identify, by exclusion, negative family history or 'sporadic' cases (Kendler & Hays, 1982). In large samples, narrow and broad definitions may be used in tandem to discriminate subgroups with different degrees of underlying liability in accord with multiple threshold models. In all cases minimum guidelines for ascertainment of family history should be followed: the interview of at least one related informant, with systematic recording of demographic information to include number and current age (or age at death) of relatives. This will allow subsequent age-correction and between-group matching for the age-corrected number of relatives at risk, if desired. Besides symptoms, diagnostic information for relatives should include age of onset, and method and agency of treatment, which may be supplemented by data from casenotes and, where appropriate, death certificates. Operational criteria for diagnosis in relatives are available for the Research Diagnostic Criteria (Andreasen *et al*, 1977) and have also been developed for spectrum disorders (Kendler *et al*, 1984).

Conclusions

Coherent threads can now be discerned in the unhappy tangle of research into the likely causes of schizophrenia. That genetic factors are important can no longer be disputed, while brain-imaging techniques have revealed cerebral abnormalities in a substantial proportion of patients. Our interpretation of the evidence available is that these abnormalities occur preferentially in patients without an obvious family history. Antecedent environmental insults such as encephalitides, head injury, and perinatal complications offer plausible mechanisms by which both cerebral pathology and the later illness itself arise in some patients. We have reviewed these mechanisms in greater detail elsewhere (Murray *et al*, 1985). The availability of exciting new techniques in brain imaging and molecular genetics is no guarantee that they will be used with appropriate rigour in schizophrenia research. In order better to test hypotheses about genetic factors on the one hand and environmental factors in the other we have proposed a simple classification of patients based on the presence or absence of a family history of similar disorder. Within the familial group, those individuals with schizophrenic first-degree relatives are most likely to repay research aimed at revealing genetic markers. Conversely, within the sporadic group, patients with radiological abnormalities offer the best chance of clarifying the nature and role of environmental factors. The puzzling epiphenomena found in an uncertain proportion of schizophrenic patients, such as developmental deviance, psychometric deficits, and neuromotor abnormalities, including those of lateralisation, may also yield clues in this group.

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Expressed Emotion and Relapse in First Episodes of Schizophrenia
A Rejoinder to Macmillan *et al* (1986)

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Re-analyses of data presented by Macmillan *et al* (1986*b*) challenge their conclusions that high expressed emotion is not prognostically significant for the course of schizophrenia or unrelated to the level of behavioural disturbance prior to admission. Several equally plausible models of causal relationships between EE and duration of untreated illness are presented. We conclude that our re-analyses of the Macmillan data do not warrant premature closure regarding the significance of the EE variable.

> Comparison of relapse rates in the two Critical Comment groups revealed higher relapse/readmission rates in the group with a score of six or more ($P = 0.036$) . . . (Macmillan *et al*, 1986*b*).
>
> Neither of the two components of the CFI emerged from this study as important determinants of relapse/readmission significant at the 5% level . . . (Macmillan *et al*, 1986*b*).

Macmillan *et al* (1986*b*) studied the association between expressed emotion (EE) and outcome in a subsample of their first-break schizophrenics. The well established finding that high EE is a predictor of poor outcome has now been replicated with several major psychiatric disorders in several sociocultural settings. Studies of schizophrenics in England (Brown *et al*, 1972; Vaughn & Leff, 1976) and in the United States (Vaughn *et al*, 1984; Nuechterlein *et al*, 1986; Moline *et al*, 1986; Jenkins *et al*, 1986), and of depressed patients (Vaughn & Leff, 1976; Hooley *et al*, 1986), bipolar manics (Miklowitz *et al*, 1986) and obese patients (Leff & Vaughn, 1985) have all reported that patients living in homes with a high-EE relative are at increased risk of relapsing.

The above quotations present the reader of Macmillan *et al*'s paper with a difficult contradiction: despite finding a significant association between EE and outcome, the authors interpret their data as a failure to replicate this. Unfortunately, EE has become a focus of controversy in psychiatry: parents of schizophrenic patients have resisted what appears to be a new attempt to blame them for the illness, through the concept of stress as a cause of breakdown, while professionals who view the basis of schizophrenic illness as biological have argued against the allocation of resources to the study of psycho-social factors. Yet, this psychosocial variable has repeatedly demonstrated its utility as a predictor.

In this context, it is critical that responsible scientists evaluate, interpret, and present their data carefully and dispassionately. The paper by Macmillan *et al* is an important one, presenting new findings, coming from a respected research group, and forming part of a large and carefully conducted neuroleptic trial with a relatively unusual sample of first-break schizophrenic patients. It is likely to have a great impact on both professional and non-professional readers.

We believe that the fundamental assertion of these authors – that their results represent a failure to replicate the wellknown finding that EE is prognostically important for schizophrenia – is simply incorrect, and unwarranted on the basis of their own data. Furthermore, we are struck by a number of features of the analysis, presentation, and design of their data that reduce the credibility of their report.

Brief review of the Northwick Park design

The Northwick Park study, reported in four concurrent articles (Johnstone *et al*, 1986; Crow *et al*, 1986; Macmillan *et al*, 1986*a*,*b*), was primarily designed as a trial of neuroleptic medication versus placebo in the management of early schizophrenia. The larger study followed 120 patients who entered a randomised trial for two years, as well as a sample of 116 eligible patients who did not enter the trial and were followed in what is referred to as standard and presumably uncontrolled treatment.

The article in question reports on EE as a prognostic factor for a subsample of the patients – those residing with their families for three months prior to entry to the study, who were competent in English; patients in this study were drawn from both the randomised trial and from the standard treatment. It is important that the patients receiving standard treatment were a different population, with poorer prognostic features, as documented by Macmillan *et al* (1986*a*);

they had a longer period between the onset of illness and admission, longer stay in hospital, and less likelihood of employment after discharge. Even more important, the criteria for relapse were different: in the active and placebo treatment groups, the "main outcome variable . . . was relapse ascertained by the responsible clinician", whereas in the standard treatment group, relapse was defined only by re-admission – a much more limited criterion. Perhaps this is why outcomes were *better* in the standard treatment group, despite their having poorer prognostic indicators (Macmillan *et al*, 1986*a*).

In the major report on neuroleptic outcomes (Crow *et al*, 1986), data were presented only for those patients who entered the randomised trial. This is appropriate, since the patients in standard treatment were different from those in the randomised trial, and different outcome criteria had to be used. Macmillan *et al* (1986*a*), discussing differences in outcome between patients who entered the randomised trial and those in standard treatment, analysed all three groups separately. Even so, we regard the inclusion of standard and randomised patients in one analysis as questionable because the outcome criteria were meaningfully different.

Most importantly, in the EE study under discussion here, patients on active neuroleptic medication from the randomised trial and those in standard treatment were pooled together as one group. This is simply inappropriate, and it obscured differences between these groups that significantly affected interpretation of the results.

Why did Macmillan *et al* interpret their findings as a non-replication?

The primary method of analysis employed by Macmillan *et al* was life table or survival analysis with covariates; they acknowledge EE as a predictor of relapse in their survival analyses, and state that, "Comparison of relapse rates in the two CC groups revealed higher relapse/readmission rates in the group with a score of six or more . . .". They then dismiss this finding because the EE–relapse relationship was non-significant at the $P = 0.05$ level, when duration of illness prior to hospital admission and treatment group were included in a multivariate survival analysis. In their sample, EE turned out to be confounded (correlated) with treatment assignment (more patients from high-EE homes were assigned to placebo – a group with poorer outcomes), and with duration of untreated illness (high EE was associated with longer untreated duration – itself a poor prognostic indicator).

The confounding of EE and treatment

With regard to the confounding of EE and treatment assignment, Macmillan *et al*'s Table IV provided the data necessary for one re-analysis; we could not do a survival analysis, but were able to estimate the final two-year relapse percentages in each treatment group (Table I). The numbers in this table represent the percentage of patients who relapsed, excluding those lost to follow-up, while the numbers in parentheses represent the possible range of relapse rates when the cases lost to follow-up are included, with the lower figure representing the relapse rate if all the lost cases did not relapse, and the higher figure representing the relapse rate if all lost cases relapsed.

TABLE I
Relapse rates by EE and treatment group

	Treatment group		
	Active	*Standard*	*Placebo*
Low EE	50%	10% (8–25%)	60%
High EE	86% (67–89%)	46% (43–50%)	78%

There were only five patients in this sample who were lost to follow-up; these may have had any of 18 possible patterns of outcome. For example, the two in the low-EE standard-treatment group could have had three patterns of outcome (both non-relapsed, both relapsed, or one relapsed, one non-relapsed). The same is true for the two lost cases in the high-EE active-treatment group. Finally, the one lost case in the high-EE standard-treatment group could have had two outcomes (relapsed or not). Thus, there are $3 \times 3 \times 2 = 18$ possible ways in which these lost cases might have fared.

We analysed the data for all 18 possible outcomes, using a log–linear model with EE, treatment group, and their interaction. Fourteen of these yielded a significant EE effect at the two-tailed 0.05 level, and 17 were significant at the one-tailed 0.05 level (a one-tailed test is justified in view of the large body of prior research). The only analysis that was non-significant was based on the unlikely assumption that all three high-EE patients lost to follow-up had good outcomes and that both low-EE patients lost to follow-up relapsed. That analysis still yielded a one-tailed P-value for EE of 0.06. We conclude that confounding of EE with treatment did *not* account for the association between EE and outcome which Macmillan *et al* found.

The confounding of EE and duration of untreated illness

When a measure of 'duration of untreated illness' was included in the multivariate survival model, the significance of EE as a predictor of relapse/readmission was reported to be attenuated, and no longer significant at the 5% level. In fact, the significance of EE in a multivariate model which included treatment and duration was 0.07 (one-tailed test). It is unfortunately commonplace to interpret the 0.05 level as a firm dividing line between real and chance relationships.

In any event, the fact that the partial association of EE and outcome was non-significant – even if it were ultimately proven to be zero in the population when viewed in the context of these other variables – is in no way inconsistent with the view that EE plays a causal role in outcome. Figures 1–3 describe three causal models, which we have characterised as 'spurious correlation,' 'indirect causation', and 'mediating variable'. Each of these models includes EE, duration, and outcome, and each is consistent with the data of Macmillan *et al.*

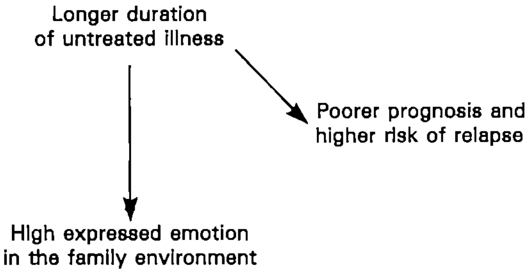

FIG. 1 Model I: spurious correlation.

In Fig. 1, the relationship between EE and outcome is spurious: it results from the fact that EE and outcome both have a common cause, and with this model, controlling for duration eliminates the relationship between EE and outcome. This appears to be the model assumed by Macmillan *et al*; it suggests that high EE is a reaction to a longer period of illness, and that the relationship between EE and relapse is simply a statistical artifact, explained by this confounding. This interpretation is certainly plausible and intriguing, and deserves further investigation.

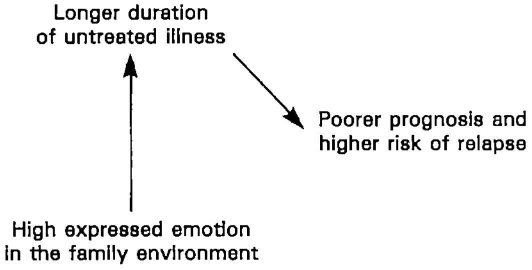

FIG. 2 Model II: indirect causation.

In Fig. 2, the causal relationship is simply reversed. This model acknowledges the causal importance of untreated duration for outcome, but assumes that high EE in the home itself is a factor that causes duration to be longer. How could this be? We offer a few hypotheses, noting that the measure of duration used by Macmillan *et al* was largely derived from reports by the relatives themselves – the same source used for the ratings of EE.

One plausible suggestion is that high-EE relatives are less likely to attribute to illness any deviance which occurs in their children prior to their first clear psychotic break, resulting in longer untreated durations and poorer outcomes. Alternatively, since the length of illness prior to admission was derived at least in part from relatives' reports, it is possible that high-EE relatives might simply report longer periods of illness than low-EE relatives, and that these reports would not be consistent with those of other informants. Perhaps high-EE relatives are more accurately sensitive to early signs of schizophrenia, or more easily bothered by mild signs of deviance, or perhaps these reports of earlier onset prior to treatment simply reflect another criticism of the patient – a characteristic that defines the EE status of the relative.

Any of these scenarios might underlie the causal model of Fig. 2. They are all consistent with a zero partial association between EE and outcome, when duration is controlled, but none imply that EE is either prognostically or causally insignificant.

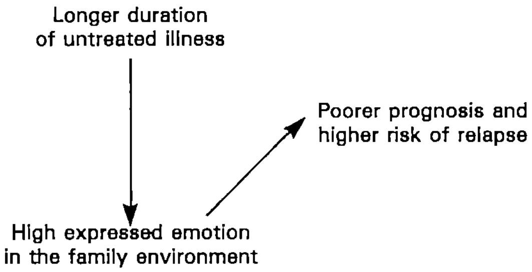

FIG. 3. Model III: mediating variable.

Figure 3 presents another model of indirect causation – one which we consider particularly appealing. This model views EE as an intermediate causal factor – a reaction to the stress of untreated illness in a relative, and a stressor that itself has malignant prognostic implications. Here too, controlling the duration variables would lead to a non-significant association between EE and outcome.

Macmillan *et al*'s interpretation of their data is very likely to be taken as discounting EE as a causal variable, particularly since their discussion firmly discounts EE as a determinant of relapse:

> Although the findings of the present study are not decisive, we regard them as casting considerable doubt on recent speculations that important predictors of outcome are present in the family environment. If such factors exist, it is not clear why they should not have been more apparent in the present relatively homogeneous and well-documented population of patients with early schizophrenia.

It should be clear from Figs 1–3 that causal interpretations of partial associations derive from causal models, not from the statistics themselves. Macmillan *et al* acknowledge that there is no basis for clear specification of a model at this time:

> There is a significant association between longer duration of illness prior to admission and higher CC, the causal sequence of which is unclear.

Psycho-social and pharmacological effects

> Neither of the two components of the CFI emerged as important determinants of relapse/readmission . . . while duration of illness and neuroleptic treatment after discharge are related to relapse (Crow *et al*, 1986). (Macmillan *et al*, 1986*b*).

This statement may appeal to biologically orientated readers, but it is misleading. Their study of EE and outcome was based on a subsample of cases from the larger neuroleptic trial reported by Crow *et al* (1986). When Macmillan *et al* state that the CFI was not a determinant of relapse, they are (mis)interpreting the results of a multivariate survival model, based on their reduced EE subsample. In stating that duration and neuroleptics were "related to relapse", they allude to data from a different sample of cases and to an analysis that did not include EE.

They state that a "crude examination of the percentage relapse/readmission rates in the present study reveals results apparently discrepant with earlier work", and that "we regard them [the findings] as casting considerable doubt on recent speculations that important predictors of outcome are present in the family environment". We disagree.

In Table I the parenthesised figures show both the lowest and highest possible relapse rates within each cell: in every case, the lowest relapse estimate in the high-EE groups is higher than the highest relapse estimate in the low-EE groups. The most *conservative* estimate, taking the lowest possible figure from the high-EE groups and the highest possible figure from the low-EE groups, yields differences of 17%, 18%, and 18% respectively across the three treatment conditions; estimates which exclude the five cases lost to follow-up are considerably higher.

In fact, in these authors' subsample, EE is a *better* predictor of outcome than active neuroleptic treatment within the clinical trial. In our Table I, the difference in relapse rates (excluding dropouts) attributable to EE is roughly 30%, regardless of treatment group; on the other hand, relapse rates for placebo and active treatments in this subsample of cases were not meaningfully different. If there is a medication effect in this table, it is entirely due to the low relapse rates in standard treatment, but this is most likely an artifact, because the criterion for relapse in that group was limited to readmission.

Credibility gap

There are at least two significant errors in Macmillan *et al*'s Table IV and Fig. 1. The first is the reversal of the relapse/no-relapse rates for the low-EE active/standard-treatment conditions in Fig. 1. The table shows the relapse rate as 63% and the no-relapse rate as 31%, but in fact, the correct rates are 31% relapsed and 63% not relapsed, as indicated by the numbers in Table IV. This may be a simple typographical mistake, but it is highly misleading, and it happens to be consistent with the authors' conclusions.

More subtle but equally important is the fact that the numbers in Fig. 1 and Table IV are not consistent, and cannot be correct. In this case, we do not believe the error is likely to be typographical, since all of the marginal statistics in Table IV and Fig. 1 agree.

Table IV shows that six high-EE active-treatment patients relapsed. Since none of the active-treatment patients with high social contact (SC) relapsed (Table IV), all of these six must have been in the high-EE low-SC active-treatment group. The total number of high-EE low-SC patients who relapsed in combined active and standard treatment is reported as 11 in Fig. 1. Since six of these were in the active-treatment group, the remaining five relapsers must have been in the high-EE low-SC standard-treatment group. However, Table IV shows that the total number of low-SC standard-treatment group relapses is only four. Since all the marginals agree, this error must affect more than one cell, to an unknown degree.

Studies of treatment of EE

Studies of the value of treatment of EE in families have obvious bearing on the causal interpretation of the EE–relapse association. If EE is simply a reaction to the behaviour of patients, with no causal importance, it is not clear why reducing it should improve prognosis unless the treatment is also reducing forms of behaviour that are causally relevant. In their discussion, the authors state that "we do not wish to suggest that support and counselling [for families] are not worthwhile . . . but their role in determining outcome has yet to be demonstrated". They thereby ignore a substantial number of controlled clinical trials that have evaluated the efficacy of family intervention programmes which, in conjunction with medication, have been found to significantly reduce relapse rates compared with control conditions (Goldstein, *et al*, 1978; Leff *et al*, 1982; Falloon *et al*, 1985; Hogarty *et al*, 1986).

Is EE relevant to enough schizophrenics?

The authors state that study of EE is not likely to be clinically productive, because relatively few patients (about 30%) return to live with families. Thus, the typical small total *n* in most clinical studies can reach vanishing point in some cells, if one attempts to cross-tabulate EE with other relevant variables, such as amount of contact with high-EE relatives.

Adequacy of sample size to analyse complex multivariate models is a pandemic problem for clinical research: it is certainly not unique to EE, and is no reason to dismiss an important correlate of outcome of schizophrenia. In any event, it is not at all clear that the EE effect is limited to a few unpopulated cells of a complex multivariate model. In fact, the current study found a significant main effect for EE on relapse.

It should be noted that Macmillan *et al* restricted their sample in two significant ways: firstly, they included only patients who had lived with families for three months prior to admission; secondly, they excluded all families who were not fluent in English. Recent research on EE has used a less stringent criterion of frequency of contact – one month out of the three preceding admission (Vaughn *et al*, 1984; Nuechterlein *et al*, 1986). The exclusion of non-English-speaking families may have been logistically convenient, but hardly bears on the relevance of the *concept* of EE for schizophrenic relapse. In fact, there is a recent replication of the basic EE-relapse association with Spanish-speaking families (Jenkins *et al*, 1986). Macmillan *et al*'s experience in England may be that relatively few patients return to family environments, but we must caution against ethnocentric interpretations. In other cultural settings (Jenkins *et al*, 1986), return to a familial setting may in fact be the norm.

Furthermore, it is important that EE may also be a significant prognostic factor outside the family environment. A basic notion underlying EE research is that interpersonal stress may be an important determinant of relapse: although EE has not yet been the focus of detailed empirical study outside the family setting, this would seem to be a reasonable and important extension of existing research. Study of important prognostic factors may ultimately lead to better understanding of the process of relapse, even if initial study must be confined to a select and hard-to-find group of patients.

Methodological details on EE assessment

Macmillan *et al* interviewed only the relative with whom the patient had the most contact, and this is not typical research procedure in EE studies. Usually, relatives with whom the patient has significant contact (Nuechterlein *et al*, 1986; Jenkins *et al*, 1986) are interviewed. Obviously, the more relatives interviewed, the more likely it is that a family will be classified as high in EE (Kottgen *et al*, 1984). Thus, Macmillan *et al* are likely to have under-identified high-EE homes to some degree.

EE and disturbed behaviour ratings (DBR)

Macmillan *et al* state that "the data are consistent with a model of partial independence . . . with DBR unassociated with either CC [critical comments] or SC [social contact]." However, we reanalysed the data in their Table III in several ways. Firstly, we dichotomised DBR at the median (0–2 versus 3 or more), and analysed the matrix with a log–linear procedure (BMDP4F): the analysis yielded a significant effect for EE ($\chi^2=6.07$, $P<0.014$), and a borderline significant interaction between EE and SC ($\chi^2=3.20$, $P<0.074$). Because the interaction term was of borderline significance, the data were reanalysed separately for groups with high and low social contact: these analyses yielded significant results in the high-SC group ($\chi^2=4.25$, $P<0.007$), but there was no significant EE effect in the low-SC group. Dichotomising at 0–1 versus 2 or more also yielded an EE × SC interaction of borderline significance ($\chi^2=3.1$, $P<0.08$), and a significant χ^2 for EE in the high-SC group ($\chi^2=4.02$, $P<0.045$).

The data were also reanalysed using a 2×2 factorial ANOVA. Since the original Table III pooled DBR scores above 3, cases above 3 were assigned a score of 3. Our Table II shows the means: the ANOVA yielded a significant main effect for EE ($F=6.88$; d.f. = 1, 77; $P<0.011$), and an EE × SC interaction of borderline significance ($F=3.68$; d.f. = 1, 77; $P<0.059$).

TABLE II
Disturbed Behaviour Rating (DBR) score by EE and social contact

	Social contact	
	High	*Low*
EE high	2.6	1.7
EE low	1.3	1.5

Furthermore, a Tukey (HSD) test revealed a statistically significant effect of EE for the high-SC group. In short, our analyses are entirely consistent, and demonstrate a significant association between EE and DBR for patients with high SC.

The strategic decision by Macmillan *et al* to dichotomise with a cut-off at 1 is thus quite questionable, and it is difficult to understand the authors' statement that "in the first analysis (DBR dichotomised) the main effect of this variable could also be eliminated . . .". How could this be so with marginals of 12 (DBR = 0) versus 69 (DBR = 1 or more)? In fact, it is not so. When we used that cut-off point, we obtained a χ^2 of 44.33 ($P<0.0001$) for the DBR main effect.

The importance of this reanalysis is that the relationship found here between DBR and EE is consistent with the novel finding that duration of illness prior to admission was related to EE. The relationship between DBR and EE is striking, since the authors took care to "avoid contamination with the factor of duration of symptoms before admission" by devising a "revised form of rating . . . in which the temporal element was removed". This association may simply be a peculiarity of their sample, since prior research has uniformly failed to find a relationship between EE and level of behavioural disturbance.

On the other hand, the possibility remains that Macmillan *et al* have uncovered a new and potentially important set of relationships of EE and the attributes of patients (duration and disturbed behaviour prior to initial treatment). In fact, currently, we are actively pursuing some of the leads suggested by their report in another sample of first-break cases.

Closing comment

It is unfortunate that Macmillan *et al* chose to present their research on the prognostic importance of psychosocial factors in a manner that implies that such research in the area of schizophrenic outcome somehow conflicts with research on pharmacological treatment. In fact, there is growing interest in the integration of these two areas of study.

> If genuine determinants of poor outcome are present in the immediate families of patients with schizophrenia, they are somewhat elusive and when present, their effects are unlikely to be large. The search for such influences should not detract from the much better documented, although limited prophylactic effects of neuroleptic medication. (Macmillan *et al*, 1986*a*)

We are certainly not suggesting that further research on neuroleptics is not needed. What we are suggesting is that premature closure on *any* research area, particularly a variable with such strong research credentials as EE, would be unfortunate for the advance of knowledge of the determinants of outcome in schizophrenia.

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Expressed Emotion and Relapse in First Episodes of Schizophrenia

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In the preceding paper, Mintz *et al* have raised a number of criticisms and points relating to our study, to which we are pleased to respond.

Their rejoinder is headed by two quotations from our paper which are described as presenting the reader with a "difficult contradiction". The first quotation reports a statistically significant ($P = 0.036$) association between the level of critical comments and relapse, while the second states that this component of the Camberwell Family Interview was not a determinant of relapse at the 5% level. The first quotation refers to a direct (unadjusted) comparison of relapse rates in the groups with low and high levels of critical comments (CC), while the second refers to the same comparison adjusted for two prognostic factors, treatment and duration of illness. There is therefore no contradiction between the statements.

The single association between critical comments and relapse that reached 'generally accepted levels of statistical significance' was unadjusted for two prognostic factors. The prognostic importance of these factors had been indicated by our previous analysis (Crow *et al*, 1986). These factors were not identified by statistically significant tests carried out in the sample of patients rated for expressed emotion (see Altman, 1985). Obviously there could be other confounding effects produced by other variables which we have not assessed.

Adjustment of the CC comparison by treatment is not only justified but necessary since some patients received placebo and a difference in outcome between treatment groups *is expected*. Similarly, it is logical to adjust the CC comparison by a measure of severity of illness; such adjustment is a common feature of epidemiological studies of disease progression.

Amalgamation of the 'active' and 'standard' treatment groups is of course not necessary. When the data are analysed with patients stratified into three treatment groups (active, placebo and standard), results are essentially similar to those presented in our paper; comparison of relapse rates in the two CC groups adjusted for treatment and the period between onset and admission gives $\chi^2 = 2.16$, d.f. $= 1$, $P = 0.14$. If the comparison of the two CC groups is adjusted *only* for treatment the statistical significance of the unadjusted comparison also 'disappears' whether active and standard treatment are combined ($\chi^2=2.84$, d.f. = 1, $P=0.092$) or not ($\chi^2=3.34$, d.f. = 1, $P=0.068$). Therefore neither pooling the findings in our active and standard treatment groups nor adjustment for duration of illness is necessary for us to conclude that critical comments do not significantly predict outcome. The effect of high CC estimated from the Cox model including treatment *only* gives a hazard rate ratio of 1.88 (with 95% confidence limits at 0.97 and 3.64). Ignoring the confounding effect of treatment would be inconsistent with the results of our study, and inconsistent with a very large body of research upon the effect of neuroleptic prophylaxis in schizophrenia (Davis, 1975).

None of these comparisons yield a significant effect for CC, and it should be noted that the different definitions of relapse do not invalidate our analysis. In these survival models, stratification allows the assumption of a multiplicative effect for EE without any assumption about the form of the hazard for relapse. Indeed this hazard may be quite different in the separate strata. With only 40 relapses in 77 patients we do not have a sensitive test of the assumption of a constant multiplicative effect for EE.

EE and treatment

Mintz *et al* suggest that our Table IV provides sufficient data for an analysis of crude 2-year relapse rates. This is not correct. Patients entered in our study were followed from hospital discharge to relapse/readmission, loss to follow-up, or to 1 August 1982 (up to a maximum of 2 years). Thus patients entered after 1 August 1980 were followed up for less than 2 years; since recruitment did not terminate until February 1982, some were followed for considerably less. Further, the presentation by Mintz *et al* provides no estimate of the variation in relapse rates. Table I shows actuarial percentage relapse-free rates (and 95% confidence limits) at one year from discharge.

TABLE I

Actuarial percentages (and 95% confidence limits) relapse-free at one year from hospital discharge

		Treatment group	
	Active	*Standard*	*Placebo*
Low EE	63 (36, 89)	91 (73, 100)	38 (6, 69)
High EE	43 (6, 80)	63 (36, 89)	33 (11, 55)

We chose this interval because the number of patients at risk after one year in each subgroup was very small. Simply taking differences between the percentages in Table I of Mintz *et al* fails to take account of sampling variation; the 95% confidence limits in our Table I show that the variation is large.

Mintz *et al* have drawn attention to errors in our Fig. 1. It should be as shown here.

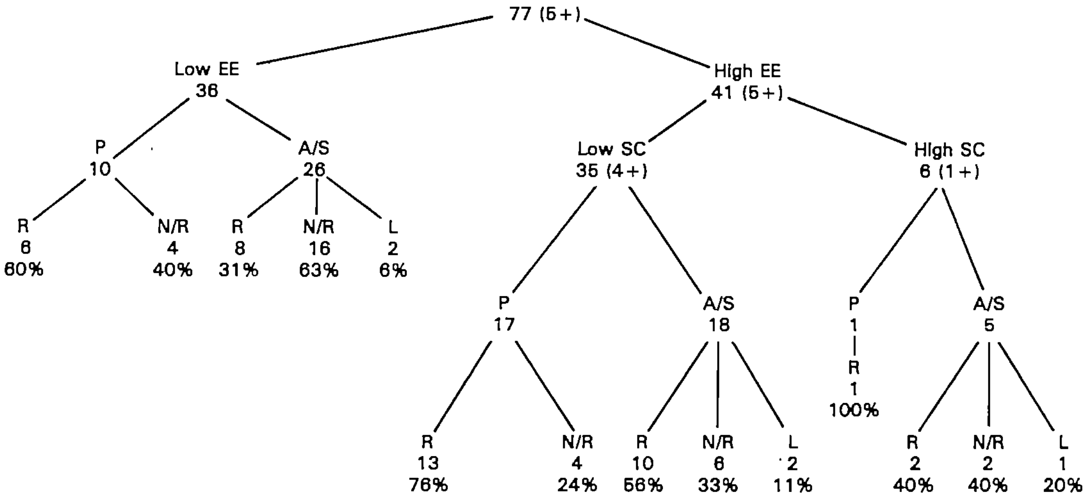

FIG. 1 Branching diagram of relapse rates in patients divided by expressed emotion and allocation of medication. EE = expressed emotion, SC = social contact, P = placebo medication, A/S = active or standard medication, R = relapsed, N/R = not relapsed at conclusion of study, L = lost to contact. Numbers represent individual cases; those in brackets refer to patients not discharged from hospital.

EE and duration of untreated illness

Mintz *et al* propose three models of interaction between level of critical comments, duration of onset and time of relapse. We make no comment, as the estimates of the effect of high relative to low critical comments on relapse rates are similar whether adjusted for treatment alone or for both treatment and duration of illness, being non-significant at the 5% level and having wide 95% confidence limits.

Studies of treatment of EE

There is a well recognised bias in favour of publishing positive results especially in clinical trials (Begg, 1985), and the majority of studies of expressed emotion have incorporated only small samples of patients.

The most recent, and by far the largest of the studies referred to (Hogarty *et al*, 1986) illustrates some of the limitations of current knowledge. In discussing their findings, Hogarty *et al* note that 'causal' inferences regarding EE and relapse need to be qualified:

> It is possible that the high EE status of households with a relapsed patient is due to the relapse event itself . . . it is as possible to argue that some families changed their EE status because the patient's condition improved as it is to suggest that lowered EE 'caused' a better patient outcome.

These authors continue:

> we wish to emphasise that these early prophylactic effects are more likely related to the *delay* of schizophrenic relapse than to its prevention. . . . Although a main effect of family treatment is being sustained, and much of the relapse is associated with drug non-compliance, we are clearly not preventing schizophrenic relapse. Recently Leff *et al.* (1985) reported the first two-year follow-up of a nine-month controlled family study and argued that only one (14%) of the seven family therapy patients relapsed when EE on 'face to face contact' had been reduced, compared with 78% relapse among drug treated controls. However, when one examines the outcome of their original sample of 12 experimental and 12 control patients, 75% of controls indeed relapsed (seven of these while receiving medication), but 50% of experimental patients also had a poor outcome: two relapsed while receiving drug, two relapsed while not receiving medication and two committed suicide.

Hogarty *et al* also note that "only Falloon *et al* described a second year outcome of family intervention significantly better than what one would exponentially predict for drug-maintained patients", and go on to discuss the apparently high rates of relapse in the drug-treated controls in these studies:

> nine-month relapse rates with maintenance chemotherapy alone exceed the 12-month 41% relapse rate of our own trial and the characteristic 12-month rate among published, drug-controlled maintenance studies. . . . As such, relapse rates among our own controls either reflects a prophylactic effect of our 'active' control treatment . . . or indicates that drug controls in the contrasting studies (i.e. those of Goldstein *et al.* 1978, Leff *et al.* 1985, Falloon *et al.* 1982 and Köttgen *et al.* 1984) might contain covert drug defaulters.

Perusal of Hogarty *et al*'s frank and critical discussion indicates that this literature is at an early stage. We doubt whether there is as yet a body of research which is sufficiently rigorous to convince a sceptical observer (or to justify the use of one-tailed significance tests).

Is EE relevant to enough schizophrenics?

Mintz *et al* have misinterpreted our remarks about the relevance of expressed emotion to schizophrenic patients. We reiterate that despite our efforts, this assessment could be performed in only one-third of our total sample, and the combination of high critical comments and high social contact was very uncommon (7 out of an original sample of 253 patients). We do not feel in a position to comment about the effects of expressed emotion outside the family environment. We do not consider our sample particularly ethnocentric except that the population was drawn from within and around North West Thames Region (a metropolitan area which contains a spectrum of social classes and some recent immigrant groups).

EE and Disturbed Behaviour Ratings

We were interested in the reanalysis of the relationships between EE and Disturbed Behaviour Rating (DBR), although Mintz *et al* have misunderstood our presentation. We did not dichotomise DBR below 1, but above 1, as stated clearly in our paper. The appropriate marginals are 39 (DBR = 1) and 42 (DBR = 2), and so the elimination of the main effect of DBR is fully justified. Dichotomising DBR as 2 or less versus 3 or more gives marginals of 58 and 23 respectively, and is not consistent with dichotomising at the median.

We have followed through the suggestion of Mintz *et al* to analyse the DBR scores by analysis of variance. Some care is necessary because the DBR scores are not distributed symmetrically. The means presented in their Table II are not sensible summary statistics for these data which have a pooled standard deviation of 1.0. Using the methods described by Snedecor & Cochran (1967) for the analysis of unbalanced data in a 2 × 2 factorial design, we have first repeated the analysis reported by Mintz *et al*, but with somewhat different results; in our analysis we find a non-significant main effect of CC ($F=3.80$; d.f. = 1, 77; $0.05<P<0.10$) and non-significant interaction ($F=3.68$; d.f. = 1, 77; $0.05<P<0.10$). Secondly, we have used actual DBR scores (without recoding scores of 4 and 5 to 3), but have transformed them by adding one and taking square roots, yielding stable subgroup variances and a symmetrical distribution of scores. Here there is a significant main effect for CC ($F=4.52$; d.f. = 1, 77; $0.025<P<0.05$), but not for social contact (SC) ($F=1.88$; d.f. = 1, 77; $0.10<P<0.20$) or their interaction ($F=3.52$; d.f. = 1, 77; $0.05<P<0.10$). The actual DBR scores as well as back-transformed means and 95% confidence limits are presented in Table II.

TABLE II

Distribution of DBR scores by critical comments and social contact

DBR scores	*Social contact <35 hours*		*Social contact >35 hours*	
	CC<6	*CC>6*	*CC<6*	*CC>6*
0	4	5	3	–
1	6	14	6	1
2	6	8	4	1
3	4	9	–	2
4	–	2	2	3
5	–	1	–	–
Mean*	1.4	1.7	1.3	2.9
(95% CL)*	(0.9, 1.9)	(1.3, 2.1)	(0.8, 1.9)	(1.9, 4.1)

*Back-transformed after using $\sqrt{(x+1)}$ transformation.

Therefore there appears to be a relationship between critical comments and disturbed behaviour, but it is less clear-cut than Mintz *et al*'s analysis suggests.

In conclusion

Notwithstanding the points that Mintz *et al* have raised, we consider our findings cast doubt on the generality of the relationship between expressed emotion and relapse: in our sample of first-episode schizophrenic patients, no feature of the Camberwell Family Interview emerged as an important predictor. Obviously more research is required. Progress will be made when findings can be replicated by both sceptics and believers.

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The Phenomenology of Acute Organic Psychosis

Comparison with Acute Schizophrenia

J. CUTTING

The psychopathology of acute organic psychosis was investigated by interviewing 74 patients using the Present State Examination (PSE). Their delusions, perceptual disturbance, thought disorder, and emotional disorder were categorised and then compared with those seen in 74 acute schizophrenics. In acute organic psychosis there was a particular pattern to the delusions, perceptual disturbance, and thought disorder, which was quite unlike that seen in acute schizophrenia. These results have implications for theories claiming that schizophrenia is an organic psychosis. It is suggested that the psychopathology in acute organic psychosis has very different origins from that seen in schizophrenia.

Over the past decade, several convincing pieces of evidence have emerged to suggest an organic cause for schizophrenia. Chief among these are the facts that schizophrenics have an unusually high rate of perinatal complications (Reveley *et al*, 1984), that they tend to have been born in the winter months when viral infections are rife (Hare & Walter, 1978), and that a minority have ventricular enlargement on CT scan (Johnstone *et al*, 1976).

Facts such as these have meant that the psychogenic and social formulations of the 1950s and 1960s have given way to a more committed organic position, illustrated by the title of a recent American book, *Schizophrenia as a Brain Disease* (Henn & Nasrallah, 1982). In the 1950s and 1960s, an organic cause for schizophrenia was so disputed that psychoses with phenomena similar to those of typical schizophrenia arising in epileptics (Slater *et al*, 1963) or in those with endocrine disease (Bleuler, 1951) had to be labelled as schizophreniform psychoses: now, the situation is reversed, and certain organic psychoses, particularly those occurring with temporal lobe damage, are regarded by some psychiatrists (e.g. Flor-Henry, 1983) as a model for typical schizophrenia itself.

What is forgotten in this resurgence of interest in the organic basis of schizophrenia is the fact that those psychiatrists who first described the condition were at pains to stress the *fundamental differences* between schizophrenia and all known organic conditions. Bleuler (1911) wrote:

> In schizophrenia, even when well-advanced, all the simple mental functions, as far as we know, are intact. In particular, memory, unlike what happens in true dementia, is unaffected . . . One may find to one's surprise that under an apparent envelope of dementia, intelligence is much less affected than one might imagine, as if it were only asleep.

Jaspers (1913) commented:

> We may note the profound psychopathological difference between, for example, general paralysis and schizophrenia, between the crude organic *destruction* – machinery smashed up – and the *distraction* of mind.

The purpose of this paper is not to undermine the organic claims concerning the cause of schizophrenia, but to show that the clinical picture of schizophrenia is not the same as that of a symptomatic psychosis. Therefore, if there is a biological basis for schizophrenia, it cannot be the same as that underlying the usual symptomatic psychoses in which an essentially similar mental state may be produced by very diverse physical conditions. The aim is to show that, although delusions, hallucinations, emotional disturbance and formal thought disorder are common to both organic psychosis and schizophrenia, the patterns in the two cases are quite different. It might be considered naïve to point out such differences, when the distinction between 'functional' and 'organic' phenomena is so well known. However, the recent blurring of the functional–organic distinction, as outlined above, makes it worthwhile to re-examine the classic phenomena of an organic psychosis. The point at issue is whether differences from schizophrenia are as great as traditional teaching would have us believe, or as little as some contemporary psychiatrists are now suggesting.

The traditional view of a uniform pattern to the clinical presentation of an acute organic psychosis despite a multitude of causes, as well as its distinction from functional psychosis, can be attributed to the writings of Bonhoeffer (1909). He wrote:

> Crude exogenous organic damage of the most varying kind can produce acute psychotic clinical pictures of a basically uniform kind.

The detailed study by Wolff & Curran (1935) supported this view, and more recently Lipowski (1980) has suggested a physiological mechanism, involving arousal and sleep, which explains how different physical conditions can produce the same clinical picture. In fact the literature on the general phenomenology of acute organic psychosis is rather meagre, most work on the subject being reports of yet another physical condition or drug which can cause the clinical syndrome, with little appreciation on the part of the author that they are dealing with a 'uniform' disorder. On the question of the difference between an acute delirious picture and schizophrenia, the different course and the frequent EEG abnormalities in the former are undoubtedly powerful aids in differential diagnosis. It is the clinical differences or similarities, however, which are the focus of this paper. To quote Bonhoeffer again, it is the following statement of his which is to be examined:

> It is clear that a complete distinction between the symptomatology of the exogenous clinical pictures and that of the psychoses which we regard as endogenous cannot be maintained.

Method

Patients

Organic psychotics

The general characteristics, physical state and cognitive impairment of the 74 patients included in the present study have already been reported (Cutting, 1980). In that paper, however, only brief mention was made of their psychopathology, and it is this issue which is examined here.

They were consecutive psychiatric referrals from the general wards of two London teaching hospitals, who fulfilled the following criteria: they had to be psychotic (delusions, gross perceptual disturbance or marked thought disorder), and the psychosis had to be secondary to some clearly identifiable organic cause. Of the 100 psychotic patients seen, 26 had to be excluded, 16 because the psychosis had antedated the physical condition for which they were admitted, and 10 because the psychosis could equally well have been psychogenic or psychosocial in cause, there being no real evidence of cerebral dysfunction. This left 74 clear-cut cases of acute organic psychosis, none of whom had been psychotic before they developed their physical condition.

Each patient was interviewed using the Present State Examination (PSE; Wing *et al*, 1974), and was given a short battery of cognitive tests: four subtests of the Wechsler Memory Scale, comprising orientation, general information, forward digit span and paired-associate learning.

The mean age of the patients was 57.4 years and the sex ratio was exactly equal.

Acute schizophrenics

The controls were 74 acute schizophrenics (Research Diagnostic Criteria (RDC): Spitzer *et al* (1975)). They had all been seen personally and interviewed using either the PSE or the SADS (Schedule for Affective Disorders and Schizophrenia: Spitzer & Endicott (1978)). The change in the interviewing instrument is not of much consequence because many of the psychotic items in the SADS are identical to or derived from PSE items. For the present study, those patients interviewed with the SADS were reassigned PSE symptoms to make the comparison with organic psychotics possible. The schizophrenics were in-patients in an acute admission ward, and had been interviewed as part of a variety of research projects over the years. For the purpose of comparison with first-episode acute organic psychotics, as many as possible of the 74 schizophrenics chosen were also in their first episode. Due to the relative rarity of such cases, the 62 patients in their first-ever episode were complemented with 12 patients in a second discrete episode. The mean age of the schizophrenics was 27.1; there were 41 males and 33 females.

Procedure

Delusions

The organic patients' delusions were analysed with three main aims: (a) to see if they met the phenomenological criteria for schizophrenia according to the RDC (Spitzer *et al*, 1975) or Catego schemes (Wing *et al*, 1974); (b) to examine the validity of a recently suggested classification of organic delusions (Cummings, 1985) into 'simple persecutory', 'complex persecutory', 'hypomanic' and 'those associated with specific neurological defects'; and (c) to search for specific characteristics.

The schizophrenics with delusions were grouped in a way which allowed the best comparison with organic patients. Four groups were distinguished: those with only first-rank symptom delusions (thought interference, passivity, control, delusional perception) or whose persecutory delusions stemmed from these; those with other non-persecutory delusions only; those with both the above types; and those with only persecutory delusions, who fulfilled other criteria for schizophrenia. The first group were typical Catego schizophrenics, the second group were typical RDC schizophrenics and the third group were typical of both.

TABLE I
Simple persecutory delusions in organic patients

Case	*Sex*	*Age*	*Cause*	*Content of delusion*
1	M	49	Alcohol	Doctor had gun to kill him
2	M	60	Respiratory failure	Someone slipped something in drink to cause him trouble with police
3	F	51	Carcinoma	Husband conspiring against her
4	F	47	Alcohol	Evil eye about; nurses not treating her right
5	M	41	Liver failure	He was being experimented upon
6	F	79	Myxoedema	Nurses poisoning her
7	M	63	Carcinoma	People conspiring to frighten him
8	M	73	Right parietal CVA	Nurses trying to kill him

TABLE II
Mood-congruent delusions in organic patients

Case	*Sex*	*Age*	*Cause*	*Content of delusion*
9	M	64	Carcinoma	Director of Wimpey was his friend; he was filthy; doctors going to kill him
10	M	77	Drugs	He was an 'honourable'; his grandfather was Duke of Bedford; he had royal blood in his veins
11	F	88	Drugs	She was a nuisance; nurses opened her letters; stains on curtains meant she was sick; kicked in mouth
12	F	52	Renal failure	She was a nuisance; patients nearby thought she was getting too much attention; she didn't take tablets
13	F	80	Cardiac failure	She was not paying her way; pension-book not kept up to date
14	M	57	Respiratory failure	He had insulted doctors; doctors were in league with saboteurs
15	F	56	Drugs	She was accused of something wrong (e.g. tearing up box of tissues); porters jeering; she was being given wrong pills
16	M	63	Alcohol	He was on blasphemy charge for masturbating into cider and drinking it; he had defrauded Social Security
17	F	49	SLE	Someone accusing her of walking barefoot; doctors trying to poison her with arsenic in chips

Perceptual disturbance

The modality (visual, auditory etc.), form (hallucination, illusion, distortion, first person, second person) and content (verbal, non-verbal, animal, human etc.) were all analysed to test the traditional view that organic perceptual disturbance is characteristically visual rather than auditory, illusion and distortion rather than hallucination, and non-verbal rather than verbal.

Thought disorder

Any disorder of thinking other than delusion was categorised using the nine headings in the PSE: slowness, pressure, non-social speech, muteness, restricted quantity, neologisms, incoherence, flight of ideas and poverty of content. These proved inadequate for both groups of patients and a further categorisation using Andreasen's (1979) scale of Thought, Language and Communication disorders (TLC) was carried out on the protocols, as it was published after the data had been collected.

Emotional disturbance

The predominant observed emotional disturbance was noted, and was recorded as one of the ten PSE categories: anxious, depressed, histrionic, hypomanic, hostile and irritable, suspicious, perplexed, labile, blunted and incongruous. (Lability was regarded as the presence in the course of the interview of at least three of these.)

Results

Delusions

The organic patients' delusions are summarised in Tables I–III. Thirty-five of the 74 patients had delusions. Cummings' scheme, with some alterations, was helpful in

TABLE III
Complex, bizarre or multiple delusions in organic patients

Case	*Sex*	*Age*	*Cause*	*Content of delusion*
18	F	70	Right parietal CVA	When people washed in sink they scrubbed her back; things shown to her became muddled up in pillow; carrot in windpipe
19	F	69	Drugs	Thoughts controlled; something funny going on; she was dying; woman in next bed being harmed
20	M	58	Cardiac failure	Irish Catholics from Streatham driving to Brixton and harming locals by transmitting decomposed matter into their and his own necks by affecting circulatory system parallel to bloodstream called 'man's money'
21	M	76	Respiratory failure	Wife had left him
22	M	59	Drugs	X-ray girls involved in sexual misdemeanours; wife outside curtain; something in head turning to dust
23	F	40	Carcinoma	Mother, sister and herself dying; relatives in ward; nurses going to kill her
24	F	76	Myxoedema	Relatives and herself had died; doctor on ward going to marry a beautiful person
25	F	69	Electrolyte imbalance	Husband eaten by cannibal; drunken nurses obtaining money from patients by doing unspeakable things; she had VD
26	F	48	Electrolyte imbalance	Fire in hospital; children coming to Casualty choking; husband and friend underneath bed, moving it; psychologist testing her
27	M	68	Carcinoma	Unpleasant incidents on ward blamed on him; all the excrement in ward is his; hospital floating on water; daughter to be shot
28	F	37	Alcohol	Doctor taking blood out of her and putting it back into neck; brother out to kill her, her dog and old ladies
29	M	81	Respiratory failure	People exporting illicit material, giving dummy drugs; he was substitute patient for someone else
30	M	62	Cardiac failure	About to die; nurses moving things around in bags, possibly dynamite, and involving IRA; doctors not real doctors
31	M	27	Epilepsy	About to die; people changing things to test him; discrepancy between his thoughts and what he expected to think
32	F	28	SLE	Something bad going to happen; mother dying
33	M	63	Cardiac failure	Building bombarded; police all around
34	F	57	Drugs	Son down road taking drugs; men out to kill husband
35	F	37	Carcinoma	Baby taken away; nurses met daily to decide who was worst patient on the ward

providing a framework for classification. Eight patients had simple persecutory delusions alone (Table I). Nine had predominantly *mood-congruent delusions* (Table II). This replaces Cummings' term 'hypomanic delusions', as only one patient had delusions congruent with elation, whereas one had a mixed affective picture, and seven had depressive delusions. The remaining 18 deluded patients had complex, multiple or bizarre delusions, the nature of which were inadequately conveyed by Cummings' term 'complex persecutory delusions'. Table III shows that they were neither mood-congruent (although a depressive content was often present) nor predominantly persecutory (although this component was often part of the picture). One patient (case 18), with a right parietal lesion, believed that objects in front of her were physically enmeshed with her body and bed, a belief which probably derived from the sort of perceptual distortions described by Critchley (1953) in patients with parietal damage. For this reason it was placed in Cummings' category of 'delusions associated with specific neurological defects'. (His cases within this category – cortical blindness and its denial, aphasia and its denial, reduplicative paramnesia – are, in fact, better regarded as 'delusions based on a specific neuropsychological deficit', as many deluded and non-deluded people might have an associated neurological lesion without this constituting a cause.)

If Cummings' term inadequately represents the delusions in Table III, is there any better way of characterising them? Only two deluded patients could be regarded as showing first-rank symptoms (cases 19 and 20), compared with 56 of the 67 deluded schizophrenics (Table IV), and in the first

TABLE IV

Grouping of deluded schizophrenics and organic patients

Group	*Schizophrenia (number of patients)*	*Organic psychosis (number of patients)*
1. First-rank symptom delusions only (thought intrusion, broadcasting, withdrawal, echo; control, alien penetration; primary), with or without persecutory delusions	37	0
2. First-rank symptom delusions with non-persecutory delusions	19	2
3. Non-persecutory delusions without first-rank symptom delusions	10	16
4. Persecutory delusions only	1	8
5. Mood-congruent delusions only	0	9
6. No delusions	7	39
	74	74

TABLE V

Delusional themes in 29 schizophrenics with non-first-rank-symptom delusions

1. Night club singer in love with him
2. Passing on classified secrets
3. Going to have eight babies; father died and came to life
4. Brain rotating; other people wearing her clothes
5. Time altered and everything had happened before
6. Pregnant
7. Sister measuring doctor's pulse on landing below
8. Son of God; brother on same aeroplane; blood sucked out
9. Turned into a dog; world ending; she and others dying
10. Turned into different person
11. Back broken
12. Body would wash away if he had shower
13. Mother died by falling off donkey
14. Vagina burnt
15. Half Chinese and 10% woman
16. Going to die in 100 days
17. Had different name
18. Everything added up to 20
19. Parents not his real parents
20. Right side of body empty; personality shifted to left shoulder
21. Russians invading South London and affecting weather
22. Second coming about to occur; testicles going to be cut off
23. Throat cut; going to die; boyfriend would be harmed if she said something
24. Dying; being turned into a man; bleeding to death
25. Wife being impersonated on phone and wired up; head blown up
26. Christ
27. Throat diseased
28. Died several times in past
29. Things which happened in past (e.g. kissing in 1967) meant rain in 1982

of these there was marked clouding of consciousness, so only one patient out of the entire 35 can be regarded as a Catego nuclear schizophrenic (S+ in Wing *et al*'s scheme). And even though 15 had bizarre or multiple delusions, one of the criteria for probable schizophrenia in the RDC scheme, in only six of these did the delusions last 2 weeks, another necessary criterion.

Two characteristic themes can be discerned, however, in the organically induced delusions in Table III. These can be described as a belief in *imminent misadventure to others* and a belief in *bizarre happenings in the immediate vicinity*. Fear of misadventure to others was evident in 12 organic cases, and bizarre goings-on in the immediate vicinity in 10 organic cases. The particular link with organic psychosis is emphasised if one inspects Table V, which summarises the themes of the schizophrenics' non-first-rank-symptom delusions. Only six of the 67 deluded schizophrenics had comparable themes: something going to happen to boyfriend if she said something (case 23), nursing sister at top of stairs measuring pulse of doctor in ward below (case 7), she and others were dying as world was ending (case 9), mother had died by falling off a donkey into the sea (case 13), father had died and then returned to life (case 3) and wife was being impersonated and wired up (case 25). The most significant discriminator between organic and schizophrenic delusional themes appeared to be whether others were the *victims or principal characters* in an unfolding drama as opposed to *agents perpetrating some action against the self*. In 15 of the 35 deluded organic patients the self was *not* the principal target or centre of the drama, whereas this was true in only six of 67 deluded schizophrenics ($\chi^2 = 16.2$, $P < 0.01$). One cannot simply argue that the organic delusions were mere misinterpretations of real events in the immediate environment, because most were taking place at a distance (e.g. down the road, in the X-ray department, outside the building, in the locality of Brixton, in Casualty). Indeed, it was the precise spatial location of the events recounted that gave the organic delusions much of their character.

Perceptual disturbance

Of the organic psychotics, 25 had visual hallucinations (people in eleven cases, people and animals in five, animals or insects in six and objects in three). Auditory hallucinations occurred in 13 cases (non-verbal in two (bagpipes, babies crying), own name called out in two, relatives calling out for help in two, snatches of conversation in two (lovers' tiffs, ward gossip), God's voice in one (content of which patient would not divulge), tape-recorder in one (content unknown), voices talking to the patient in one (with whom she was conversing), and voices talking about the patient with an affective content in two ('Jesus Christ he's on a masturbation charge.' 'She'd better come with us otherwise she'll come to harm.'). Three patients had tactile hallucinations (something crawling on her, bed felt wet, bed moving). Three patients had visual distortions (things appeared elevated and as if in a mirror, things appeared to become enmeshed in her bed and body, corner of room looked like a dirty old barn).

In the schizophrenic group, by comparison, 12 patients had visual hallucinations (people in two, animals in one, funeral scene in one, witches in one, words on ticker-tape in one, disembodied parts of body - eye, leg - in two, objects in one and not divulged in three). There were 25 patients with auditory hallucinations, all voices (discussing or commenting or arguing in third person in ten, echoing thoughts in three, telling the patient what to think or do in three, calling name or single words in four and content not divulged in five). Tactile hallucinations occurred in three patients (pressure on eyeball, burning in vagina, blows on back). Visual distortions were present in 25 patients (predominantly in six that colours were vivid, in eleven that objects around them were strange, and in eight that people looked distorted).

One feature that was confined to the organic group was mistaken identification of people: ten of the organic patients either mistook ward staff for close relatives or thought a visiting relative was someone else. None of the schizophrenics did this.

Thought disorder

Formal thought disorder was present in 47 organic patients. (This rating was made independently of the presence of delusions: 18 of these 47 were also deluded, and 17 of the 27 non-thought-disordered were deluded.) Using the PSE items (Table VI), only 20 of these 47 could be categorised. Using Andreasen's scale, however, all 47 could be placed in at least one of her 18 categories, albeit with some difficulty in the case of tangentiality and illogicality.

The largest group was made up of 15 patients whose thinking was illogical to a marked degree. In all cases this emerged during questions about orientation. One woman claimed that it must be 20 May because her sister had died then, without explaining why the date of her sister's death should determine today's date. The remaining 14 gave fantastic or grossly incorrect answers to questions about orientation in place (e.g. East Finchley airport, waiting at a bus-stop, in Wales one minute and in London the next, on a boat) and when asked to justify their statements resorted to evasion (e.g. 'Well, it looks a bit like an airport') or inconsequential remarks (e.g. 'It's a boat because all my friends are here'). Their disorientation could not be attributed entirely to memory or perceptual disorder, because of the bizarre choice of place in some instances (even non-existent, e.g. East Finchley airport) and because in several instances a patient would try to explain a correct perception within the false framework (e.g. one man, who believed he was at home and found himself surrounded by beds, was puzzled why his wife had bought so many beds).

TABLE VI
Formal thought disorder

	Number of patients with marked abnormality[1]	
	Organic patients (n = 74)	*Schizophrenics (n = 74)*
PSE items		
Slowness	14[2]	3
Pressure	2[2]	0
Non-social speech	0	0
Muteness	0	0
Restricted quantity	14[2]	1
Neologisms	2	4
Incoherence	1	3
Flight of ideas	2[2]	0
Poverty of content	1	1
Total PSE items	20	12
TLC items		
Poverty of speech	14	1
Poverty of content of speech	1	1
Pressure of speech	2	0
Distractible speech	2	0
Tangentiality	6[3]	2
Derailment	0	10
Incoherence	1	3
Illogicality	15[4]	0
Clanging	0	0
Neologisms	0	1
Word-approximation	2	0
Circumstantiality	4	1
Loss of goal	0	1
Perseveration	0	0
Echolalia	0	0
Blocking	0	1
Stilted speech	0	2
Self-reference	0	0
Total TLC items	47	23

1. Only predominant feature counted.
2. The two patients with flight of ideas also had pressure, and the 14 with slowness also had restricted quantity.
3. Speech better regarded as irrelevant rather than tangential (see text).
4. Of these, 14 had fantastic disorientation which they justified illogically (see text).

The next largest group - 14 patients - had a combination of apathy, slow speech and restricted or impoverished output. They were not grossly disorientated but complained subjectively of muddled thinking and did poorly on the verbal learning test.

Six patients were categorised as having tangential speech. According to Andreasen (1979), tangentiality is

> replying to a question in an oblique, tangential or even irrelevant manner. The reply may be related to the question in some distant way. Or the reply may be unrelated and seem totally irrelevant.

TABLE VII
Emotional disturbance

	Number of patients with marked disturbance[1]	
	Organic patients	*Schizophrenics*
Anxiety	8	7
Depression	14	10
Histrionics	0	0
Hypomania	10	3
Irritability/hostility	4	1
Suspiciousness	3	2
Perplexity	5	8
Lability	11	3
Blunting	0	1
Inappropriateness	0	6
Total	55	41

1. Only predominant emotion counted.

In the organic patients the replies were irrelevant rather than tangential. For example, one man, when asked where he thought he was, announced that he was allergic to flowers. Another, asked what the matter was, said that he kept seeing 'a hand like a thieving monkey's paw crossing my vision'. Another replied to a simple question on orientation: 'You're nervous of people, I can see it.'

The remaining 12 organic patients with thought disorder had either prominent circumstantiality (giving long rambling accounts), distractibility (interrupting the interview every time there was a noise from a neighbouring bed), word-approximations ('doctoress' for housewoman, 'man's money' (case 20 in Table III)), pressure of speech with flight of ideas, incoherence (mumbled ungrammatical statements) or poverty of content (vacuous answers).

The 23 acute schizophrenics with prominent thought disorder (at least grade 2 on Andreasen's scale) had a different pattern, although numbers were too small for any but three differences to be significant (poverty of speech, $\chi^2=12.5$, $P<0.01$; derailment, $\chi^2=9.8$, $P<0.01$; illogicality, $\chi^2=15.8$, $P<0.01$). In Andreasen's scheme, derailment and tangentiality have the same form, but in the former the abnormality is most obvious in sustained discourse, while the latter refers to oblique or irrelevant replies to questions. Schizophrenics were more likely to offer lengthy explanations of how they felt and thus derailment was more noticeable. In the schizophrenics, in contrast to the organic patients, the link between one statement and another, or question and answer, was usually just discernible, as in the following schizophrenic's answer to my question, 'Do you think you had a breakdown?' 'Yes it's in your. I suppose it's a lack of faith. It's as if you want to arrive at some results and fear things as if improvement. It's just a question of learning.' The organic patients tended to answer some questions quite competently and then intersperse a single irrelevant remark.

Emotional disturbance

There were no marked differences between the two diagnostic groups (Table VII). The organic patients were more likely to be hypomanic ($\chi^2=4.1$, $P<0.05$) or labile ($\chi^2=5.0$, $P<0.05$), and more likely to have an overall emotional disturbance ($\chi^2=13.0$, $P<0.01$).

Discussion

The aim of the study was to reconsider the traditional view that acute schizophrenia and acute organic psychosis are quite distinct phenomenologically. Several authors in recent years have been trying to link these conditions at a biological level, and it might be that phenomenological differences have been exaggerated in the past. The overwhelming conclusion must be that although delusions, hallucinations, formal thought disorder and emotional disorder are common to both, with the exception of the last of these the pattern in each case is quite distinct. A subsidiary aim was to draw attention to the psychopathology of acute organic psychosis itself, a subject which has received little attention in the recent literature. On this point, although the observations were rather impressionistic, it appeared that the delusions, perceptual disturbance and thought disorder had characteristics which made them quite unlike those seen in other psychoses. (The type of hallucinations seen has long been regarded as characteristic, but less is written about the delusions and thought disorder.)

Some methodological problems

Patients with an acute organic psychosis tend to fluctuate in their mental state, even from minute to minute, and a single interview is liable to be unrepresentative of their psychopathology, particularly since they may be amnesic for what they believed, felt or experienced a short while ago. This is likely to be less true in the case of acute schizophrenia, but even here one cannot hope to elicit the entire range of abnormal beliefs in one interview.

In the organic patients it was sometimes difficult to be sure whether a false statement (e.g. my mother is dying) was an autochthonous belief, a mistaken belief through misperceiving another patient for a relative, a true hallucination, or muddled reasoning mediated by a variety of cognitive defects including amnesia and inattention. In many cases, however, the content of the organic delusion either was very bizarre or concerned events outside the patient's immediate environment, so that simple perceptual explanations could be rejected. In any case some schizophrenics' statements are difficult to categorise precisely as perceptual or ideational, and even their delusions, according to some authorities, may originate in an earlier perceptual disturbance (e.g. delusional mood (Conrad, 1958)), so the problem in categorising the organic phenomena is not specific.

The difference in age between the two groups could conceivably account for some of the differences in delusional content; but if one subdivides the organic patients into a younger and older group, the 39 patients aged 58 or older contained four with paranoid, five with mood-congruent and nine with bizarre delusions, and the 35 patients aged 57 or under contained four with paranoid, four with mood-congruent and nine with bizarre delusions, virtually identical proportions. Grahame (1984) reported that patients with late paraphrenia (schizophrenia of old age) were less likely to show some of the first-rank symptoms (particularly thought insertion and withdrawal) than younger schizophrenics. However, nearly 60% of his patients, all of whom were over the age of 60, had at least one first-rank symptom, a proportion which is no different from that reported in younger schizophrenics.

Next, it might be argued that it is inappropriate to use acute organic psychotics as a subject group because the claims about the similarity between schizophrenia and organic conditions have been made with chronic organic states (e.g. temporal lobe lesions (Flor-Henry, 1983) and birth injury (Murray *et al*, 1985)). This criticism is difficult to refute, and indeed it is undoubtedly true that some chronic organic psychoses (e.g. alcoholic hallucinosis (Cutting, 1978), epileptic psychosis (Toone *et al*, 1982)) are phenomenologically identical to schizophrenia, with no apparent organic basis. One can question, however, whether schizophrenia as diagnosed, for example, by DSM-III criteria, is *too* broadly conceived. If organic psychosis does indeed produce a characteristic set of phenomena, then this should lead to more refined criteria for 'familial' or 'sporadic' schizophrenia. It should then be possible to demonstrate that DSM-III criteria encompass a heterogeneous collection of psychoses. In this respect the study of acute organic psychosis would be justified as helping to delineate the boundaries of organic and 'functional' psychoses.

It could also be argued that it is inappropriate to use schizophrenics as a control group if the issue in question is the boundary between organic and functional psychotic phenomena. A better control group might have been a consecutive series of non-organic psychotics, including manics, depressive psychotics and paranoid psychotics as well as schizophrenics. This heterogeneous control group was employed in an earlier study (Cutting, 1980), and even fewer functional psychotics than in the current schizophrenic sample had the common organic delusional themes of imminent misadventure to others or bizarre happenings in the immedate vicinity. The present control group was chosen in this study because the main issue addressed was differences between a symptomatic organic psychosis and schizophrenia.

Main findings

Of the four areas of psychopathology examined – delusions, perceptual disturbance, formal thought disorder and emotional disorder – the least useful in differentiating acute organic and schizophrenic psychosis was a disorder of emotion. This is not surprising, considering that neither condition incorporates this as a major diagnostic criterion, if one excludes flattening or inappropriate affect, which was found to be rare in first-admission schizophrenics. Lability of emotions was the most reliable indicator of an organic psychosis, but was neither particularly specific nor sensitive to the condition.

The form of the perceptual disturbance was relatively specific to each condition. Visual hallucinations occurred in 34% of acute organic psychotics (people in 22% and animals in 15%) and only 16% of schizophrenics (people in 3% and animals in 1%). Auditory hallucinations, on the other hand, occurred in 34% of schizophrenics (all voices) but in only 18% of organic psychotics (voices 15%). Unknown voices in the second or third person, commenting, arguing, instructing or echoing thoughts, were the best discriminator, occurring in 22% of schizophrenics and only 4% of organic patients. In this study misidentification of persons was confined to organic psychotics, occurring in 14% of patients, although it is not unknown in acute schizophrenia (Cutting, 1985).

Formal thought disorder was common, occurring in 64% of organic patients compared with 31% of schizophrenics. Although there was a suggestion that the patterns in the two groups of patients were different, it was difficult to confirm this, because the scales used had been devised for schizophrenics and depressives, and the categories reflected this. For example, 19% of the organic psychotics were apathetic, responded monosyllabically to questions and complained of subjective confusion. The nearest category in Andreasen's scheme was poverty of speech, which, although rare in acute schizophrenics in this study, occurred in 22% of Andreasen's depressed subjects. Of the organic psychotics, 20% gave absurd answers to questions on orientation, most of which could not be attributed to inattention, amnesia or perceptual disorder. These were placed within the category of illogicality. Although not seen to this degree in acute schizophrenics in this study, this abnormality was noted in 27% of Andreasen's acute schizophrenics. The most striking abnormality in organic patients, although it was only seen in 8%, was the occurrence of intermittently irrelevant comments in the course of an otherwise fairly lucid conversation. This was categorised as tangentiality, although it appeared to lack the subtle links which mark the speech of schizophrenics. In summary, the thought disorder of organic patients appeared to have a quality which set it apart from that seen in the major functional psychoses, although how this should be categorised remained unclear.

The most surprising finding in the study was the common themes discerned in the delusions of organic patients, and the difference from those characteristically seen in schizophrenia. Not surprisingly, first-rank symptom delusions were very rare, and paranoid and mood-congruent delusions were common; but the most striking finding was that nearly a quarter of organic patients and nearly half of all the deluded organic patients shared a common theme of *imminent misadventure to others* or *bizarre happenings in the immediate vicinity*. This was rarely encountered in schizophrenics. The core feature appeared to be the enactment of some tragedy or misdeed by others, with the self a helpless onlooker, whereas in schizophrenics, even when similar themes occurred, the self was intimately involved. There are several explanations for this common theme. The delusions might have had a basis in misperception of events in the environment; this, however, is not an adequate explanation as the events tended to be outside the patient's immediate surroundings. Another possibility is that the delusions were confabulatory phenomena, with fluctuating amnesia the basic ingredient. This also cannot have been true for all delusional themes, because the events were in the main fantastic and implausible and were not mere filling-in of memory gaps (e.g. hospital floating on water, husband and friend underneath bed). One plausible and general explanation is that the delusions are a form of dream-like thinking. This would fit Lipowski's (1980) view of delirium as 'a disorder of wakefulness . . . with vivid dreams, nightmares, and oneiric or dreamlike mentation'. The absurd content of the delusions is also reminiscent of the thematic material in dreams, as described by Freud (1900) and others.

In conclusion, the phenomenology of an acute organic psychosis bears little resemblance to that of acute schizophrenia. There are therefore grounds for believing that if acute schizophrenia is due to organic causes then these must have a very different effect from the way in which an acute organic psychosis is brought about.

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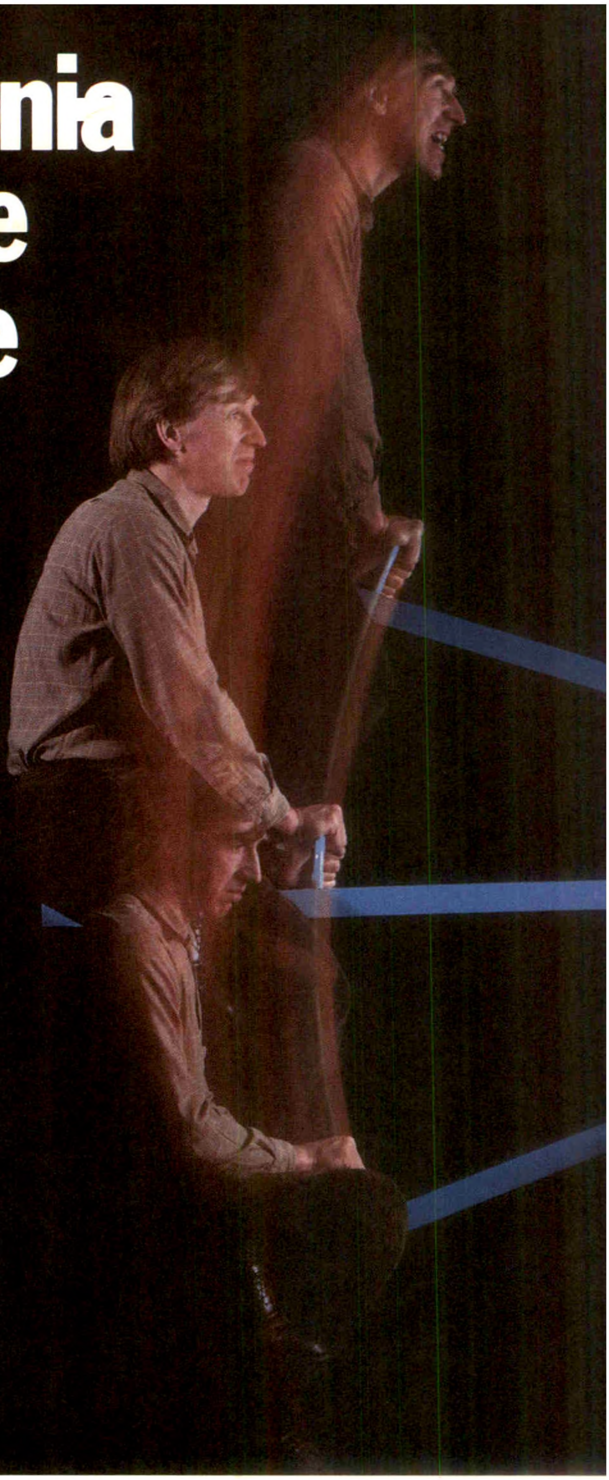
Schizophrenia therapy: the importance of balance



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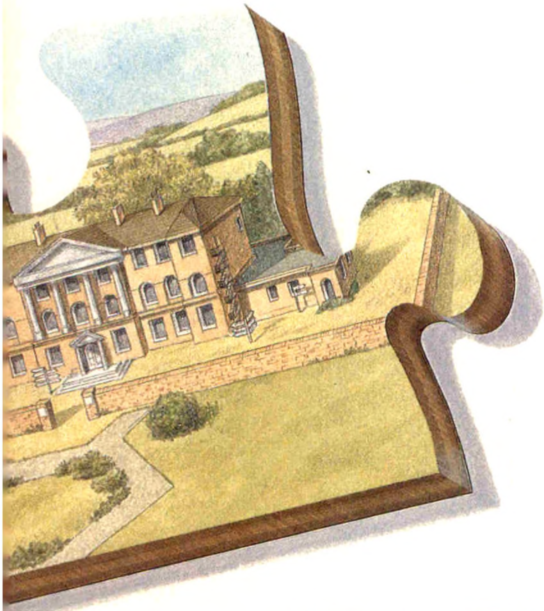

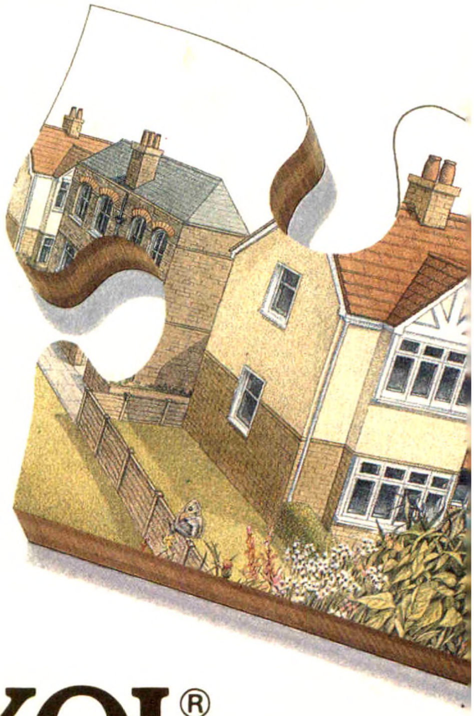

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British Journal of Psychiatry (1987), **151**, 333–336

Haloperidol Decanoate *v*. Fluphenazine Decanoate as Maintenance Therapy in Chronic Schizophrenic In-patients

J. P. McKANE, A. D. T. ROBINSON, D. H. WILES, R. G. McCREADIE and G. S. STIRLING

In a double-blind study of 38 chronic schizophrenic in-patients, haloperidol decanoate was compared with fluphenazine decanoate as maintenance therapy over 60 weeks. Both drugs were given by injection at 4-week intervals. Haloperidol and fluphenazine were assumed to be equipotent; the mean starting dose of the former was 127 mg and of the latter 106 mg. The number of withdrawals over 60 weeks was similar in both groups but relapses, strictly defined, were significantly more frequent in the haloperidol group. When patients were switched to haloperidol, Parkinsonism diminished more quickly than in the fluphenazine group, but after 60 weeks there was no difference in severity in the two drug groups. The higher relapse rate and the quicker reduction in Parkinsonism in the haloperidol group might be due to a misjudgement in equivalent doses of the two drugs. Plasma haloperidol steady state levels were reached in most patients by 8–12 weeks. Plasma neuroleptic and prolactin levels, week-by-week systemic drug availability and Parkinsonism showed less variation between injections with haloperidol than with fluphenazine.

Haloperidol decanoate was introduced into the United Kingdom in 1982 for use primarily as maintenance therapy in chronic schizophrenics. Although oral haloperidol has been widely investigated (Ayd, 1980), there have been only four published double-blind controlled studies comparing haloperidol decanoate with other active medication in schizophrenic patients (Chouinard *et al*, 1984; Wistedt *et al*, 1984; Kissling *et al*, 1985; Bechelli *et al*, 1985). The principal drawback in these studies was the short follow-up period: 5–8 months. Firstly, the present study examines, over a 60-week period, the efficacy and Parkinsonian side-effects of haloperidol decanoate when used as maintenance therapy in chronic schizophrenic in-patients, by comparing it with fluphenazine decanoate; and, secondly, as an injection interval of 4 weeks is recommended by the manufacturer, the study examines plasma haloperidol levels and week-by-week systemic drug availability.

Method

Patient selection

All in-patients in the hostel and long stay non-geriatric wards of Crichton Royal Hospital, Dumfries, were considered ($n = 180$). A patient entered the study if (a) he (or she) was physically fit; (b) he fulfilled the criteria of Feighner *et al* (1972) for definite or probable schizophrenia; (c) in the opinion of medical and nursing staff he was well controlled on and benefiting from antipsychotic medication; (d) he, and where traceable, his next of kin, gave consent. The criteria were fulfilled by 38 patients – 22 men and 16 women; their mean age was 56 years (range 31–71 years) and mean length of illness 31 years (range 10–50 years).

Medication

With the stipulation that equal numbers should receive each drug, patients were randomly and blindly allocated to injections every 4 weeks of haloperidol decanoate or fluphenazine decanoate; thus 19 patients received haloperidol and 19 fluphenazine. The starting dose of the latter was the approximate therapeutic equivalent of the previously prescribed neuroleptic (Davis, 1976; Wyatt & Torgow, 1976; British National Formulary, 1984); the haloperidol dose was calculated on the assumption that 1 mg haloperidol was equivalent in potency to 1 mg fluphenazine (as suggested by Suy *et al*, 1982). For the first 12 weeks, the 'run-in period', thioridazine at a dose of up to 100 mg four times a day could be added if a patient's mental state or behaviour deteriorated. Where this happened, the next dose of haloperidol or fluphenazine was increased by an amount equivalent to the additional thioridazine required. In this way, it was intended that patients would enter the 'study proper' on an adequate and appropriate dose. If a patient was still receiving thioridazine at the end of the run-in period, he did not immediately proceed to the study proper, but had subsequent doses of study medication increased until stabilisation of mental state and behaviour was achieved. After the 12-week run-in period, the study proper lasted a further 48 weeks.

Anti-Parkinsonian medication could be prescribed if Parkinsonism was rated as more than 'mild' (see below).

Assessment

Before the run-in period, week 0, and at weeks 4, 8, 12 (the beginning of the study proper), 28, 44 and 60, and at the time of any relapse, one of two psychiatrists assessed the patient's mental state by the Krawiecka Scale (Krawiecka *et al*, 1977), Parkinsonism by the Simpson & Angus Scale (Simpson & Angus, 1970) and a global five-point scale (0 = absent, 1 = doubtful, 2 = mild, 3 = moderate, 4 = severe), and tardive dyskinesia (TD) by the AIMS Scale (US Department of Health, Education and Welfare, 1976). Akathisia was not assessed. Nurses assessed behaviour by the Wing Ward Behaviour Scale (Wing, 1961). To assess inter-rater agreement, 12% of mental state ratings ($n = 26$, from 18 different patients), 8% of Parkinsonism ($n = 45$, from 24 patients) and 7% of TD ratings ($n = 42$, from 25 patients) were made independently by both psychiatrists. Patients were weighed and injection sites were examined at regular intervals.

A patient was deemed to have relapsed if after entry into the study proper there was a deterioration in mental state or behaviour which required thioridazine at a dose of 100 mg four times a day for more than one week. It was the decision of the consultant psychiatrist who was responsible for the patient's care, and who was blind to which drug the patient was receiving, to prescribe thioridazine after discussion with other medical and nursing staff.

Blood samples were taken immediately before an injection at regular intervals over the 60 weeks to measure plasma haloperidol and fluphenazine levels by radioimmunoassay techniques (Wiles & Franklin, 1978), full blood count and liver function tests. Also, to study in detail between-injection plasma neuroleptic and prolactin levels, blood samples were taken from six patients in each drug group immediately before the eighth injection, and then 2, 4, 6, 8, 12 and 24 hours, and 3, 7, 14, 21 and 28 days later. At each sample time, Parkinsonism was assessed by the Simpson & Angus Scale.

Results

At week 0 there were no statistically significant between-group differences in age, sex distribution, and severity of illness and side-effects as measured by the Krawiecka, Wing, Simpson & Angus and AIMS scales (Table I). The mean monthly doses of haloperidol and fluphenazine decanoate at week 0 were 127 mg and 106 mg respectively; and at week 12, 120 mg and 105 mg. Inter-rater agreement on mental state assessment was high (product-moment correlation coefficient $r = +0.85$, $P < 0.001$) as was that on Parkinsonism ($r = +0.90$, $P < 0.001$) and that on TD ($r = +0.80$, $P < 0.001$).

Run-in period

Five patients, two on haloperidol and three on fluphenazine, did not proceed to the study proper: it was impossible to discontinue thioridazine in one patient, a second withdrew, a third became physically ill, a fourth (on fluphenazine) committed suicide, and a fifth developed an acute anxiety state after the suicide. Three patients on haloperidol and four on fluphenazine required thioridazine. Eight patients on haloperidol and ten on fluphenazine required anti-Parkinsonian drugs. In the 33 patients who entered the study proper, Parkinsonism as measured by the Simpson & Angus Scale was significantly less in the haloperidol than the fluphenazine group at the end of the run-in period (t test, $P < 0.01$).

Study proper

Seventeen patients on haloperidol and 16 on fluphenazine entered the study proper.

Relapsers

Significantly more patients on haloperidol relapsed (6 of 17 on haloperidol, 35%; 1 of 16 on fluphenazine, 6%; Fisher's Exact Probability Test, $P < 0.05$). One relapser on haloperidol committed suicide. The monthly dose of haloperidol decanoate at weeks 0 and 12 in the relapsers was 153 mg and 173 mg respectively, compared with 92 mg at both time points in the 11 patients from the haloperidol group who completed the study.

Non-relapsers

Eleven patients on haloperidol and 12 on fluphenazine completed 60 weeks of treatment. Three of the 15 non-relapsers on fluphenazine were withdrawn for other reasons: two refused medication, one developed severe akathisia. At the end of the study the mental state of the 11 patients on haloperidol decanoate was better than that of the 12

TABLE I
Initial severity of illness and side-effects

	Haloperidol group			*Fluphenazine group*		
Rating	*Mean score*	*Standard deviation*	*Range*	*Mean score*	*Standard deviation*	*Range*
Krawiecka total score	8.5	5.0	1–18	8.2	5.4	1–21
Wing total score	5.5	4.1	0–14	4.8	4.4	0–18
Simpson & Angus total score	3.5	3.2	0–12	4.0	3.0	0–10
AIMS total score (items 1–7)	4.6	5.1	0–20	2.9	2.9	0–9

patients on fluphenazine, and ward behaviour in the former group worse (t tests, both $P<0.05$). There were no other statistically significant between- or within-group differences in scores on mental state, behaviour and TD scales.

There were no significant between-group differences in Parkinsonism scores at the end of the study. At some time during the 48-week study proper, eight patients on haloperidol and eight on fluphenazine required anti-Parkinsonian drugs.

After 60 weeks' treatment, there were no significant changes in weight, full blood count or liver function tests. No abnormalities were noted at the site of injection in either treatment group.

TABLE II
Number of patients in each drug group at different stages of the study

	Number of patients receiving haloperidol decanoate	*Number of patients receiving fluphenazine decanoate*
Entry to study	19	19
End of run-in period	17	16
End of study proper	11	12

Plasma neuroleptic levels

(Full details of plasma neuroleptic levels are available on request to RGMcC.)

Plasma haloperidol levels in the 11 patients who completed the study suggest steady state levels were reached in most patients 8–12 weeks after the first injection; t-tests showed there was no statistically significant rise in mean plasma levels when compared with the previous month after week 8. However, in one patient the level was still rising between weeks 44 and 60 and in another between weeks 28 and 44. In the fluphenazine assay, cross-reactions with previously prescribed neuroleptics, especially flupenthixol decanoate and clopenthixol decanoate, made interpretation of plasma 'fluphenazine' levels in the first months of the study impossible, and thus time to reach steady state could not be estimated.

Mean plasma neuroleptic and prolactin levels and Parkinsonism scores were studied in detail in the two subgroups over a 4-week period after the eighth injection. In the fluphenazine group there were significant differences over time in plasma fluphenazine and prolactin levels and Simpson & Angus total scores (F tests, all $P<0.01$). The differences were due to higher measurements in the first few days after injection. No such differences were found in the haloperidol group. There were also significant ($P<0.01$) positive relationships between plasma levels and both prolactin levels and Simpson & Angus scores in the fluphenazine group, but no significant linear relationships in the haloperidol group. There were no significant between-group differences in prolactin levels. Systemic drug availability as measured by the area under the plasma level/time curve was more consistent week-by-week in the haloperidol group; in the fourth week after injection, systemic availability was 85% of that found during the first week, whereas in the fluphenazine group it was 63%.

Discussion

The study examined chronic schizophrenic in-patients previously well maintained on antipsychotic medication. Such patients do not have to face the stresses of life outside hospital and therefore are less likely to relapse (Leff & Vaughn, 1980). A more rigorous test of the comparative efficacy of haloperidol and fluphenazine decanoate would be to examine their use in out-patients or day-patients. Nonetheless, 35% of patients receiving haloperidol relapsed over a 48-week period, a figure significantly higher than that for fluphenazine. There are several possible explanations for this difference in relapse rates. Firstly, haloperidol decanoate may be less effective than fluphenazine decanoate as maintenance therapy. Secondly, the dose of haloperidol may have been too low. In calculating the starting dose it was assumed to be equipotent with fluphenazine. Previous studies have assumed a haloperidol/fluphenazine dose ratio of 1.4:1 (Wistedt *et al*, 1984) or 3:1 (Chouinard *et al*, 1984). It is noteworthy, however, that in the 12-patient subgroup in the pharmacokinetic study there was no significant between-group difference in plasma prolactin levels, a measurement of dopamine blockade, in patients on haloperidol decanoate and fluphenazine decanoate receiving broadly similar amounts of drug. Finally, relapse in the present study was strictly defined. If withdrawal rates for *all* reasons over the 60 weeks are compared, then the significant difference disappears (42% on haloperidol, 37% on fluphenazine).

Although at the end of the study there were no significant between-group differences in Parkinsonism, Parkinsonism diminished more quickly in patients switched from the previously prescribed neuroleptic to haloperidol decanoate than in those switched to fluphenazine. This again may be a dose effect. Where the haloperidol/fluphenazine dose ratio was higher than in the present study (Chouinard *et al*, 1984) there was no between-drug difference in extrapyramidal side-effects; where the ratio was similar (Wistedt *et al*, 1984) anti-Parkinsonian medication was used less in the haloperidol decanoate group.

The time to reach plasma haloperidol steady state levels in most patients, 8–12 weeks, was broadly similar to that in other studies (Derberdt *et al*, 1980; Reyntjens *et al*, 1982). However, in one patient at least, plasma levels were still rising after 44 weeks of treatment. The reason for this is not clear, although it is noteworthy this patient was receiving the highest dose of haloperidol decanoate, 325 mg at 4-week intervals. Perhaps in patients on high doses a 4-week interval between injections is too short.

Injections at 4-week intervals produced flatter plasma neuroleptic/time curves and a more consistent week-by-week systemic drug availability with haloperidol than with fluphenazine. The clinical significance of this is not clear, as the relapse rate with fluphenazine was lower. There is as yet no information as to what plasma level of haloperidol or fluphenazine should be achieved before the chance of relapse is substantially reduced. However, an increase in plasma fluphenazine levels in the first few days after injection was associated with an increase in Parkinsonism.

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A Controlled Comparison of Fluoxetine and Amitriptyline in Depressed Out-patients

J. P. R. YOUNG, A. COLEMAN and M. H. LADER

Fluoxetine, a selective serotonin uptake inhibitor (mean dose 73 mg each morning) was compared with amitriptyline (mean dose 122 mg at night) in a double-blind study of 64 depressed out-patients. Fifty patients completed the 6-week trial. The drugs did not differ with respect to psychiatrists' ratings, but amitriptyline was slightly superior with respect to patients' ratings. The amitriptyline-treated group had complaints of dry mouth and dizziness on standing; the fluoxetine-treated group of sleep disturbances, nausea, and headaches.

Fluoxetine is a potent, selective inhibitor of 5-hydroxytryptamine (5HT, serotonin) (Wong *et al*, 1974, 1975). Unlike many other selective inhibitors such as clomipramine, fluoxetine does not lose selectivity when metabolised to desmethylfluoxetine (Fuller *et al*, 1978). Because of this selectivity it has been extensively evaluated as a possible antidepressant with a low incidence of unwanted effects (Wernicke, 1985). An extensive series of studies have been carried out to compare the efficacy and safety of fluoxetine to those of placebo, imipramine, amitriptyline, and doxepin (Stark & Hardison, 1985). This brief account concerns a controlled double-blind comparison of fluoxetine with amitriptyline, conducted in a busy psychiatric out-patient department of a London teaching hospital.

Method

Physically healthy adult out-patients, aged 20–65, presenting with moderately severe unipolar depression (Research Diagnostic Criteria (RDC); Feighner *et al*, 1972) not requiring electric convulsion therapy (ECT) were eligible for inclusion. A minimum Hamilton Rating Scale for Depression (HRSD) score of 18 was required for recruitment. All patients with schizophrenia or organic features, or those who had been receiving antidepressant drugs or ECT during the 4 weeks before referral, were excluded. All patients were new referrals from general practitioners. Most patients entered were, however, taking sedatives or tranquillisers when first seen in the out-patient department. Successive patients who were suitable for drug treatment were allocated randomly either to fluoxetine or amitriptyline treatment for a period of 6 weeks.

Both drugs and placebo were identically formulated, each capsule containing 20 mg fluoxetine or 50 mg amitriptyline. The dose was limited during the first week to 40 mg fluoxetine in the morning plus placebo at night, or 50 mg amitriptyline at night plus placebo in the morning. During the remaining 5 weeks of the trial a flexible dosage schedule was used to allow 40–80 mg fluoxetine in the morning or 50–150 mg amitriptyline at night, depending on the clinical response and side-effects. Each patient was seen for initial assessment and after 1, 3 and 6 weeks of treatment.

A standard psychiatric history was taken at the first visit. At each visit the patient completed the Beck Scale for Depression (BSD) and two analogue self-assessment scales for depression and anxiety. An independent assessor scored the patient on the HRSD and the Hamilton Rating Scale for Anxiety (HRSA), and assessed the overall severity of the depression. The Beck Scale was also completed by the patient 3 days after entry to the trial. A standard form for rating side-effects was completed by the second psychiatrist, who also regulated drug dosage. This prevented knowledge of side-effects biasing the clinical ratings of the other assessor. On each visit the patient was weighed and ratings made for changes in appetite and for changes in sleep pattern, libido, and diurnal variation of mood and energy. Before starting treatment, and on completion of the trial, blood samples were taken for routine laboratory tests including haematology, renal, hepatic, and thyroid function.

Drop-outs

Of the 64 patients initially entered in the double-blind trial, 14 were withdrawn (seven in the fluoxetine group, seven in the amitriptyline group). The commonest cause (four in each group) was failure to re-attend. No amitriptyline and two fluoxetine patients dropped out because of side-effects (persistent sleep disturbances).

Pre-treatment matching of groups

The fluoxetine group comprised 25 patients (17 females, 8 males) with a mean age of 46.1 years. Five patients had a family history of affective illness; ten patients had experienced previous depressive episodes. In the amitriptyline group of 25 (17 females, 8 males) the mean age was 46.6 years; five patients had a family history of affective illness and ten patients had experienced depressive episodes. Before entering the trial, 15 patients had previously received tricyclic antidepressants. Of these, nine were given fluoxetine treatment and six received amitriptyline during the course of the trial. Analysis of variance showed no significant pre-treatment differences between the groups for any of the variables.

Dosages attained

The mean daily dosages by week 3 were 59 mg fluoxetine and 90 mg amitriptyline; by week 6 they were 73 mg and 122 mg respectively. No additional medication was prescribed apart from diazepam and temazepam in low dosage as daytime and night-time sedatives respectively. These were mostly continued from previous drug therapy. In the fluoxetine group, diazepam was prescribed for 21 patients (mean dosage 5.8 mg daily) and temazepam for 24 patients (mean dose 12.4 mg nightly). In the amitriptyline group, diazepam was given to 23 patients (6.5 mg) and temazepam to 23 patients (13.3 mg). There were therefore no major differences between the treatment groups.

Results

Change with treatment

Analyses of variance were carried out on the data from the rating occasions, differences between drug effects being obtained from the drugs × week interaction term. Mean scores on the HRSD and HRSA scales and on overall severity improved highly significantly during the 6 weeks of treatment, with no differences in efficacy between fluoxetine and amitriptyline. The HRSD items were grouped into those which were 'pure' depressive items and those that assessed concomitant anxiety, excluding those that could be affected by drug side-effects such as sleep and appetite. No differences between the drugs were found with respect to improvement in the depressive items. Patients were also rated at the end of the 6 weeks with respect to their degree of improvement. No differences were found between the drugs with respect to these categories.

Differences between the drugs reached significance with respect to weight and appetite (Table I), amitriptyline increasing but fluoxetine decreasing both variables.

The only clinical rating to show differences between the two drugs was the Beck Scale for Depression (Table I): the diminution with fluoxetine treatment was significantly less than that with amitriptyline. The 'pure' depressive items of the scale were grouped and analysed separately; no drug differences were found. Furthermore, the overall self-ratings did not show drug differences.

Unwanted effects

The number of patients at each assessment with ratings of each side-effect higher than at the pre-treatment assessment (treatment-emergent side-effects) was computed (Table II). Complaints of dryness of the mouth and of dizziness on standing up (postural hypotension) were significant in patients on amitriptyline – dryness of the mouth was marked throughout, hypotension at week 3 only. The only treatment-emergent side-effects rated by patients on fluoxetine were nausea, especially towards the end, and headache at week 1. Two of the patients initially entered into the trial dropped out because of side-effects – both were in the fluoxetine group and had sleep disturbances. No significant abnormalities in the laboratory tests were found.

Predictors of improvement

Family history of affective disorder, age, sex, and previous treatment with a tricyclic antidepressant in the current episode were evaluated as possible factors predicting response to fluoxetine or amitriptyline on the HRSD or HRSA scales. No relationship was found.

TABLE I
Comparison of fluoxetine and amitriptyline

Variable	*Drug*	*Pre-treatment*	*Time of examination 3 days*	*1 week*	*3 weeks*	*6 weeks*	*Drugs × weeks*	*Analysis of variance weeks*
Weight: kg	Amitriptyline	65.9		66.4	66.8	67.2	NS	10.2 $P<0.001$
	Fluoxetine	65.6		65.0	64.7	64.0	(d.f. = 3,144)	(d.f. = 3,144)
Appetite[1]	Amitriptyline	5.8		6.2	7.0	6.9	NS	3.1 $P<0.03$
	Fluoxetine	6.1		5.6	5.5	6.0	(d.f. = 3,144)	(d.f. = 3,144)
Beck Scale for Depression[2]	Amitriptyline	48.1	47.9	43.0	36.4	33.0	43.9	2.6 $P<0.05$
	Fluoxetine	44.0	45.4	39.8	36.6	35.3	$P<0.001$ (d.f. = 4,192	(d.f. = 4,192)

1. Rated on a 13-point scale
2. 21 items; maximum score 84.

TABLE II

Numbers of patients with treatment-emergent side-effects

		Week					
		1		*3*		*6*	
Side-effect	*Drug*	*n*	*P*	*n*	*P*	*n*	*P*
Dryness of mouth	Amitriptyline	16	<0.01	18	<0.02	15	<0.01
	Fluoxetine	4		7		6	
Nausea	Amitriptyline	3	<0.09	2	<0.07	2	<0.01
	Fluoxetine	8		7		11	
Orthostatic hypotension	Amitriptyline	3	NS	10	<0.01	4	NS
	Fluoxetine	5		1		2	
Headache	Antitriptypline	0	<0.05	2	NS	2	NS
	Fluoxetine	4		2		2	

Discussion

This trial was conducted in a busy out-patient department. Patients were referred by general practitioners or other consultants, and were usually already taking benzodiazepines to allay anxiety or to induce sleep, at the time their primary depressive illness was diagnosed. Current clinical practice is not to attempt to wean such patients from their benzodiazepine medication, because of the subsequent recrudescence of anxiety, rebound of anxiety, or even withdrawal problems. Instead, the existing medication was standardised, diazepam being established as the daytime anxiolytic and temazepam as the hypnotic, and the antidepressant added. It is noteworthy that there were no differences in numbers of patients receiving benzodiazepines, nor in average doses attained, despite amitriptyline's marked sedative properties. The benzodiazepine dosages in both groups were modest.

The patients met RDC criteria, and were typical of psychiatric department out-patients. Almost all had been ill for more than 6 months, but the initial HRSD scores were only around 26, suggesting persistent illnesses of moderate severity. A third of the patients had previously failed to respond to tricyclic antidepressants, but often the dosage had been low. In the present trial amitriptyline dosage averaged 122 mg by week 6 (a typical UK dosage), and fluoxetine averaged 73 mg, in agreement with most previous studies (e.g. Chouinard, 1985). Feighner (1985), however, used higher doses of amitriptyline (145–173 mg/day) and lower ones of fluoxetine (51–59 mg/day).

Efficacy was similar in the two groups, with the exception of the BSD, on which fluoxetine proved just significantly inferior to amitriptyline. However, analysis of the 'pure' depressive items showed no drug differences. Chouinard (1985) reported an identical finding with the Zung depression scale, and suggested that the sedative effects of amitriptyline may have been misperceived by the patients as therapeutic, whereas psychiatrists' assessments were relatively unbiased. However, most of our patients received sedatives as well, and the differences could therefore not be due to lack of sedation in the fluoxetine patients. In another trial (Feighner, 1985), self-ratings were more favourable for fluoxetine. Overall, both drugs proved to be effective antidepressants over the 6-week trial, with end-point values similar to most other efficacy trials (e.g. Stark & Hardison, 1985; Feighner, 1985).

The apparent efficacy of fluoxetine has some interesting implications for biochemical hypotheses of antidepressant actions. Fluoxetine has no effect on down-regulating noradrenergic or 5HT receptor function (Peroutka & Snyder, 1980), generally held to be related in some way to long-term antidepressant effects (Willner, 1985). Nor does it have the typical behavioural pattern in animals of an antidepressant (Stark *et al*, 1985).

The most obvious differences between the drugs concerned appetite and weight, which increased in the amitriptyline and decreased in the fluoxetine patients. In general, depressed patients lose appetite and weight, and treatment-related increases are to be welcomed. However, some patients increase their weight when depressed, and others complain that tricyclic drugs make them obese; in both these groups fluoxetine might have a particular utility. These effects of fluoxetine accord with the known functions of 5HT in the brain (Lemberger *et al*, 1985).

The assessment of side-effects in clinical trials is controversial, particularly with depressed patients who typically have many somatic symptoms related to the illness. No amitriptyline and only two fluoxetine patients dropped out because of side-effects. This contrasts with the Feighner (1985) trial, where the drop-out on amitriptyline was high, presumably because of the high dose. In the study we concentrated on symptoms which were significantly treatment-emergent. Amitriptyline patients complained of dry mouth throughout, and of dizziness on standing at the 3-week point. Nausea was the cardinal side-effect in the fluoxetine patients, affecting nearly half by week 6. This is the commonest side-effect generally reported (Wernicke, 1985).

In practical clinical terms both drugs proved equally efficacious, most patients showing moderate clinical improvement. For ethical reasons a placebo group could not be included, so the absolute usefulness of both drugs can only be assumed. Side-effects were less troublesome in the fluoxetine patients, but they did not rate themselves as improved as the other group. The curious discrepancy between observer and self-ratings needs more detailed evaluation.

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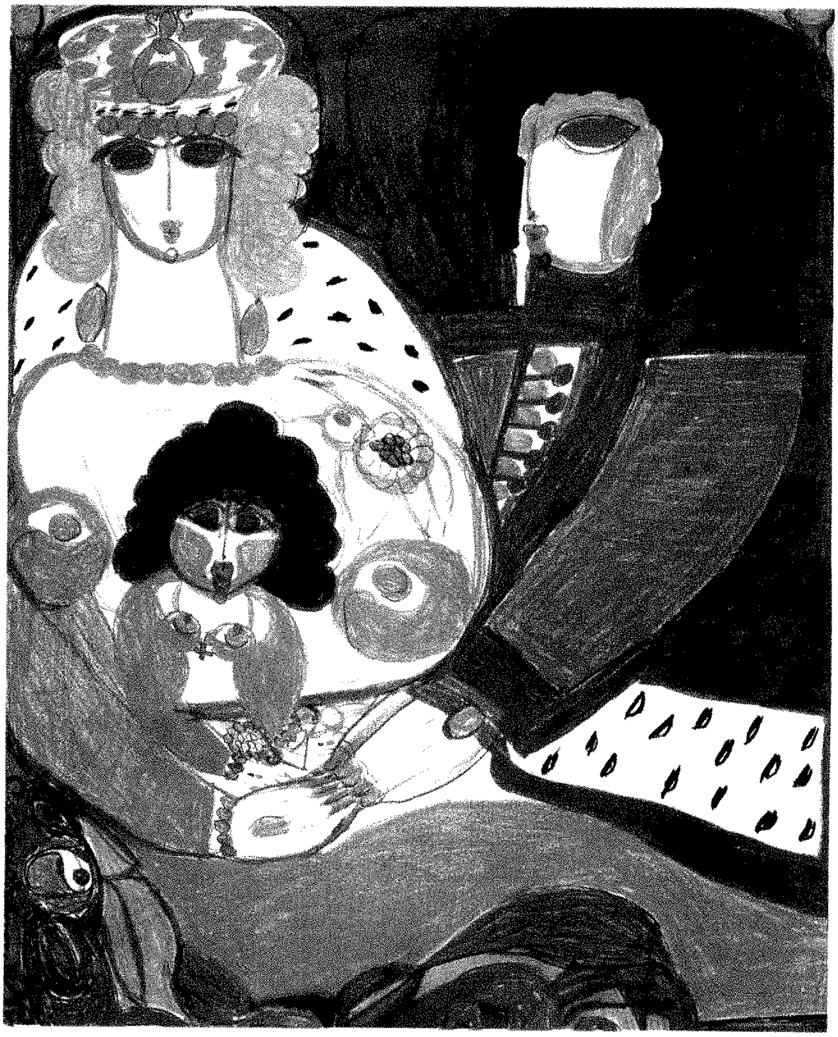

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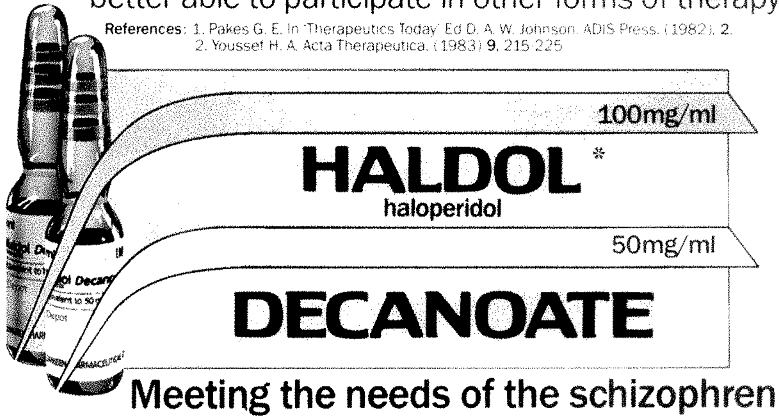

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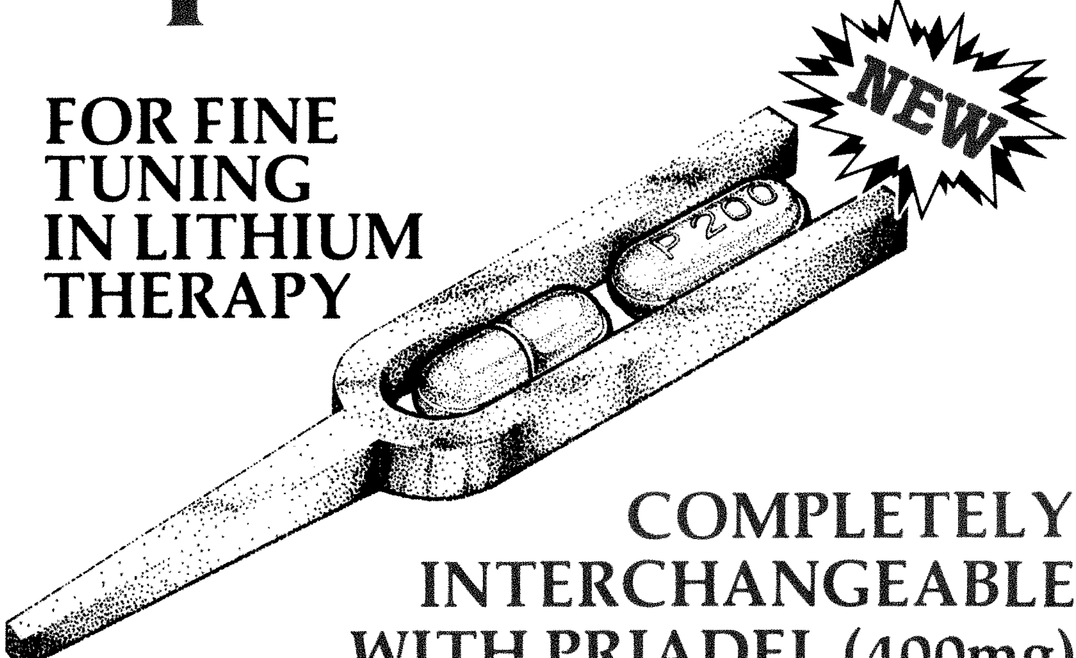

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British Journal of Psychiatry (1987), **151**, 341–346

The Social Breakdown Syndrome in the Elderly Population Living in the Community: The Helping Study

T. S. RADEBAUGH, F. J. HOOPER and E. M. GRUENBERG

A representative sample of elderly people residing in the community was examined to establish their psychiatric status. An interview with a close friend or relative, focusing on a one-week period in 1981, was used to investigate each subject's functional limitations and troublesome behaviour, these being the two components of the Social Breakdown Syndrome. The data from the sample were weighted to allow estimates of the characteristics of the general population. No cases of SBS at its most extreme were identified, and almost the entire population was found to be functioning at an adequate or near-adequate level: all cases of severe SBS were attributable to troublesome behaviour. Severe SBS was shown to increase with age and to be most common in non-white males. Persons with dementing disorders were more likely than their non-demented counterparts to show severe/moderate SBS, but in the majority of cases of SBS there was no mental disorder.

The 'greying of America' is well under way, and the demographic changes which the USA will undergo in the next 20 years have been projected and described thoroughly by the Bureau of the Census (1977). The provision of medical care, health care and day-to-day care for this growing legion of elderly people is of increasing importance.

The scientific literature about elderly people is already massive, but there have been two important omissions from the research work: the first has been lack of attention to mental and neurological disorders and the consequences of such disorders; the second has been the failure to investigate populations of elderly persons living in the community. Much of the available information has been derived from persons seeking medical or social services: these are not necessarily representative of the elderly population as a whole.

Most elderly people do reside in the community: only 5% of those aged 65 or more reside in some type of long-term-care institution, although it is important to note this figure conceals extraordinary biases in colour, sex, and marital status. Most of the day-to-day care of ill and frail elderly people is thus provided not by organised medical, nursing or social services but by friends, relatives and neighbours who are often elderly themselves.

Method

Eastern Baltimore Mental Health Survey

The Eastern Baltimore Mental Health Survey (EBMHS) is attempting to fill some of the gaps in our knowledge about the life circumstances and day-to-day management of elderly people living in the community. It is an epidemiological field survey of the prevalence (Wave I) and incidence (Wave II) of mental disorders, and the patterns of health care and mental health care, in a geographically defined population, namely the residents of the eastern one-third of Baltimore City (Eaton *et al*, 1981).

For the EBMHS a representative sample of households was identified through a probability sampling plan, and all members of these households aged 18 or over were asked to participate in a lengthy structured interview containing the NIMH Diagnostic Interview Schedule (DIS). To screen for cognitive deterioration, the Mini-Mental State Examination (Folstein *et al*, 1975) was also included in the household interview, which was completed by 3481 people.

Every individual who scored poorly on the Mini-Mental State Examination or who responded in a particular (pre-specified) way to one or more key questions on the DIS (thus indicating possible mental disorder or cognitive deterioration) was offered an examination by a research psychiatrist. In addition, a randomly selected 17% of those who gave no such response suggestive of mental disorder or cognitive deterioration were invited to participate in either the *Standardized Psychiatric Examination* of Romanoski & Chahal (1981, unpublished) or a Clinical Reappraisal (Folstein *et al*, 1985*b*) which yielded diagnoses of mental disorder using DSM-III criteria. Table I contains a summary of responses to the various stages of the survey among those aged 65 or over, analysed according to sex and colour.

The Helping Study

Upon completion of the clinical reappraisal, individuals aged 65 or over ($n = 228$) were invited to participate in the 'Helping Study'. This required them to identify an informant, i.e. one of the people who were most familiar

TABLE I

Eastern Baltimore Mental Health Survey, Wave I (prevalence study): response rates for persons aged 65 or over, classified by sex and colour

	Whites		*Non-whites*		
	Males	*Females*	*Males*	*Females*	*Total*
Selected for household survey	386	618	93	145	1242
Completed household survey	274 (71%)	439 (71%)	77 (83%)	133 (92%)	923 (74%)
Offered clinical reappraisal	87	144	44	64	339
Completed clinical reappraisal	59 (68%)	87 (60%)	37 (84%)	45 (70%)	228 (67%)
Invited to join Helping Study	59	87	37	45	228
Completed Helping Study	53 (90%)	79 (91%)	32 (86%)	38 (84%)	202 (89%)
Total population (estimated)	8769	14 122	2171	2689	27 751

with their day-to-day activities, who would agree to an interview. Informants were used to avoid relying on subjects with cognitive impairment for reports of their day-to-day affairs. However, some subjects served as their own informants: these people were either so isolated that no other individual knew the intricacies of their daily lives, or they were very concerned with privacy and did not want anyone to talk to an interviewer about them. There were 48 such cases.

The interview was of the non-schedule standardised type (Richardson *et al*, 1965), and averaged about 90 minutes in duration.

The Social Breakdown Syndrome

One of the principal questions we wished to answer for each subject in the Helping Study (with reference to a long list of activities necessary for living in the community) was:

> *"During the seven days prior to the interview, what did the subject do for him/herself?"*

The standard interview used with the informant to obtain an answer to this question is firmly rooted in the original work on the Social Breakdown Syndrome (Gruenberg *et al*, 1966). The Social Breakdown Syndrome (SBS) is characterised by a failure of social and personal care. It is thought to occur independently of specific psychiatric diagnoses (American Public Health Association, 1961) and is observed in a wide variety of illnesses and conditions. In fact, the SBS corresponds closely to the concept of 'disability' employed by the World Health Organization:

> In the context of health experience, a disability is any restriction or lack (resulting from an impairment) of ability to perform an activity in the manner or within the range considered normal for a human being . . . Disability is concerned with abilities, in the form of composite activities and behaviours, that are generally accepted as essential components of everyday life. Examples include disturbances in behaving in an appropriate manner, in personal care (such as excretory control and the ability to wash and feed oneself), in the performance of other activities of daily living, and in locomotor activities (such as the ability to walk). *(World Health Organization, 1980)*

The SBS has two dimensions: functional level (FL) and troublesome behaviour (TB) (Gruenberg *et al*, 1966). The *functional level* is defined on the basis of useful contributions of work around the house or yard, productive hobbies, recreation, reading or writing, and being able to be left alone. Subjects' *behaviour* was classed as *troublesome* if they required physical restraint; or needed assistance to eat, or to get in and out of bed; if they were incontinent or used a bedpan; if they attacked other people or made threats towards them; if they harmed themselves; or if they were mute or refused to speak. The Appendix describes in detail our classification of functional levels and degrees of troublesome behaviour.

The Helping Study data record was constructed on the general pattern of that used in the original work on SBS, but it was not an exact replica; e.g. it did not incorporate specific durations for the behaviour reported. The original SBS analysis revealed that people were rarely left alone for very short periods, so the SBS criteria for "was S left alone?" required that the subject was left alone for at least three hours. Thus the Helping Study data-recording procedures necessitated assuming that, if the subject was left alone, he or she was left alone for at least three hours.

Subjects

In summary: 228 individuals were invited to participate in the Helping Study, and an interview with the subject or an informant was obtained for 202 subjects. This sample differed from the general population aged 65 or more in Eastern Baltimore ($n = 277\ 751$) in terms of sex-, colour- and age-distribution; but knowing the response rate in each demographic sub-group (Table I) allowed us to weight the data from the sample to make it representative. Our sample also contained a disproportionate excess of individuals with a diagnosed mental disorder, due to our selection procedure, and we also weighted to compensate for this effect. Thus each of our subjects effectively represented a known but

TABLE II

Helping Study sample analysed (a) by sex and race, (b) by age, and (c) by diagnostic category; plus a projection of this analysis on to the elderly population of Eastern Baltimore

	Sample $n = 202$	*Population (estimated)* $n = 27751$
Sex and colour:		
White males	53 (26.2%)	8769 (31.6%)
White females	79 (39.1%)	14 121 (50.9%)
Non-white males	32 (15.8%)	2173 (7.8%)
Non-white females	38 (18.8%)	2689 (9.7%)
Age distribution:		
65–74 years	118 (58.4%)	17 154 (61.8%)
75–84 years	72 (35.7%)	8562 (30.9%)
85 years and over	12 (5.9%)	2035 (7.3%)
Diagnostic categories:		
Dementia	35 (17.3%)	2441 (8.8%)
Other mental disorders	59 (29.2%)	6270 (22.6%)
No mental disorder	108 (53.5%)	19 040 (68.6%)

TABLE III

Estimated population distribution of the two components of the social breakdown syndrome (SBS)

	Troublesome Behaviour			
Functional level	*Very*	*Moderate*	*Not*	*Total*
Low	0[d]	0[d]	0	0
Minimal	0[d]	48[c]	0	48
Almost adequate	108[c]	2490[c]	3419[b]	6017
Adequate	2712[c]	2832[b]	16 142[a]	21 686
Total	2820	5370	19 561	27 751

a = No SBS ($n = 16142$)
b = Moderate SBS ($n = 6251$)
c = Severe SBS ($n = 5358$)
d = Extreme SBS ($n = 0$)

unique number of individuals in the general population, allowing us to estimate the prevalence of psychiatric disorders and of the Social Breakdown Syndrome in the elderly population of the community.

Results

As Table II shows, our sample of 202 subjects was 42% male and 58% female (not importantly different from the estimated population). Non-whites accounted for 35% of the sample compared with 17.5% of the general population.

An examination of the age-distribution of the study sample compared with our estimates of the population did not reveal marked differences (Table II), but very few subjects over the age of 85 participated in any phase of the EBMHS, thereby limiting the statistical reliability of results. However, no better data have yet been published.

Each of the 202 subjects was assigned to one of three diagnostic categories (dementia/other mental disorder/no mental disorder) on the basis of information from the clinical reappraisal, except when he/she underwent a medical examination for the differential diagnosis of a dementia (suspected at clinical reappraisal), when the resulting diagnosis was used. Subjects with both dementia and another mental disorder were assigned to the 'dementia' category. The category 'other mental disorders' was composed primarily of alcohol problems, affective disorders, phobic disorders and personality disorders, occurring singly or in combination. The category 'no mental disorder' included persons with tobacco use disorder, uncomplicated bereavement and minor sexual disorders only. For a more complete description of the diagnostic process see Eaton *et al* (1981) and other papers still in preparation. Table II presents the distribution of diagnoses for the study sample and the estimated distribution in the population. The differences between the sample and the estimated population distribution are directly attributable to the EBMHS study design.

Our estimates of the population distribution of the two components of the Social Breakdown Syndrome (functional level and troublesome behaviour) are displayed in Table III. The present study did not identify any cases of the most extreme levels of SBS, and only a very small proportion of the total estimated population demonstrated minimal functioning with moderate troublesomeness. Most importantly, it appeared that almost the entire population functioned at an adequate or almost adequate level: all cases of severe SBS were attributable to moderately troublesome or very troublesome behaviour. Table IV displays the frequency of different levels of Social Breakdown Syndrome in the study sample and the estimated frequency in the general population.

Age is an important factor in the prevalence of Social Breakdown Syndrome and dementia. The estimated prevalence in different age-groups of SBS with and without dementia is shown in Table V. The prevalence of severe SBS is highest (41%) among those aged 85 or over. Severe SBS combined with dementia is also most frequent in the oldest age-group. However, the prevalence of severe SBS *without* dementia does not appear to increase dramatically with age.

Moderate SBS is most common in the 65–74 age-group, in which it is rarely associated with dementia. In fact, only a small fraction of the total burden of SBS in the community is associated with dementing disorders *except in the very old* (85 and over).

It is not surprising that dementia occurs without SBS in some cases, even among the oldest. This cross-sectional picture may capture developmentally differing stages of the condition.

Table V also displays the prevalence among males and females, white and non-white, of Social Breakdown Syndrome with and without dementia, and reveals extraordinary differences between sub-groups. Severe SBS is almost three times as common among non-white males as among white males, but this dissimilarity is not observed

TABLE IV

Sample distribution and estimated population distribution of different levels of the Social Breakdown Syndrome (SBS)

Degree of SBS	*Sample (n = 202)*	*Estimated population (n = 27 751)*
Severe	42 (20.8%)	5358 (19.3%)
Moderate	62 (30.7%)	6251 (22.5%)
None	98 (48.5%)	16 142 (58.2%)

among females. Moderate SBS is far more common in white males than in white females.

While almost two-thirds of white females in the estimated population display neither SBS nor dementia, only one-third of the non-white males are free of both conditions. However, since white females comprise an estimated 51% of the population and non-white males only 8%, the former group imposes the largest absolute burden on society in terms of SBS.

Diagnoses of mental disorder diagnoses and levels of Social Breakdown Syndrome were compared with the number of physical health problems reported by the informants. The inquiry about physical health was based on a short list of illnesses and conditions, including cancer, heart disease, high blood pressure, stroke and diabetes: Table VI should therefore be viewed with great caution because of the restricted list of health problems, the use of informants for such an inquiry, and the absence of any examination of the subject. We estimate that the majority of the population have one or more physical health problems.

Discussion

Although this study is unique in being population-based and using a standardised psychiatric examination, it must be re-emphasised that the sample is small. However, these data are the best presently available on the specific issues considered.

One of the striking findings in this cross-sectional study was that we found no examples of Social Breakdown Syndrome at its extreme (a combination of low functional level and very troublesome behaviour). There are several possible explanations:

1. Disability of such extremes may not be tolerated by helping networks in the community
2. Disability of such extremes may occur very infrequently in the community; it may be, in fact, primarily the product of institutional settings
3. The subjects with the most limited and troublesome behaviour may have dropped out before referral to The Helping Study, or refused to cooperate
4. Our informants may have failed to report the actual extent of the most embarrassing and disagreeable behaviour of their loved ones.

It is likely that each explanation is partially correct. For example, the interviewers and the first author contacted and attempted to enlist into the study a particular married couple: on the basis of a brief meeting and a visual inspection of their living

TABLE VI

Estimated prevalence of physical health problems among the elderly population with different degrees of the Social Breakdown Syndrome

Number of physical health problems	*Degree of SBS*		
	Nil	*Moderate*	*Severe*
None	4%	20%	30%
1–3	86%	65%	40%
4 or more	10%	15%	30%
	100%	*100%*	*100%*

TABLE V

Estimated population distribution of different degrees of SBS with/without dementia: (a) in different age-groups; (b) in males and females of different ethnic groups

	Age-group			*Whites*		*Non-whites*	
Degree of SBS ± dementia	*65–74 yrs (n = 17 154)*	*75–84 yrs (n = 8562)*	*85 yrs & over (n = 2035)*	*Males (n = 8769)*	*Females (n = 14 121)*	*Males (n = 2173)*	*Females (n = 2689)*
Severe SBS:	19%	16%	41%	11.4%	22.1%	32.8%	19.6%
Not demented	17%	14%	21%	7%	21%	19%	14%
Demented	2%	2%	20%	4%	1%	14%	5%
Moderate SBS:	26%	20%	7%	34.3%	13.5%	25.8%	28.6%
Not demented	25%	14%	0%	32%	10%	19%	25%
Demented	1%	6%	7%	2%	3%	7%	3%
No SBS:	56%	65%	52%	54.2%	64.4%	41.2%	51.8%
Not demented	55%	60%	45%	53%	62%	33%	48%
Demented	1%	5%	7%	1%	2%	9%	4%

quarters it appeared that they probably represented the 'Diogenes Syndrome' discussed by Clark & Mankikar (1975). Their home was filthy, rubbish-filled and fly-infested. The couple were unco-operative.

Our investigation points to a multiplicity of origins for the Social Breakdown Syndrome. It may be the net result of dementing disorders, other kinds of mental disorders, chronic and debilitating physical conditions, and perhaps particular personality configurations of hostility and withdrawal, and the interactions of these conditions and afflictions. However, the bulk of the community burden of Social Breakdown Syndrome is *not* associated with dementing disorders (only 15.2% of those with moderate or severe SBS were demented) or with other disorders, since 67.8% of those with moderate/severe SBS had no mental disorder (compared with 68.6% in the general population over 65). In fact, the proportion of persons with mental disorders other than dementia was lower among those with moderate/severe SBS (17%) than in the general population over 65 (22.6%). However, given that dementia was found in only 8.8% of the general population aged 65 or over but in 15.2% of those with moderate/severe SBS, it is clear that persons with dementing disorders do in fact contribute disproportionately to the community burden of functional failure and troublesome behaviour.

Both advanced age and physical health problems appear to be associated with SBS, but it seems quite possible that it is not age *per se* that is important in SBS but that age is a proxy variable for some unknown underlying diseases or conditions.

It appears paradoxical that 30% of those with severe SBS had no physical health problems. This group may have been comprised of persons with dementia or other mental disorders. Alternatively, this finding may be an artefact of our questions and methods and may in fact be misleading: other authors (Clark & Mankikar, 1975; Macmillan & Shaw, 1966) have noted that medical problems are frequent and important in self-care failures by elderly people. The Helping Study was not designed to collect detailed information about the role of physical health problems in the Social Breakdown Syndrome.

The differences in prevalence of moderate and severe SBS in whites and non-whites, males and females, are, at first glance, impressive. Non-white males appear to be most affected by SBS. However, these rates are estimates compiled from reports by informants; furthermore, family structures and living arrangements may explain part of the observed variations. For instance, older non-white men may tend to remain in the community rather than go into care because of the availability of extended family or protective living arrangements; alternatively, the barriers to admission to long-term care facilities may be slightly different for the various sex/colour sub-groups. Longitudinal follow-up studies and investigation of the frequency of SBS in patients admitted to long-term-care facilities are required to assist in unravelling the meaning of the observed differences in prevalence between sub-groups.

Conclusions

There have been many reports by clinical investigators of the enormous burden on families who care for the demented elderly. The implication has been that much of the community burden of care is attributable to dementing disorders. What has been demonstrated in this study is that most of the functional limitations and troublesome behaviour of elderly people in the community are unrelated to mental disorders. Analysis of Wave II of the Helping Study (12-month incidence data) may reveal other risk factors for the development, progression and resolution of the Social Breakdown Syndrome.

Nonetheless, persons with dementing disorders contribute disproportionately to the community burden of disability, demonstrating the epidemiological principle that observations in clinical settings may represent only the tip of an iceberg of unknown shape and size. This is a point that those delivering services to families of the elderly or planning services for the elderly should heed.

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Appendix

The Social Breakdown Syndrome (SBS) has two components; poor functioning and troublesome behaviour. For our investigation of the SBS, we defined four levels of functioning and three categories of troublesome behaviour. The four levels of functioning were: *low/minimal/almost adequate/adequate*.

To be rated as *'low functional level'* the subject had to satisfy *all* the following negative criteria:

1. Subject did *no work* (i.e. no paid work; or work around the house or yard; or cleaning his/her room; or fixing things around the house; or doing the laundry)
2. Subject had *no productive hobbies* (gardening, sewing, knitting, woodwork, modelling)

3. Subject engaged in *no recreation* (visiting friends/relatives; going to church; going to sports events or clubs; using the telephone; watching TV; going to the movies; other recreations)
4. Subject *did not read or write*
5. Subject was *never left alone*

A subject meeting four of these five negative criteria was rated as *'almost adequate functional level'*.

To be rated as *'adequate functional level'*, the subject had to meet three criteria:

1. Subject did *some work* (as previously defined) OR engaged in a *productive hobby*
2. Subject engaged in *active recreation* OR *read or wrote*
3. Subject was *sometimes left alone*

The category *'minimal functional level'* was a residual category.

Behaviour was rated as *very troublesome/moderately troublesome/not troublesome*. It was rated as *'very troublesome'* if the subject fulfilled *any* of the following criteria:

1. Subject had to be physically restrained
2. Subject had to be urged and helped to eat
3. Subject needed help to get in and out of bed
4. Subject was incontinent
5. Subject was mute (did not speak for an entire day)
6. Subject harmed himself/herself or someone else, or self-injury had to be forcibly prevented

Behaviour was rated as *'moderately troublesome'* if *any* of the following criteria were met:

1. Subject had to be told to stay in one place, or needed restraining but was not restrained
2. Subject had to be helped to eat
3. Subject toileted in the wrong place, or needed help toileting at night
4. Subject spoke only when spoken to
5. Subject wanted to harm himself/herself but did not

Subjects whose behaviour did not satisfy any of the above criteria were classified as *'not troublesome'*.

The way in which different levels of functioning and different degrees of troublesome behaviour were considered together to give an overall rating of SBS (*nil/moderate/severe/extreme*) is indicated in Table III. Thus a subject functioning at an adequate level and exhibiting no troublesome behaviour was regarded as not suffering from SBS; moderate SBS corresponded to a combination of *either* adequate function with moderately troublesome behaviour *or* non-troublesome behaviour with 'almost adequate' functioning; and so on.

The following fictional examples indicate the types of problems and behaviour corresponding to different levels of functioning and different degrees of troublesome behaviour.

Example of adequate functioning

Mr J. is an 82-year-old man who cares for his demented 86-year-old sister. During the last week he has replaced a screen in the front door, cleaned the house a bit, tended his flower garden, gone to visit a neighbour, read the newspaper, and spent half of Saturday by himself while his niece took the demented sister to a dental appointment.

Example of low functional level

Miss J. is unable to do anything around the house or yard. She passes her days sitting on the couch and staring out of the window at passersby on the street. She is never left alone in the house.

Example of very troublesome behaviour

Mr S. is 72 years old and has no mental disorder. He is arthritic, almost blind, and has an amputation above the left knee. He has to be helped in and out of bed, cannot feed himself, and usually requires a bedpan twice during the night.

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Quality of Life for 'New' Long-stay Psychiatric In-patients

The Effects of Moving to a Hostel

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We observed 15 'new' long-stay patients in wards in a District General Hospital Unit and a mental hospital, and again when they had lived for a year in a new hospital-hostel. There were significant changes in time spent in the community, social interaction, activity and abnormal behaviour. Residents believed they had more freedom and none wanted to return to wards. Six patients remained on wards. They showed no comparable changes and all wanted to live elsewhere.

It is now clear that a small number of adult psychiatric patients admitted to hospital will remain there for long periods, in spite of active treatment and rehabilitation. In England, for every 100 000 of the general population aged 15–64, between 24 and 36 adults in 1979 were likely to be newly recruited long-stay psychiatric patients (Jennings, 1983). A national survey of such patients found that most could not look after themselves, and were not acceptable to local authorities or voluntary bodies providing accommodation, because of their multiple handicaps and high levels of psychotic and difficult behaviour (Mann & Cree, 1976). These patients are not a homogeneous group: in this paper we are concerned only with those under 65, who were admitted to hospital from one health district between 1979 and 1983, stayed for at least a year, and did not have multiple handicaps that required care in psychogeriatric or other special units.

Such patients are now cared for in long-stay mental hospital wards; or in District General Hospital (DGH) units; or in new hospital–hostels, which are intended to provide continuing treatment and 24-hour nursing care, but in a more domestic and less institutional setting (DHSS, 1975). Evaluative studies suggest that patients living in hospital–hostels show more behavioural improvement than patients living in hospital wards (Wykes, 1982; Gibbons, 1986). But the new hospital–hostels are also intended to provide a better quality of life than can be offered by either the old long-stay wards or the new DGH units. This paper evaluates the extent to which patients' quality of life improved when they moved from hospital wards to a new hospital–hostel.

Measurement of environment

In the pioneering study by Townsend (1962) of old people's homes, a series of items describing such features as the building, access to possessions and staff attitudes made up a quantitative scale which could be used to evaluate and compare quality of care in different institutions. Jones & Fowles (1984) point out

> the difficulty, as with all such scales, is that it is necessary to defend both the choice of items and the weighting accorded to them; and when this is done what appears to be an objective and reliable and reliable measurement often turns out to be both subjective and unreliable.

Moos (1974), within a theoretical framework of social ecology, developed a Ward Atmosphere Scale to measure perceived organisational climate. The numerous items had to be rated independently by staff and patients, and compliance was low on chronic wards. The scales were not designed to measure patients' quality of life. For these reasons the WAS was not suitable for our purposes.

The more management practices in institutions are characterised by rigid routines, regimentation of individuals into groups and social distance between staff and inmates, the more an institution approaches the 'total' model of Goffman (1961). King *et al* (1971), in a study of management practices in residential institutions for children, assumed that the less 'institution-orientated' regimes were the more desirable. Although they did not have independent measures of outcome, 'restrictive' management practices have been associated with undesirable outcomes (Apte, 1968). Restrictiveness, as measured by the Hospital/Hostel Practices Profile, was much more marked in hospital wards than in a new hospital–hostel (Wykes, 1982). Hewett (1979) criticised this measure as an index of quality of life, suggesting that many of its so-called 'restrictive' practices were in fact 'caring' and necessarily protective in relation to the needs of severely handicapped residents. The measure had the further disadvantage, for our purposes, that it was derived entirely from the perceptions of a senior staff member.

The principle of *normalisation* – 'making available to the mentally retarded patterns and conditions of everyday life which are as close as possible to the norms and patterns of the mainstream of society' (Nirje, 1967) – also appeared relevant to our attempt to assess the experiences of mentally ill people in different residential environments. Normalisation does not imply 'dumping people into communities from institutions without support', but rather the creation of 'highly specialised support services and clinical/technical expertise which can be used to help people achieve greater degrees of independence.' In judging the quality of a particular residential service, account is taken of how far its regime conforms to the principle of normalisation in a number of specific areas (Pieper & Cappuccilli, 1980).

We wished to describe the impact of different regimes upon residents and obtain their own views of their experience. A recent survey of Los Angeles board-and-care homes showed that three-quarters of mentally disabled respondents were able to take part in structured interviews about their quality of life (Lehman, 1983). This contrasted with the difficulty residents in the Maudsley hospital–hostel had had in answering simple yes/no questions. We therefore decided to gather data by a combination of direct observation and semi-structured interviews with residents. We assessed their quality of life on hospital wards and after moving to a hospital–hostel along the following dimensions:

(a) integration: nature and site of building; amount of time spent in community; contact with relatives and friends
(b) living situation: space; privacy and quiet; security; freedom; social relations
(c) finances: weekly income
(d) activity: time spent on work activity; time spent doing nothing; inappropriate behaviour
(e) satisfaction with accommodation.

The settings

The three residential treatment settings are described below.

The first setting was long-stay wards in the mental hospital (MH wards). The mental hospital dated from the nineteenth century and stood in spacious grounds 12 miles from the city. The long-stay wards to its rear were typically institutional in design. When the research began in 1983 there were two high-dependency single-sex wards with a ratio of two nurses to every three patients, the remaining wards being mixed-sex, with lower staff-patient ratios. By 1985 the hospital had changed. It was scheduled to close in a decade; many 'old' long-stay patients had been moved out to low-dependency hostels or private care, and the remaining wards reorganised with more rehabilitative aims. There were less than two nurses to every three patients.

The second setting was a rehabilitation ward on an upper floor in the DGH unit. The unit opened in 1979 and took all admissions from the catchment area. It was a purpose-built 95-bedded unit next to a general hospital and near the city centre, in an area with a reputation for social problems. It had no grounds and space was at a premium. The rehabilitation ward provided a 3-month programme for most of its 20 inmates, the rest following a slower pace; all, however, had some planned programme. There were 18 nursing staff for the 20 patients, and higher medical coverage than in the MH wards.

The third setting was the new hospital–hostel opened in January 1984 to provide for the district's long-stay. It was in two converted, linked houses on four floors, approximately 5 minutes' walk from the DGH unit across a busy main road. It had 20 places, with 20 nursing staff. Medical cover was provided by a consultant and senior registrar, and there was a part-time psychologist and occupational therapists. The staffing ratio was only slightly better than in the DGH ward but very much better than the MH wards, and was approximately twice that recommended for the highest dependency group (the elderly mentally infirm) in voluntary residential care by a DHSS working party (Avebury, 1984). The building was not marked as an institution and did not stand out from the neighbouring small hotels and blocks of flats. It shared with the DGH ward the advantages and disadvantages of its position near the city centre. As in that ward, the aims were rehabilitative, though the pace was slower and it was recognised that many residents would never live independently. Every resident had a nursing plan, in some cases fairly precise, specifying goals and tasks, but in other cases merely prescribing a general nursing regime. The hostel staff consciously tried to create a warm, informal climate and to avoid institutional routines and foster independence to a greater extent than was possible in the hospital wards. The hostel also differed from the DGH ward in its domestic scale and furnishings and the additional space provided by a large garden.

TABLE I
Patients admitted 1979–1983 who remained in hospital for at least a year: status at September 1985

					Hospital first diagnosis		
	n	*Mean age*	*Men: n*	*Mean psych. adm.*	*Schizophrenic*	*Organic*	*Other*
Hospital–hostel	15	46.8	10	9.5	9	0	6
Rejected: in wards	6	44.3	3	7.5	3	2	1
Ineligible: in special units	4	46.7	3	1.5	0	4	0
Discharged	12	35.8	5	8.8	6	0	6
Dead	5	56.6	4	13.0	1	2	2
Total	42	44.5	25	8.7	19	8	15

The patients

The sample consisted of all residents who had lived in the new hospital–hostel for a mean of 15 months (11–18) by August 1985. There were 15. All had been studied previously, when they had been continuously on hospital wards for a year. Table I shows their characteristics in comparison with those of all the patients under 65 admitted from the catchment area 1979–1983, and staying in hospital for over a year. Of the 42 identified cases, 12 had been discharged before August 1985 (two from the hostel to less sheltered settings) and five had died. Four cases were excluded (primary organic conditions needing nursing in special units). Thus 21 cases were 'eligible' for the hostel, of whom 15 had been accepted into it. Eleven of these had moved to the hostel from the DGH ward and four from the MH wards.

Method

We used the Behaviour Observation Instrument (Alevizos *et al*, 1978) to study sample patients on the wards and after they had lived in the hostel for 11–18 months (residents moved in at different times). During July 1983 and 1984 and August 1985 we observed in daily shifts from Monday to Saturday. A shift was 09:00 to 13:00; 13:00 to 17:00; or 17:00 to 21:00. Within the shift we observed all patients at approximately hourly intervals in a random order. The observer needed a brief training to become familiar with the instrument's categories. She had to watch the patient unobtrusively for 5 s and then withdraw to code the observed behaviour on to a schedule, with a brief description. When subjects could not be found on hospital property the observer tracked them to local destinations, such as a cafe, shop or the League of Friends, where they could be observed unobtrusively from the street. When subjects were out on longer trips during observation periods, the observer established their whereabouts by questioning staff and checking with the subjects themselves on their return. When patients' whereabouts could not be established, or they were known to be in another hospital, observations for that period were omitted. In 1983 the authors carried out 19 observations on each patient independently but simultaneously. Inter-rater reliability was 97% and a similar level of agreement was reached in reliability exercises in 1984 and 1985.

For the analysis a notional weekday was created consisting of the 12 hours from 09:00 to 21:00. Three observations for each hour, taken on different days, were drawn, making 36 total observations during a weekday for each case. The same procedure was used for Saturday, except that the notional day lasted from 09:00 to 17:00. We also observed patients' material circumstances of life in wards and in the hostel.

In 1985, at the end of the research, 14 of the 15 patients completed a semi-structured interview about their own perceptions of quality of life. One patient could answer only a few questions. The questionnaire items were derived from areas considered important in the literature on normalisation and from preliminary unstructured observations made by the senior author. She visited the living room to engage residents in conversations about aspects of hostel life that were important to them. No pilot interviews were carried out. Sample questions are shown in Appendix 1.

Results

Integration

The building

The mental hospital was the least 'normalising' setting, clearly marked out as a segregated institution and without easy access to community resources. The DGH unit was in the city, giving easier access, but equally institutional in appearance and reputation. The hostel was the most normalising, sharing the DGH unit's easy access but without its distinguishing institutional symbols. However, with its 20 places it was too large for residents to be easily assimilated into a local community, and they were 'patients' in a treatment setting, without the rights of tenure enjoyed by other citizens.

Amount of time in the community

Table II shows that as patients had used space differently in the MH and DGH wards, their pattern of change on moving to the hotel also differed. Former MH patients spent significantly less time in occupational therapy and more time in the community after moving. Former DGH patients spent significantly less time in bed, less time in corridors and

Table II

Use of space in wards and hostel: weekdays

	Patients moving from MH to hostel (4)		*Patients moving from DGH to hostel (11)*	
	MH	*Hostel*	*DGH*	*Hostel*
Bed: % of time	3	8	23	12***
Occupational therapy: % of time	23	1***	3	0
Kitchen: % of time	1	4	0	3
Other rooms: % of time	51	53	35	59***
Public space: % of time	13	8	27	10***
Grounds: % of time	8	7	0	2
In community: % of time	1	19***	12	14
Total observations	136	144	369	381

χ^2 test ***$P<0.001$.

hallways and more time in living rooms. They increased the proportion of time they spent in the community, but not to a significant extent: they had already been spending greater periods of time outside hospital property when they were in the DGH unit.

On Saturdays MH patients went out more (18% of the time) and increased this further, though not to a significant extent, after they moved to the hostel. In the DGH unit and the hostel the use of space was much the same on Saturdays as it was on weekdays. In the mental hospital distance, expense and the structured weekday apparently combined to prevent short trips out from hospital property, so there was a dramatic increase in time spent in the community after the move to the hostel.

We examined the extent to which patients' time in the community was organised by others: by the hospital in the form of organised recreation or trips with a member of staff; and by relatives in the form of arranged home visits. Patients in the mental hospital made no trips into the community on their own initiative, but when they were in the hostel 86% of their time outside was self-initiated. In the DGH unit patients were able to go into the community of their own accord, and they maintained much the same pattern in the hostel.

Hostel residents went out to obtain goods and services that previously had been available within the hospital. Instead of going to the hospital shop or the leisure centre they went to the local newsagents, food shops and café. Some went out to the hairdresser instead of being visited by her in hospital. They went for walks in the town centre or on the local common and made use of the local cinema and pubs. Some residents visited friends, who were usually also former patients. There were occasional problems when hostel residents paid unplanned and unwanted visits home.

Altogether, hostel residents increased their time in the community from 9% to 15%. Nine of the 15 spent more time in the community.

Contact with relatives and friends

The move to the hostel made little difference to the pattern of contact with relatives. Four of the 15 had no contacts with any relatives, two expressing regret: 'My whole family have all gone away'. 'My sibling ones, my senior ones all gone away.' Seven exchanged visits with relatives at least weekly; the remainder saw relatives several times a month. Six out of 15 wanted to spend more time with relatives but most were satisfied. Seven out of 15 were visited by friends outside hospital and/or went to visit them. In most cases, but not always, these were friends made in hospital who were now in the community. The move to the hostel did not result in residents seeing more of their families, but at least two stopped absconding home, which had caused distress when they were in hospital.

Living situation

Space

Four residents had had small single rooms on the wards at baseline; the rest had been in dormitories. In the hostel only one had a single room, three were together and the rest in double rooms. The bedrooms were all large and pleasantly furnished. Only one person complained of insufficient space in the bedroom, but four thought the hostel's leisure space was insufficient, compared with the wards, or complained of competition for scarce space: 'The staff claim the green room so we cannot use it.' The hostel had a dining room, a small lounge/TV room and two other small leisure rooms as well as the garden. The basement space was unattractive to residents and seldom used. To an observer the leisure space appeared cramped and noisy when residents were together, although it was on a more domestic scale than in the wards.

Privacy and quiet

Only three residents complained of insufficient privacy in the hostel as compared to the wards. Most did not want to be alone: 'I don't see any point in being alone. It wouldn't be a good idea to be able to lock the bedroom door.' Residents had various strategies for finding privacy and quiet when they needed it, the commonest being to go to their rooms while their roomate was elsewhere or to go out: 'I like solitude. I find it on my bicycle.' Six mentioned that it was sometimes too noisy in the living room.

Security

All residents had lockable wardrobes and chests for their personal possessions, as they had had on the wards. The majority expressed no fears for their own safety or that of their belongings, but three mentioned violent episodes and five mentioned thefts or losses. In general residents felt safer in the hostel: 'I have a lock to my chest of drawers and I keep the key with me. At [mental hospital] the key would be taken from me if I fell asleep.'

Freedom

We examined restrictiveness in relation to rules about getting up and going to bed; access to kitchen and laundry facilities and leaving the building. On the wards there were rules about getting up and going to bed, which were operated more firmly in the mental hospital. In the hostel 14 out of 15 believed they could get up and go to bed when they liked: 'Sometimes I feel so tired I stay in bed all day.' In MH wards laundry was done centrally, but patients had had access to a washing machine in the DGH ward and most had done some of their own washing. In the hostel all residents had free access to the laundry room. Five managed all their own washing, the rest getting some assistance from staff. On the wards none had free access to the kitchen. In the hostel all residents breakfasted at their own time in the kitchen, washing up their own things. But residents disagreed over whether there was free access to the kitchen at other times, six believing there was not: 'I can use it but not wander in as I want.' 'You can use the kitchen if you ask. Our kettle got burnt out because somebody didn't turn if off so you can understand.' Access to the kitchen was free in theory but limited in practice because of staff concern about risk and using up supplies. All residents believed they could go in and out of the building as they liked: 'You can go out without saying, but advice is to tell staff so they can save a meal.' This was little different from the rules on the wards, but individual patients had been more restricted there, notably by being kept in pyjamas in the MH wards to prevent unauthorised journeys.

Social relations

We first examined the proportion of time patients spent in talking, or being talked to, in the different settings. The proportion of time spent in talk increased from 18% on the wards to 34% in the hostel – a difference that would have happened by chance less than 1 in 1000 times. The increase was similar for former MH and DGH residents.

We next examined whether interaction differed qualitatively: in the people chosen, or the topics of discussion. In both wards and hostel 59% of interactions observed were with staff. In the hostel there was a slight increase in the proportion of observed interactions with visitors (10% *vs* 6%), at the expense of those with other patients. Table III shows the topics with which observed interactions were concerned. On the wards, 21% of the patients' observed talk with staff had been related to cigarettes. After a year in the hostel this proportion fell to 4%, while the proportion of more general conversational interchanges with staff rose. On the other hand, the proportion of patient-to-patient talk about cigarettes increased after the move to the hostel, though the difference was not significant. On the wards, staff rationed the supplies of some patients, thus forcing them into conversation to ask for cigarettes. In the hostel, staff did not control the cigarettes, so that when residents ran out they had to bargain with each other. The proportion of observations when patients were actually smoking did not rise significantly in the hostel, so that staff control of supplies did not seem an effective way of reducing smoking.

TABLE III

Reasons for observed social interactions in wards and hostel (n = 15)

Reason	*Interactions with staff* Wards	*Interactions with staff* Hostel	*Interactions with patients* Wards	*Interactions with patients* Hostel
Cigarettes: % of interaction time	21	4**	7	15
Food: % of interaction time	13	12	7	12
Treatment: % of interaction time	10	10	11	2
Greeting etc.: % of interaction time	12	12	14	8
Other conversation: % of interaction time	46	61	61	65
Total observations (audible interactions)	52	115	28	60

**χ^2 9.81 $P<0.01$.

In interview nine residents expressed positive feelings about staff, with four critical and two not answering. Most expressed positive feelings for other residents. However, in spite of this and their increased social interaction, three-quarters said they were often or sometimes lonely: 'I feel isolated, I've no idea why. It's not because people don't want to be friendly.' 'Lonely, yes, but there's nothing to say to anyone.' 'I should pay someone to talk to me.'

Finance

The move to the hostel had not affected the poverty of most residents. Nine still had an income of less than £10 a week, derived from social security and small 'incentive payments' for doing hostel jobs. One man did odd jobs for a florist for £1 a week: no others had any outside paid work. For five, income was brought above £10 by inheritance or pension rights.

Activity

Residents spent significantly less time doing nothing at all after they moved to the hostel as compared with both MH and DGH wards (Table IV). Although, as we have seen, they no longer attended occupational therapy, they spent as much time as before on 'work' activities. (However, this was a good deal less than the time spent at 'work' in the Maudsley hospital–hostel.) In the hostel, 'work' activities were more individualised and closely related to residents' real-life roles: for example, going shopping or practising cooking individually with the occupational therapist, doing housework or jobs in the garden. We never observed residents in the simulated mass-production activities of some occupational therapy in the hospital.

In the hostel, residents also spent more time on self-care and at meals. This was due to change in former DGH ward residents. Meals there had involved queueing for some time under supervision in a corridor to enter the canteen: patients

TABLE IV
Activity in wards and hostel (n = 15)

Activity	*Weekdays* *Wards*	*Weekdays* *Hostel*	*Saturdays* *Wards*	*Saturdays* *Hostel*	*Maudsley Hostel*[1]
Eating/toilet: % of time	9	17	15	12	(21)
Work: % of time	8	8	1	1	(30)
TV/Radio: % of time	6	11	8	8	(1)
Active Leisure: % of time	3	6	2	4	(20)
Other[2]: % of time	28	31	15	48	—
Nothing[3]: % of time	46	27***	59	27***	(28)
Total observations on hospital property	460	445	129	134	—

***χ^2 test $P<0.001$.

1. Calculated estimates from the time-budget of residents in the Maudsley Hospital–Hostel (Wykes, 1982).
2. Attending meeting, receiving medication, conversation unaccompanied by other activity, other.
3. Sitting, standing, lying down, performing no activity other than smoking.

sometimes skipped meals, at which there was little or no conversation. Meals in the hostel were served by the cook in a much more domestic and sociable atmosphere, and residents' behaviour changed accordingly.

The occurrence of inappropriate social behaviour may be taken as an index of tension and so indirectly of quality of life. On the wards patients had been rated (on the observation instrument's standard definitions) as behaving abnormally 12% of the time. In the hostel there was a significant decline to 6%.

Satisfaction with accommodation

We asked residents to tell us three good and three bad things about the hostel. The most commonly mentioned good things concerned greater freedom, the hostel's central position; and its facilities: 'Not so many rules and regulations. Don't have to call staff Mr or Mrs so-and-so.' 'It's easy to go out and do what you want to do.' 'Freedom, not as strict.' 'Easy to go out in the garden. Can do cookery or art.' 'Going on the minibus.' 'Food'. Ten residents mentioned something they did not like about the hostel. Four complained about other residents: 'Some get on my nerves a bit.' Four wanted more food/cigarettes. One did not like sleeping alone. One wanted more freedom: 'I'd like it more if I was paying my way more.' We also asked where residents would prefer to live if they could have a free choice. None wanted to return to hospital wards. Six nominated the hostel as their ideal accommodation; three wanted to live independently in their own house; three wanted to live with parents; one in another hostel; one in a hotel; and one 'in the country'.

Cases who remained on wards

We observed the six patients rejected for the hostel at baseline and follow-up periods, and we also interviewed them in the same way about their quality of life. These rejected cases were less withdrawn and more active and aggressive than cases accepted into the hostel (Gibbons, 1986). Table V shows that, in contrast to the hostel residents, those remaining on wards spent less time in the community and more time doing nothing; they hardly increased their social interaction.

The patients left in wards expressed less satisfaction in interview. Four complained of noise and lack of privacy: 'I can hear people arguing, shouting, walking, closing the door, how they go to bed. You don't even get much peace in the living room. Its a bit quieter where I sit [in the corridor].' Five of the six expressed fears of other patients and thought their property was not safe. All said they were lonely, although four saw relatives regularly. There were more rules: none could use the kitchen; all but one believed

TABLE V
Observational indices of quality of life: change in 15 patients who moved to hostel and six who stayed in wards

	Baseline *Movers*	*Baseline* *Rejected*[1]	*Follow-up* *Movers*	*Follow-up* *Rejected*[1]	*Number of individuals who improved* *Movers*	*Number of individuals who improved* *Rejected*
In community[2]: % of time	9	16	15	6	9*	1
In social interaction: % of time	18	21	34	25	13**	3
In 'work': % of time	8	17	8	9	7	1
Doing nothing: % of time	46	22	27	24	12**	1
Behaving inappropriately: % of time	12	3	6	1	9*	1

1. All rejected cases were studied at baseline in 1983 and at follow-up in 1985.
2. The fall in time spent in the community for the movers is mainly due to a change in one case, who was in the DGH unit at baseline but had been moved to the mental hospital at follow-up.

McNemar Test for Significance of Change: *$P<0.05$; **$P<0.01$.

they could not get up and go to bed when they liked; two believed they could not go out freely.

None wanted to go on living in a hospital ward; two said they wanted to move to a hostel; three wanted their own home or family; and one did not answer.

Discussion

We conclude that while the hospital–hostel provided a better quality of life on the majority of the indices we devised, its large size meant that it was somewhat overcrowded and noisy. It did not provide single rooms, considered to be highly desirable in the government's code of practice for residential care, though its residents did not complain about this. It was probably unreasonable to expect that residents' income could be improved in it, and this did not happen. The hostel residents went into the community more, and made more use of its resources, but subjectively the majority felt lonely and cut off, and those who lacked contact with relatives and friends did not increase it after moving. As yet no voluntary visitors or groups were involved; this might have facilitated a different kind of integration into the community. The main changes were in residents' increased feelings of freedom, their higher level of activity – even though staff were tolerating longer hours in bed – and a change in the amount and quality of their social interaction.

The hostel's geographical situation and staff/patient ratio did not account for the improved quality of life, except on the one measure of time spent in the community. The difference on this between cases from mental hospital and DGH unit suggested that once barriers of distance are removed, most patients find it quite natural to use ordinary community resources for their own needs. But the DGH ward and the hostel had the same geographical position and a very similar staffing ratio. Therefore, the significant changes in the activity and social interaction of former DGH ward patients were due to other causes. The most likely were the greater freedom, flexibility and informality of the hostel regime, mentioned by its residents in our interviews.

These findings, particularly on social interaction, are consistent with those from other studies of people who have moved from large institutions to smaller, more domestic units (Garety & Morris, 1984; Hemming *et al*, 1981; MacEachron, 1983). We would argue that it is worth paying a higher price for each place in a hospital–hostel than in a long-stay ward in order to obtain a better quality of life. However, future experiments might show that equally good results could be obtained in hostels with somewhat fewer professional staff and more links with voluntary groups; or even in a range of carefully planned, well supported domestic settings.

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Appendix 1

Residents' questionnaire: sample questions

Overcrowding

Probes: Do you have enough space in the bedroom?
Enough space for storing your belongings?
What about the dining room?
Is there enough space for leisure activities?

Privacy

Probes: Can you be by yourself if you want?
Can you be behind a locked door if you want?
What do you do if you want to be alone?

Danger to self

Probes: Are people threatening/violent?
Do you feel nervous/frightened of some people?
Has anyone hurt you?

Danger to belongings

Probes: Do you feel property is safe here?
Have you lost any property?

Loneliness

Probes: Have you got anyone you can really talk to?
Do you feel lonely?
Have you got any friends (details)?

Freedom

Probes: Can you use the kitchen as you please?
Can you go out when you want to?
Can you get up/go to bed when you want to?

Ideal accommodation

Probes: If you could live wherever you liked, where would you choose?
e.g. – Hospital ward (named hospital)
– Hostel
– Group home
– With family
– On your own
– Somewhere else.

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Can Categorical and Dimensional Views of Psychiatric Illness be Distinguished?

D. A. GRAYSON

Bimodality in a distribution of symptoms is often claimed to be convincing evidence that a disorder is categorical, a discrete disease entity, rather than the extreme on a continuous dimension. However, using concepts from contemporary psychometric theory it is shown that bimodality can arise from the dimensional viewpoint. In fact, contrary to the usual belief, bimodality would be expected to occur in many research contexts if the dimensional alternative were correct.

In recent psychiatric research, debate has arisen as to whether certain disorders are categorical or dimensional. For example, in depression there is debate about the binary or bipolar nature of the illness (Garside & Roth, 1978; Kendell & Gourlay, 1970*a*). One unchallenged principle is that the existence of discrete disease entities is confirmed when a linear function of scores on a symptom inventory shows bimodality. Moreover, such data are believed to be inconsistent with the opposing dimensional view (Moran, 1966). Indeed, the point is made by Fleiss (1972), Everitt (1981) and Garside & Roth (1978) that such bimodality is a sufficient, but not necessary, indicator of disease categories. The implication of this is that the demonstration of mild bimodality, or even just skew, is enough for an underlying categorical illness to be inferred. For instance, Cloninger *et al* (1985) cite, as evidence in favour of categories, the results of 'admixture analysis' showing that two normals are better than one in fitting a discriminant function distribution.

Few would argue with the description given by Kendell (1975) of the conceptual linkage from a categorical illness to bimodality. But before one makes the reverse inference from bimodality to a categorical illness, there is a fundamental obligation to show that such bimodality cannot arise from a dimensional illness. Ruling out this latter alternative has not received appropriate attention.

Everitt (1981) states what is probably a widely held belief:

> Now for a *population* frequency curve, bimodality is (except in pathological cases) a sufficient (although not a necessary) condition for the presence of subtypes and certainly if, in a fairly large sample, bimodality appeared no matter how the data were arranged, it would be pedantic to insist it might be an artefact.

Garside & Roth (1978) provide a more substantial argument for believing bimodality is inconsistent with a dimensional illness. In discussing bimodality of a linear discriminant function score, they state:

> If a significantly non-unimodal distribution is then obtained, it seems safe to reject the hypothesis that the population distribution is unimodal, and to conclude that distinct groups of patients exist.

This conclusion is supported by an earlier statement to the effect that the central limit theorem entails unimodality in the sum of a number of (almost) uncorrelated (0, 1) variables. However, to infer distinct groups, and not a dimensional illness, requires the truth of a premiss suppressed in their reasoning: it must be true that a dimensional illness will yield data wherein pairs of symptoms show little or no correlation.

This premiss is in fact false, and the main thesis of this paper is that symptom–symptom correlations yielding bimodality can arise from a dimensional illness, and that such a state of affairs is far from being a 'pathological' case, as has hitherto been assumed.

The paper is divided into three major sections. The first demonstrates how bimodality can arise from a dimensional illness and exhibits similarities in the way symptom inventory data may arise from either dimensional or categorical illnesses. The second section discusses research biases which tend to ensure that dimensional and categorical illness will be indistinguishable. The final section deals with the implications of these issues.

From here on it is assumed that the bimodality under discussion arises from a symptom inventory of dichotomous symptoms scored 1 or Yes in the direction of illness. It is further assumed that the score at issue is the simple sum of endorsed symptoms. These simplifications are made in the interest of clarity, and the points made below readily generalise to weighted symptoms sums and finer symptom scoring than (0, 1).

Bimodality with dimensional illnesses

This section explains Latent Trait models (contemporary psychometric accounts of questionnaire data assuming a latent dimension) and presents distributions of the symptom sum that explore the conditions under which a dimensional illness will yield bimodality. The close conceptual similarity between such dimensional models and disease entity models is examined.

Latent Trait model

The main applications of the Latent Trait theory are in aptitude-testing (Rasch, 1960; Birnbaum, 1968; Lord, 1980), and the theory seeks to account for questionnaire data in terms of a single latent dimension and aspects of the individual items. If one equates the items with symptoms and regards the questionnaire as an inventory relating to an underlying dimension of illness severity, then the theory is quite appropriate in psychiatric contexts. Such applications are now appearing in the literature (Gibbons *et al*, 1985; Duncan-Jones *et al*, 1986; Grayson, 1986).

The model usually assumes that the population takes a standard normal distribution (mean zero, unit variance) on the underlying illness dimension. Those with positive scores are 'sicker' than those with negative scores. The remaining feature of the model attempts to capture how the individual symptoms relate to this latent illness dimension. Generally it is assumed, for each symptom, that probability of endorsement is an increasing function of illness severity only. The higher a subject is on the illness dimension, the greater his probability of endorsing a given symptom. This assumption is very general, and in applications the symptom functions are given explicit functional form.

Figure 1 represents such a structure. On the x-axis the value on the latent dimension is plotted. The lower graph shows, inverted, the normal distribution of subjects on this dimension, and the upper graph shows, for a given symptom, the function relating endorsement probability to the value on the latent dimension. Subjects higher on the dimension ('sicker') have higher probability of symptom endorsement. This particular symptom function has the 2-parameter logistic representation. If $P_j(z)$ is the probability of symptom j endorsement for a subject of illness dimension value z, then

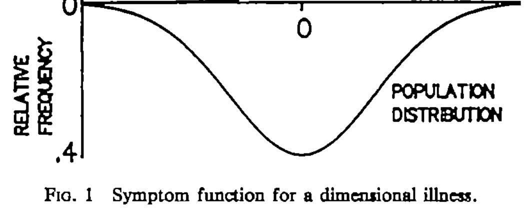

FIG. 1 Symptom function for a dimensional illness.

$$P_j(z) = \frac{\exp\,[s_j(z-t_j)]}{1+\exp\,[s_j(z-t_j)]}$$

or

$$\log\left(\frac{P_j(z)}{1-P_j(z)}\right) = s_j(z-t_j)$$

The expression is characterised by two parameters, s_j and t_j. When $z=t_j$, $P_j(z)=0.5$. So t_j is the value of the illness dimension that yields a 50% chance of endorsing symptom j. It is called the 'threshold' of the symptom, and serves to locate the symptom at a particular place on the illness dimension. This logistic curve rises slowly from the left, has maximum slope at $z=t_j$, and flattens out above this value. The slope at $z=t_j$ is $s_j/4$, *and the* s_j parameter is called the 'slope'. The larger the s_j value, the better the symptom is discriminating at threshold, where it must discriminate maximally. The symptom in Fig. 1 has a threshold at $t_j=1.0$ and a slope of $s_j=2.0$, indicating that it is discriminating well among moderately sick (positive z) subjects, but not among the well or the extremely sick.

Dynamics of dimensional bimodality

By choosing a number of symptoms with particular (s_j, t_j) combinations, it is possible to generate the data arising from application of a symptom inventory to a dimensional illness, and thus to investigate the conditions under which bimodality occurs. Such theoretical distributions are presented in Tables I–III.

Table I shows the distributions of this number-endorsed score for a variety of inventories applied to a normally distributed latent dimensional illness. In each inventory all symptoms are identical, with $t_j=0$ and s_j as indicated in the table. The number of symptoms in the inventory is also specified ($n=6$ or $n=23$). The slopes range from 16 to 1.9.

In all these examples, distinct bimodality occurs. The dynamics of this effect seem clear. All symptoms are located in the middle of the population ($t_j=0$). Thus, the bulk of the subjects low on the dimension rarely endorse many symptoms, and the bulk of those high on the dimension rarely fail to endorse many symptoms. This is more so for steeper slopes. Thus over intermediate patterns of symptoms there is a preponderance of all-zero or all-one patterns of responses, and hence bimodality in the distribution. So two conditions jointly yielding dimensional bimodality are coincident symptom thresholds and moderate to steep slopes. In other words, the symptoms must be discriminating well at a similar level of illness severity.

Examples in Table II show that the bimodality is surprisingly robust. The twelve-symptom inventory consists

TABLE I

Bimodal distributions from dimensional models; 1000 subjects; $t_j = 0$

Slope (s_j)	Inventory size (n)	Distribution of number of endorsed symptoms 0	1	2	3	4	5	6	7	8	9	10	11	12
16	6	443	30	19	17	19	20	443	—	—	—	—	—	—
16	12	425	27	15	11	9	8	8	8	9	11	15	27	425
7.94	6	389	58	37	33	37	58	389	—	—	—	—	—	—
7.94	12	354	51	29	22	19	17	17	17	19	22	29	51	354
3.95	6	293	102	72	66	72	102	293	—	—	—	—	—	—
3.95	12	235	82	54	42	37	34	33	34	37	42	54	82	235
2.60	6	218	130	103	97	103	130	218	—	—	—	—	—	—
2.60	12	149	92	70	60	54	51	50	51	54	60	70	92	149
1.90	6	161	144	131	127	131	144	161	—	—	—	—	—	—
1.90	12	91	87	79	73	70	67	67	67	70	73	79	87	91

TABLE II

Distributions from dimensional models; 1000 subjects

Inventory size (n)	Distribution of number of endorsed symptoms 0	1	2	3	4	5	6	7	8	9	10	11	12
6	140	200	112	96	112	200	140	—	—	—	—	—	—
12	70	114	112	73	57	51	49	51	57	73	112	114	70

TABLE III

Distributions from dimensional models with no extreme symptoms; 1000 subjects; $s_j = 3.0$

Thresholds	Inventory size (n)	Distribution of number of endorsed symptoms 0	1	2	3	4	5	6	7	8	9	10	11	12
0, 0.2, 0.5, 0, −0.2, −0.5	6	213	130	107	101	107	130	213	—	—	—	—	—	—
	12	149	88	69	60	55	52	52	52	55	60	69	88	149
0.1, 0.3, 0.6, −0.1, −0.3, −0.6	6	197	132	115	111	115	132	197	—	—	—	—	—	—
	12	136	86	70	63	59	57	57	57	59	63	70	86	136

of two six-symptom inventories combined, and both these inventories consist of symptoms with (s_j, t_j) combinations (4, 0), (4, 0), (4, 0.2), (4, −0.2), (1.7, 1.2), (1.7, −1.2). The total proportions endorsing the symptoms are 0.5, 0.5, 0.43, 0.57, 0.2 and 0.8, respectively. The first, third and fifth symptoms have thresholds lying at the 50th, 58th and 88th population percentiles, respectively. The fifth and sixth symptoms, at either extreme, have the effect of ensuring that few subjects at the well extreme and few subjects at the sick extreme obtain inventory patterns of all zeros or all ones respectively. Thus the distribution is bimodal with two 'humps' rather than U-shaped.

All the distributions in Table III were generated with symptoms having a common slope of 3.0. The six-symptom inventories were constructed from three such symptoms with given thresholds and three with the same, but negative thresholds. The twelve-symptom inventories, again, are two six-symptom inventories combined. Removing the extreme items restores the more extreme U-shape bimodality.

All these examples demonstrate that a dimensional illness can produce bimodality, and together they provide insight into how this occurs. The conditions seem to be that the symptoms all perform their best discrimination round about the same place on the latent dimension. In terms of the

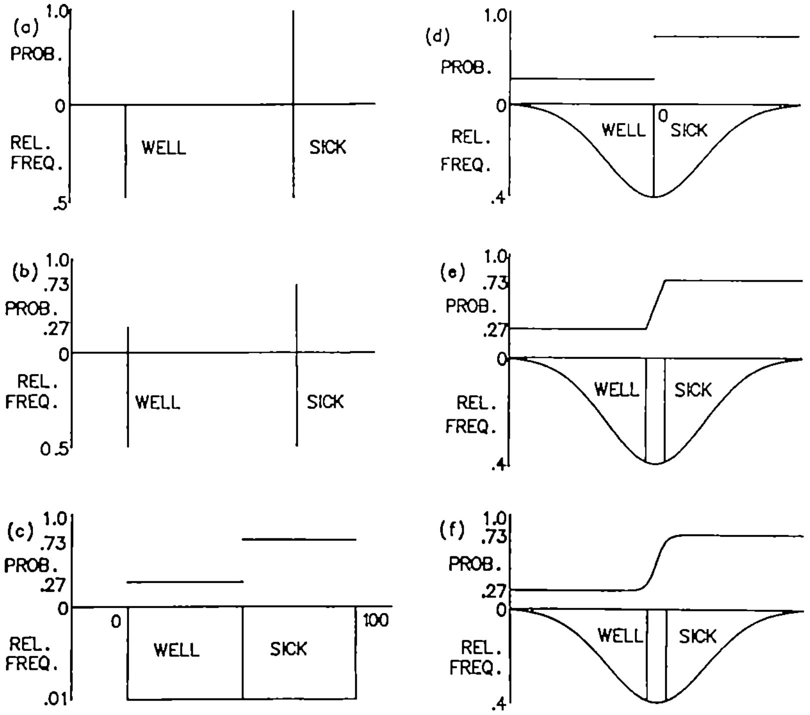

Fig. 2 Symptom function for a categorical illness.

symptom functions used here, the thresholds of a majority of symptoms must cluster about a common region on the illness dimension, making the population appear as two groups in the inventory data.

Similarity of data from dimensional and categorical illnesses

The data from a symptom inventory consist of frequencies of the possible patterns of ones and zeros across the inventory. These in turn depend on the relationship between each symptom and the underlying illness. If the responses to a given symptom can be shown, in principle, to be produced in much the same way from either type of illness, then it is, in principle, impossible to discriminate categorical from dimensional illness using data from a symptom inventory.

Part (a) of Fig. 2 represents the ideal relationship between a symptom and a categorical illness (with prevalence of 0.5 in the study population). Sick subjects, and only sick subjects, endorse the symptom. This is ideal, and an inventory of such symptoms would yield response patterns consisting of all zeros (the well group) and all ones (the sick), and nothing intermediate.

Two ways in which this unrealistic ideal might be realistically degraded are as follows. The first is by allowing the symptoms to be less than perfectly reliable. The second is by allowing the illness to be less than perfectly categorical; that is, having some subjects intermediate between well and sick. In accordance with the concepts of Kendell (1975), such subjects should be relatively infrequent.

Part (b) of Fig. 2 shows a symptom which is not perfectly reliable. Sick subjects have 0.73 probability of endorsing the symptom, well subjects 0.27. Part (c) is another way of representing Part (b), but along an artificially constructed dimension instead of illness categories. The well group is aligned fully to the left of the sick group. Within each group the subjects are ordered randomly. To each subject one can assign a percentile score reflecting the percentage of the total population to the left of that subject. Thus, well subjects will have percentile scores of 50 or less, and the sick will have greater than 50. A given subject still retains the same symptom probability as in part (b). Expressing the mechanism giving rise to symptom data in this way is theoretically fully equivalent to that in part (b), even if artificial.

Part (d) of the figure takes the representation one step further, but is still equivalent to the two earlier representations. To each subject is assigned the z-score associated with that subject's arbitrary percentile score (obtained by using tables of a standard normal variate). The symptom's relationship to the two categories is now shown as a symptom function over a score which is normally distributed in the study population, albeit a 'score' with no empirical interpretation.

Next, the assumption of less than perfectly distinct categories is incorporated by imposing a small 'grey area' between the sick and well groups. Sick subjects still have 0.73 probability of symptom endorsement, well subjects 0.27, and intermediate subjects have intermediate symptom-endorsment probability, as in part (e) of the figure.

Finally, if we allow the reasonable possibility that within either the well or sick groups, symptom-endorsement probability is not totally homogeneous, part (f) results.

Comparison of part (f) with Fig. 1 reveals the close conceptual similarity between the mechanisms generating symptom inventory data in categorical and dimensional models of illness. It is this close similarity which, in principle, ensures that discrimination between the two views, using inventory data, will be difficult.

A categorical illness differs, in general, from a dimensional one for two reasons:

(a) If the subjects in the 'grey area' are infrequent, then the symptom functions must rise steeply above the corresponding region (the functions must have steep slopes).

(b) Each symptom function must change above the same region (the thresholds must cluster).

These are precisely the conditions under which a dimensional illness will yield bimodality, and it is argued in the next section that psychiatric research on this issue has systematic biases which tend to ensure that these conditions are met when an illness is dimensional.

Influence of bias on dimensional illness

Types of bias

Most researchers are aware of 'observer bias', outlined by Kendell (1968). Given a particular inventory, a researcher predisposed to a categorical view of the illness simultaneously classifies patients and completes the diagnostic inventory in accordance with his or her predisposition.

Symptom selection bias

The same prejudgement can operate, however, in the initial selection of the symptoms to be included in the inventory, even were it to be subsequently administered by an incorruptible computer. If an illness truly is dimensional and a researcher is predisposed to the (erroneous) categorical view, the researcher's belief or intuition must be based on observation of some sort, and presumably those extreme on the dimension will share characteristics that vaguely define the researcher's mistaken concept of a sick group. Consequently, it is to be expected that the symptoms chosen will be none other than the characteristics discriminating the dimensionally extreme from the rest. That is, the symptoms likely to be included in the inventory will be a biased selection from the possible universe of symptoms, and will have thresholds constrained to cluster about the region on the dimension corresponding to the researcher's mistaken perception of the categories' boundary.

Such a bias, if operative, is compounded by the common advice to exclude items whose overall endorsement probabilities are far from 50% (outside the 20–80% range), at least in study populations constrained to have near-equal representation of the putative sick and well groups.

Subject selection bias

In most applications of the bimodality approach, if not most psychiatric research, the study population is not the community population. Even when the community population is distributed normally along an illness dimension, if study populations overemphasise one or both extremes of this continuum, then they must artificially conform to a disease entity situation. For example, Cloninger *et al* (1985) selected hospitalised schizophrenics to compare with others, and search for bimodality. If, in reality, the illness is dimensional, then such a selected population will not include moderate schizophrenics who fail to appear at hospital, thus creating genuine distinct groups in the artificial study population.

Figure 3 represents the action of these biases. Three symptoms with slopes of 2.0 are shown in part (a). They have thresholds of 0.7, 1.0 and 1.3, indicating the operation of symptom selection bias. The population is the community population. Part (b) shows the effect of overemphasising the extreme 15% of the community population so that they

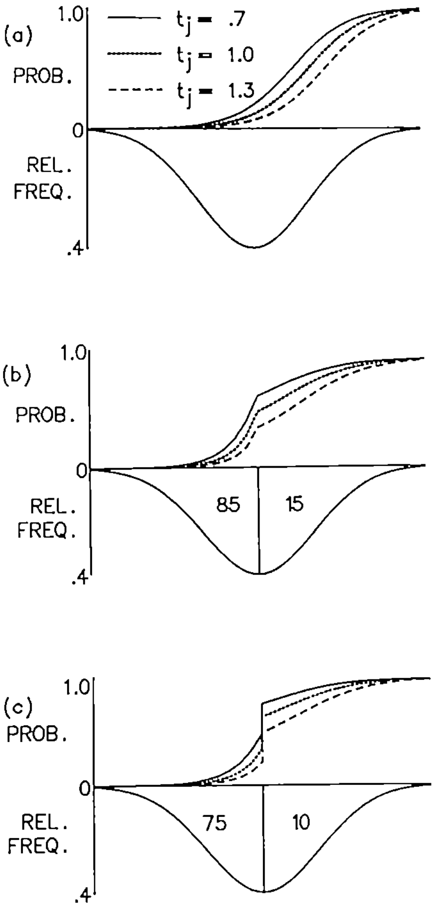

FIG. 3 Influence of biases on a dimensional illness.

become 50% in the study population. Part (c) describes the situation where the lower 75% and the upper 10% are chosen to form the artificial study population. These biases serve to ensure that the conditions required for categorical and dimensional illnesses to manifest identically are in fact met; that is, guarantee that the clustering of thresholds and steepening of slopes required for dimensional bimodality will arise.

Illnesses with two subtypes

The same problems arise in the search for binary disease entities, such as reactive and endogenous depression, although the interplay is more subtle. The study population is usually taken from treated series in hospital or clinics; for example, from hospitalised depressives, in the search for reactive and endogenous categories. As Eysenck (1970) says:

> It seems likely that selection for hospitalization is more in terms of severity . . . than in terms of endogenous or reactive . . . This might easily lead to a predominance of patients combining symptoms of endogenous and reactive depression in hospital, thus . . . disguising any bimodality that might actually exist. Conversely, it is possible that the Newcastle workers have selected patients for inclusion on the basis of extreme scores on Kendell's continuum, thus affecting the shape of the distribution in the opposite direction.

The appearance of bimodality is also related to the selection of symptoms. If the symptoms chosen are those that discriminate endogenous depressives (be they from a category or a dimensional extreme) from normals, and reactive depressives from normals, then they will not discriminate ideally between the two depressions. For this latter task, symptoms are required which are simultaneously endogenous and not reactive (or vice versa). On the other hand, symptoms which discriminate between the two groups may do so on bases other than endogenous versus reactive depression. Two groups of people will differ not only on the criterion used to segregate them, but on a variety of correlated criteria. Such other criteria, each separately correlated with the segregation criterion, need not intercorrelate among themselves, and thus will not produce bimodality, as the following example demonstrates.

Consider a two-symptom inventory applied to two groups of 16 subjects each, classified as endogenous and reactive depressives. Suppose the response patterns (1, 1), (1, 0), (0, 1) and (0, 0) occur with frequencies 8, 4, 4, 0 respectively among the reactives, and with frequencies 0, 4, 4, 8 among the endogenous depressives. For either symptom, the association with the depressive categories is given by the 2×2 table

	Symptom response		
	Yes	*No*	
Reactive	12	4	16
Endogenous	4	12	16
	16	16	32

As discriminators of the classification all items perform well; the χ^2 is 8 on 1 degree of freedom. Yet the number-endorsed distribution is unimodal, even with only two symptoms. Scores of 0, 1, 2 occur with frequencies of 8, 16, 8.

The key to understanding this apparent anomaly lies in the observation that the symptoms are uncorrelated:

		Symptom 1		
		Yes	*No*	
Symptom 2	*Yes*	8	8	16
	No	8	8	16
		16	16	32

Bimodality arises with a preponderance of patterns of two types, almost all zeros and almost all ones. This requires intercorrelations among the symptoms; it is not sufficient that they discriminate well individually. The lack of such intercorrelations indicates that the symptoms are discriminating on unrelated criteria. Constructing an inventory which is heterogeneous in this sense will ensure, even when the symptoms individually discriminate a common classification, unimodality on some dimension which is a pot-pourri of them all. Conversely, an inventory with symptoms tapping the same construct, and with all symptoms centrally located, must yield bimodality whether the construct is dimensional or categorical.

This phenomenon may underlie the apparent conflict between the depression bimodality found by Carney *et al* (1965) and the failure of Kendell & Gourlay (1970*a*) to replicate it. Close reading of the symptom selection procedures of the former shows that a major factor in selection was *a priori* content validity of the symptoms in their instrument, while the latter authors report including symptoms which discriminated their classification but appeared unrelated conceptually to the distinctions between the depressives. They also report excluding symptoms from their final inventory that would usually be included on the basis of their content. Similar comments apply to unimodality found by Kendell & Gourlay (1970*b*) in their search for schizophrenic and affective categories.

Implications

Latent Trait models have been used as a heuristic device to demonstrate that bimodality may arise from a dimensional illness. This counter-intuitive result has not been noticed in the past (Moran, 1966; Maxwell, 1971; Kendell, 1975; Garside & Roth, 1978). Far from being a pathological outcome, the likelihood of such dimensional bimodality is enhanced by symptom and subject selection biases.

Statistical analysis of symptom inventory data

If one accepts the reasoning in this paper then mixture analysis, as recommended by Fleiss (1972) and Everitt (1981), and practised by Cloninger *et al* (1985) is, unfortunately, useless for distinguishing between categorical and dimensional illness models. This statement is no less true of discriminant analysis, factor analysis, clustering, numerical taxonomy (Pilowsky *et al*, 1969) and Boolean factor analysis (Weber & Scharfetter, 1984). All of these techniques are applied to the basic symptom inventory data, and insofar as these data are incapable of distinguishing the two illness models, any subsequent analysis of such data is also incapable of making such a distinction.

The technique of Kendell & Brockington (1980) is also suspect. Non-linearity between an outcome score and a symptom inventory scale score would be quite possible whenever bimodality arises in the latter. Since this can occur with dimensional illnesses, so can the non-linearity they would interpret as a hallmark of categorical illness states.

Recommendations

From this perspective, certain conditions must be met before bimodality can be used to infer that an illness is categorical. First, the study population must not be artificially constructed so that it conforms to a categorical structure if in fact the illness is dimensional. In this sense, the community population is an ideal study population.

Secondly, it must be shown that the symptoms chosen are not preselected on *a priori* grounds which relate to an erroneous belief that the illness is categorical. This is tantamount to ensuring that no symptoms can be found which discriminate among the study population at locations other than the putative boundary between the categories.

Thirdly, the symptoms must be homogeneous, or discriminating among those with and without the disease on the same basis, namely that related to the disease in question.

Even then the inference is more an evaluative judgement than a rejection of the dimensional view on qualitative grounds.

Conclusion

In exploring the ramifications of a dimensional view of illness, the nature of a symptom inventory as an instrument has been emphasised. What is seen of the world is an interactive function of three components: reality, the viewing instrument and the viewer. It is quite possible for reality to be continuous, for the instrument to function best at just one point on that continuum, and for the viewer to point the instrument selectively at that region and to conclude that this region is a qualitative boundary. This is not to say that it is not a boundary, but it is not to say that it is. Before one so concludes, there is an onus to demonstrate that the instrument is not selective, but is perceiving reality as it is.

Where the instrument is a psychiatric inventory, Latent Trait techniques seem the most promising way of unravelling this interaction.

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Life Events and Psychiatric Disorder: The Role of a Natural Disaster

ALEXANDER C. McFARLANE

Examining the impact of natural disasters on psychological health provides an opportunity to study the role played by extreme adversity in the onset of psychiatric disorder. Four hundred and sixty-nine fire-fighters who had been intensely exposed to an Australian bushfire disaster completed a detailed inventory of their experiences four months later. They also completed a brief life events schedule and the 12-item General Health Questionnaire. Only 9% of the GHQ score variance could be accounted for by the disaster and other life events; the effects of the disaster appeared to be separate and additive. This is similar to the relationship between life events and psychiatric illness found in other settings. It is suggested that vulnerability is a more important factor in breakdown than the degree of stress experienced.

Much research has tested the hypothesis that adverse life events are causally related to the onset of psychiatric disorder (Brown *et al*, 1976; Paykel, 1978; Tennant, 1983; Cooke & Hole, 1983). Most of this research has examined the additive effect of a range of diverse experiences on psychiatric impairment. An alternative method is to study a population comprised of people who have all experienced the same life event, but with a range of intensities. This research design has been used infrequently. One setting where it might be utilised is in investigating the effects of a natural disaster on psychological disorder, since the individual experiences of disaster victims vary, although they have all been exposed to the same highly distressing event.

Previous disaster research has not universally supported the conclusion of life events research that adversity is causally related to the onset of psychiatric disorder (Green, 1982). There is a range of opinion about the psychological consequences of disasters, extending from the claim that the level of morbidity is high and long-lasting (Gleser *et al*, 1981; Leopold & Dillon, 1963) to the view that disasters have little or no important effect on mental health (Quarantelli & Dynes, 1977). A major reason for this controversy lies in the failure of much disaster research to use a quantitative rather than a qualitative methodology. Green (1982) has argued that future disaster research must carefully document the intensity of exposure and the nature of the losses sustained in the disaster, and must relate these to the levels of psychiatric morbidity.

Many potential problems exist in using this approach, because accepted measures of the impact of a disaster have not been developed. Furthermore, their validation presents problems for most researchers, because major disasters are infrequent. The experience of researchers into other life events could assist in this process of validation.

Although there are a number of common methodological and theoretical issues, there has been little cross-fertilisation of ideas between disaster research and life events research (McFarlane, 1985). For example, it has been concluded that although a link exists between adversity and neurotic impairment, the relationship accounts for only about 10% of the variance of disorder (Tennant, 1983). On the other hand, life events researches generally accept that "disasters show that when psychological stress is sufficiently severe, virtually everyone decompensates and becomes demoralised, dependent, and identifiable as a psychiatric case" (Andrews & Tennant, 1978). Such assertions about the level of psychiatric morbidity following a natural disaster were quantitatively investigated in the present study.

The nature of the interaction between different major adverse life events and the onset of psychiatric disorder is another issue to be examined in disaster victims. Holmes & Rahe (1967) suggested that events had an additive effect, whereas Brown & Harris (1978) assumed that once a single event of sufficient intensity had been experienced, other events were relatively unimportant. If the first hypothesis were correct, the experience of substantial adversity prior to a disaster would have an effect additive to that of the disaster; if the second were correct, prior adversity would have a more powerful association with psychiatric morbidity than would the disaster event.

The present study examined the two hypotheses by investigating the impact of a bushfire disaster on a group of fire-fighters who had varying exposure to it. The first hypothesis was that a significant relationship would exist between the degree of adversity experienced by individual fire-fighters and their level of psychiatric morbidity but that this would not account for more than 10% of the total variance. The second hypothesis was that major adverse life events would show a relationship with psychiatric disorder which was independent of the effects of the natural disaster.

Method

The setting: disaster and fire-fighters

On 16 February 1983 several disastrous bushfires burnt 2804 km^2 of bush, grazing land, orchards, forests, and national parks in South Australia. Twenty-eight people were killed and many hundreds injured, and over 250 000 stock animals were killed. The fire destroyed or severely damaged 385 homes, and 3200 properties were affected in some way. A conservative estimate of the cost of the damage was $A200 million. The psychological impact of the disaster has been reported elsewhere (McFarlane, 1984; McFarlane & Raphael, 1984).

A large organisation of volunteer fire-fighters exists in Australia to combat such fires. Several thousand of these firefighters, living both within and outside the fire-devastated area, attempted to control the blaze. Three were killed, a number lost their homes, others had their stock and farms destroyed, and many were injured. This group provided an ideal opportunity to study the onset of psychiatric morbidity following a disaster, because of its wide range of exposure and losses.

Questionnaire design

A preliminary questionnaire was designed, comprising an inventory of the impact of the disaster, a brief life events inventory, and the 12-item General Health Questionnaire (Goldberg, 1972). It was administered to 26 senior fire-fighters and then discussed with them. Ambiguities were corrected, and several questions added to ensure that a complete description of the fire-fighters' experiences was obtained. The inventory of the impact of the disaster consisted of 15 questions about personal and property losses, 9 about the nature and duration of the fire-fighters' exposure to the fire, and 8 documenting the injuries sustained and the type of treatment received. These allowed an overall rating of severity.

The brief inventory of life events experienced before the fire was based on items 2, 12, 13, 14, 15, 45, and 60 from Tennant & Andrews' (1976) life event inventory. These items focused on financial and legal difficulties, bereavement, and ill-health – factors that might be expected to influence the experience of a disaster. The final part was the 12-item General Health Questionnaire (Goldberg, 1972). This has been validated as a reliable measure of psychiatric impairment in an Australian population (Tennant, 1977), in which setting, used as a 'caseness' measure, it has been shown to have a specificity of 91.7%, a sensitivity of 86.6%, and a misclassification rate of 10%. For correlational analysis the Likert score of the GHQ was calculated. A cut-off of 1/2 was used for definition of 'caseness'; the prevalence of cases was also calculated at a cut-off of 2/3.

Sampling method

The sample was chosen from the volunteer fire-fighting service. This did not have an accessible current membership list at the time of the disaster, and therefore the questionnaires had to be distributed through headquarters staff. They passed them on to the brigade commanders, who in turn gave them to the volunteers. A total of 1500 questionnaires were delivered for distribution three months after the disaster. Despite a request, no record was made of exactly how many of the 1500 questionnaires were given to fire-fighters. No follow-up reminders could be sent to improve the return rate, because the names of the fire-fighters who had received the questionnaires were not recorded. A total of 489 questionnaires were returned, by prepaid postage, a mean of one month later, i.e. four months after the disaster. Twenty questionnaires were excluded because the reponses were not sufficiently complete.

The apparent low return rate of 30% raises the possibility that a potential bias existed in the sampling. However, it may have been an artefact, as subsequent discussion with headquarters staff revealed that a significant number of questionnaires had possibly not been distributed by the brigade officers, some of whom had sustained major losses themselves in the disaster and had no time to spare from reconstruction. A comparison sample of fire-fighters was therefore studied in an attempt to ascertain whether a range of demographic variables, or the nature of the individual's experience of the disaster, had biased the initial sampling. Eleven months after the fire the fire-fighting organisation had updated its membership records and these were made available. Seven brigades, selected on advice from headquarters staff because they were seen to be representative of the fire-fighters in terms of experience of the disaster and membership composition, were chosen for study as a comparison group. The original sample was resurveyed at the same time (this data is not reported in this paper, except for the GHQ scores).

Two follow-up letters were then sent to those comparison group subjects who did not return the questionnaire, to ensure an adequate response rate. By comparing this group with the original sample over a range of variables it was possible to establish the representativeness of the original sample within the organisation. Most members of the fire-fighting organisation were involved in the disaster, so no non-exposed control group was available.

Method of scaling

The range of exposure and losses sustained in the diaster was known to be substantial. The questionnaire was therefore designed to allow separate scaling of four aspects of the fire-fighters' experience of the disaster. Firstly, the personal loss sub-scale recorded the number of people known to the fire-fighter who were killed in the fire and the nature of their relationship to the subject. The weighting of losses was based on the relative weighting for change of the items in the Tennant & Andrews (1976) life events inventory, which has been validated in an Australian population (e.g. death of a close friend = 1, death of a sibling = 2, death of a spouse = 3). The names of all those killed or seriously injured in the fire were published in the media. This made it possible to validate the reporting of deaths or injury in next of kin or first degree relatives by cross-referencing the surnames of the victims with the research subjects. No misreports of personal losses were found.

Secondly, the injury sub-scale was weighted on the basis of the type of treatment required, rather than on reported severity of injury which might have been subjective (e.g. hospitalisation = 3, medical out-patient care = 2, nurse or ambulance officer = 1).

Thirdly, the exposure sub-scale items (which measured the time spent fighting the fire, the use of emergency procedures to protect the fire unit, whether the subject was trapped by the blaze, etc.) were additive and unweighted. The inter-relater reliability of this sub-scale was found to be acceptable by comparing the reports of members of the same units who had had similar, but not identical, experiences of a fire ($r = 0.76$).

Finally, the scoring of the property loss sub-scale covered a range of damage and losses. This included complete destruction of a fire-fighter's home or other property and buildings, as well as damage in varying degrees. Because a number of fire-fighters were farmers, loss of income was also recorded. The items of this sub-scale were weighted according to the amounts of practical assistance require to reparate for losses. The weighting was obtained by surveying the special team of 20 bushfire relief workers appointed to assist the disaster victims, who were able to assess independently the impact of losses in the context of the disaster; their averaged responses were used as weighting. Thus the scaling of losses was not contaminated by the distress generated. The procedure is to be described in more detail elsewhere.

Statistical analysis

The data were analysed using the Statistical Package for the Social Sciences (SPSS) (Nie *et al*, 1975; Hull & Nie, 1981); a probability level of one in twenty was used. Groups were compared using chi-squared, Mann-Whitney *U* and Student's *t*-tests, according to the characteristics of the variable being examined. The variance of the GHQ score was investigated using a multiple regression analysis (Hull & Nie, 1981) which only examines the contribution of significantly loading variables. Pearson's correlation coefficients were calculated, because of the size of the sample studied and because the SPSS only computes regression analyses using parametric statistics.

Results

Sample

When the original cohort of 469 fire-fighters was compared with the comparison group on a range of demographic variables, several statistically significant, but small, differences emerged (Table I). The mean age of the study sample was less by two years than that of the comparison group, and the comparison group was of slightly higher social class (Krupinski *et al*, 1966), more of its members being farmers. The two groups' experience of the disaster, measured by the sum of their fire-related experiences, was not significantly different, and they did not differ significantly on GHQ scores.

Disaster experience

Individual experience of the disaster varied widely. Some degree of property damage was suffered by 23% of the sample, in many cases enough to affect their livelihood. None of the subjects had lost a first degree relative,

TABLE I

Study sample and comparison group data

	Study sample ($n = 469$)	*Comparison group ($n = 162$)*	*Statistical comparison*
Return rate: %	30	59	
Married: %	73.6	72.2	$\chi^2 = 0.54$
Australian born: %	87.7	87.7	$\chi^2 = 0.0000$
Social class^1: %			$\chi^2 = 14.0^*$
1	19.3	23	
2	32.7	37.9	
3	7.4	5.0	
4	28.4	16.8	
5	5.1	10.6	
Other	7.2	6.8	
Age^2: years			$t = 2.6^*$
Mean	35.1	37.8	
s.d.	10.6	13.1	
Disaster experience			Mann-Whitney *U* test: $z = 0.26$
Mean	8.9	9.2	
s.d.	3.0	4.2	
GHQ^3 score			$t = 0.41$
Mean	1.4	1.5	
s.d.	2.3	2.9	
Proportion of cases found by GHQ: %	26.8	26.3	$\chi^2 = 0.0012$

1. Classified following Krupinski *et al* (1966).
2. At time of comparison.
3. GHQ scores 11 months after the fire.

$^*P \leq 0.05$.

TABLE II
Pearson correlations between GHQ and other variables (n = 469)

	Rating[1]			
Variable	*Mean*	*s.d.*	*r*	*P*
Exposure	7.0	1.5	0.12	0.007
Perceived threat	2.7	0.8	0.13	0.004
Injury	1.5	0.9	0.02	0.3
Property loss	0.7	1.7	0.20	0.000
Personal loss	0.5	1.2	0.03	0.2
Recent life events	0.9	1.0	0.17	0.000
Previous fires	4.8	8.2	0.12	0.006
Age	33.7	10.6	−0.01	0.4
Social class	3.6	2.1	0.06	0.1

[1] Based on questionnaire score.

although 7% had experienced some bereavement due to the death of a more distant relative or close friend. The mean during of time fighting the fire was 15.6 hours. During that time 19.7% of subjects believed they had come close to dying, and 41.2% had had to protect themselves from the fire using emergency procedures. Of the 26.6% injured, 12% had been admitted to hospital.

Relationship between GHQ and fire experience

The relationship between the GHQ and a range of variables was examined (Table II). Most fire-related variables were significantly correlated with the GHQ score, with the exception of personal loss and injury. Experience of previous fires was also significantly associated with the GHQ score, although age and social class (Krupinski *et al*, 1966) were not. Experience of recent adverse events prior to the disaster was significantly correlated correlated with the GHQ score. The size of these correlation coefficients was generally small, in part owing their significance to the size of the sample.

TABLE III
Comparison of cases with non-cases (as defined by GHQ scores)

	Rating for cases[1] *(n = 146)*		*Rating for non-cases (n = 311)*		*t*	
Variable	*Mean*	*s.d.*	*Mean*	*s.d.*	*(2-tailed)*	*P*
Exposure	7.3	1.6	6.9	1.4	−2.3	0.02
Hours fighting	18.9	19.2	14.3	12.8	−3.0	0.003
Perceived threat	2.8	0.8	2.6	0.7	−2.5	0.01
Personal loss	0.5	1.2	0.4	1.2	−0.8	0.4
Property loss	1.3	2.2	0.5	1.4	−4.7	0.000
Injury	1.5	0.9	1.5	0.9	−0.7	0.5
Previous fires	5.7	10.5	4.5	7.0	−1.4	0.2
Age	32.5	8.8	33.9	11.1	1.4	0.2
Social class	3.2	1.6	2.9	1.4	−1.4	0.1
Recent life event	1.14	1.19	0.77	.93	−3.6	0.000
GHQ	4.76	2.8	0.19	0.4	−28.3	0.000

[1] Based on GHQ$\geq$2.

The 146 'cases', defined by using a cut-off point of 1/2 on the 12-item GHQ, were compared with 311 'non-cases' (Table III). On all variables that were significantly different, those subjects demonstrating psychiatric impairment had a higher level of exposure to the disaster. Again, personal loss and injury were not significantly different between cases and non-cases.

Prevalence of disorder

The prevalence of cases among the fire-fighters was 30% using a 1/2 cut-off. When the 2/3 threshold was used, the prevalence was 23.2%.

Variance attributable to the disaster

The proportion of the variance of psychiatric impairment that could be explained by experience of the disaster and other recent life events was examined using stepwise multiple regression analysis of the GHQ. The fire-fighters' exposure, perceived threat, severity of injury, personal loss, property loss, and the recent events score were included in the equation. The only significantly loading variables were property loss, accounting for 4.9% of the variance (multiple $r = 0.23$, adjusted $r^2 = 0.049$, $f = 19.03$, $P = 0.000$), and recent life events, which predicted a further 3.8% (multiple $r = 0.30$, adjusted $r^2 = 0.087$, $f = 17.69$, $P = 0.000$). None of the other variables predicted a significant percentage of the variance.

Discussion

The data from this study provided support for the first hypothesis, i.e. that a significant relationship would exist between the degree of adversity experienced by individuals in a disaster and their level of psychiatric morbidity. However, it should be noted that this relationship was weak. For example, although the cases and non-cases were significantly different on a number of fire-related variables, the differences in mean values were not great.

The second hypothesis, about the magnitude of this association, was supported by the finding that only 9% of the variance of the GHQ could be accounted for by the disaster and recent life events. This confirms the strength of the association previously postulated as existing between life events and the onset of psychiatric disorder (Andrews & Tennant, 1978; Paykel, 1978). Thus a consensus arises from two quite different methodological approaches – one looking at the effect of multiple random life events (Holmes & Rahe, 1967; Tennant & Andrews, 1976; Paykel *et al*, 1971) and the other, employed in this study, looking at the impact of one threatening and destructive event.

There was also support for the last hypothesis examined: that major adverse life events have independent relationships with psychiatric disorder. Firstly, the effect of the disaster was independently associated with psychiatric impairment, not being 'partialled out' in the regression analysis by the contribution of threatening life events experienced before the fire. In other words, people who had experienced life events before the fire remained equally, if not more, susceptible than others to the emotional effects of the disaster itself.

Secondly, the impact of the disaster was sequential in time, the fire-fighters being exposed to the disaster before being in a position to establish their personal and property losses. All but 9% were unable to defend their own property, because they were fighting the fire elsewhere. Although exposure and perceived threat correlated with the GHQ score, the most powerful association was with property loss (Table II). It is probable that the ongoing stresses of property loss, such as loss of income for farmers, accounted for the strength of the association, which provides further support for an additive effect. Furthermore, the fact that the two variances (i.e. that accounted for by events experienced prior to the disaster and that accounted for by the disaster itself) were of a similar order of magnitude supports the view of Holmes & Rahe (1967) that major adverse life events have an additive and relatively independent effect.

This study demonstrates that investigating the psychological impact of a natural disaster provides an opportunity to examine the relationship between minor psychiatric disorder and a recent life event in the absence of several methodological problems often encountered in life event research (Paykel, 1983). This is because recall of a disaster is likely to be more consistent than that of other events, its impact is easier to document than that of a wide range of life events (Finlay-Jones, 1981), and a disaster is not a consequence of illness (Brown, 1974).

However, the current study has limitations that need to be considered in the interpretation of the data. Firstly, the questionnaire return rate was low, as is often the case in disaster research (Logue *et al*, 1981). However, the study sample was not skewed to any substantial degree. In particular, the fire-fighters studied and the comparison group did not significantly differ in their experience of the disaster or the prevalence of psychiatric morbidity, the two variables that would have suggested biased sampling. Secondly, the inventory used in this study has not been formally validated. However, there appeared to be face validity in its construction. For example, in the personal loss sub-scale the weighting was based on a carefully validated life events scale measuring these same losses (Tennant & Andrews, 1976). Formal validation can only occur following a disaster, and such events are infrequent. To this end, Green (1982) has called for careful reporting of quantitative methodology so that standardised check-lists can be developed for disaster research. The present paper is an attempt to begin the process.

Finally, it is difficult to know how typical this population was of the victims of the Ash Wednesday disaster. While it is reasonable to speculate that fire-fighters were significantly affected by exposure to the disaster, the study does not establish whether the same magnitude of association between experience of the fire and psychiatric impairment would occur in an unbiased community sample.

However, fire-fighters are of particular interest because, unlike others involved in disasters who, in general, attempt to protect themselves, most of these people were committed to facing extreme danger for hours on end, as well as risking serious injury and substantial personal and property losses. This would suggest that the relatively small association between psychiatric impairment and exposure to the disaster cannot be explained away by a relative absence of danger or loss.

Furthermore, the validity of these data is substantial because a number of potentially confounding variables, often present in disaster victims, were not operating. Two factors that can influence the reporting and prevalence of neurotic impairment among disaster and accident victims are compensation payments for psychological trauma and the neurological sequelae of head injuries (Lishman, 1973). The voluntary nature of the organisation to which the fire-fighters belonged, and the absence of head injuries among them, meant that neither of these potentially confounding variables was operating here.

In conclusion, the data suggest that the majority of the fire-fighters studied did not become cases, despite experiencing prolonged life-threatening stress, frequent injury, and property loss. This raises the possibility that psychiatric morbidity in a population affected by disaster can perhaps better be explained using a model where the disaster acts as a trigger to most of the individuals who are already vulnerable to decompensation (Hendin *et al*, 1983; McFarlane, 1984). If this proposition is correct, the exact prevalence of cases following a disaster may be more influenced by the prevalence of premorbid risk factors than by the severity of the disaster. Although the prevalence of psychiatric impairment in a population after a disaster may be substantial, such a trigger effect would lessen the size of any linear correlation between the disaster experience and psychiatric disorder if individual thresholds for decompensation were variable, as has been suggested by Paykel (1978). This may explain why only 4.9% of the variance of minor psychiatric disorder in this population could be explained by the intensity of the individual's disaster experience.

The small proportion of the variance of disorder successfully accounted for in this population raises the question of what factors may explain the rest of it. Some disaster literature has proposed that problems with mental health, where they exist, can be attributed to the destruction of kinship or social networks (Perry & Lindell, 1978). However, these firefighters had high levels of social support before, during, and after the disaster from within the firefighting organisation. Also, two studies of psychiatric morbidity among victims of this same bushfire disaster found little or no relationship between social support and the GHQ score (Hill, 1983; Wallace *et al*, 1985). Thus lack of social support did not appear to be a major factor contributing to psychological impairment.

Therefore, the other variables which may influence an individual's vulnerability to psychiatric morbidity following a disaster – such as genetic predisposition to psychiatric disorder, personality type, psychodynamic characteristics, and family relationships – all require further investigation. If preventive mental health services are to be provided after disasters, definition of the characteristics of those who are at risk of decomposition is of central importance.

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The Maternity Blues in Post-partum and Post-operative Women

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In order to establish whether the maternity blues is peculiar to the puerperium a self-rating blues questionnaire was administered to women after childbirth and after surgery. A period of dysphoria, similar in many respects to the maternity blues, was experienced by women post-operatively. The main difference between maternity and post-operative patterns of dysphoria seemed to lie in the timing of the blues, but on closer analysis many post-operative dysphoric symptoms peaked in a similar way to those in the maternity group. In the search for the cause of the maternity blues it may be helpful to study the same parameters in post-operative dysphoria.

The maternity blues is a poorly defined but common condition occurring in 50–70% of women in the early puerperium (Yalom *et al*, 1968; Pitt, 1973). The condition is characterised by transient periods of dysphoria, manifested by crying, feelings of depression and despondency, confusion, anxiety and insomnia (Robin, 1962; Kane *et al*, 1968, Yalom *et al*, 1968; Pitt, 1973; Priest, 1979). Detailed descriptions of the condition have been provided by Yalom (1968) and Stein (1982). The cause of this dysphoria is unknown, but it has been suggested that the rapid fall in sex hormones could be responsible. A fall of this magnitude is unique to the early puerperium. One way of testing whether or not the blues is caused by a fall in hormone levels would be to determine whether the blues syndrome is unique to the early puerperium. It is known that some dysphoria occurs after surgical operations. Raphael (1974) found that 60% of women experienced a bout of crying, sadness and uncontrolled distress 4–5 days after hysterectomy. However, more systematic information has not been reported regarding the frequency and nature of post-operative dysphoria.

The purpose of this study is therefore to determine whether a maternity blues-like syndrome occurs in post-operative women and, if so, to see if there is any characteristic pattern which distinguishes post-partum from post-operative dysphoria.

This preliminary study investigates and compares the manifestations and incidence of the blues between three groups of women: following childbirth, following major surgery and following minor surgery.

Method

Patients in the study comprised 37 puerperal women, 28 women after major surgery and 22 women after minor surgery. All were under the age of 50. Those delivered of a stillbirth or whose baby was seriously ill were excluded from the study, as were terminally ill post-operative patients. Apart from these exclusions, all women delivering or undergoing surgery over a short period of time were invited to take part in the study.

In the post-partum patients, 30 had been delivered normally and seven by forceps. No general anaesthetics were given. The mean age was 26.5 years (range 16–40).

The major operations were defined, arbitrarily, as surgery necessitating a stay in hospital for at least 48 h after operation and included hysterectomy, (two of the 11 hysterectomy patients also had bilateral oophorectomies), lumbar disectomy, lumbar sacral fusion and appendicectomy.

Minor surgery (necessitating a stay in hospital for less than 48 h after surgery), included dilatation and curettage, laparoscopy, meniscectomy, and arthroscopy. Terminations of pregnancy were not included. The mean age of the female post-operative patients was 37.7 years (range 17–50).

A blues-rating questionnaire (Stein, 1980) was completed by every respondent from days 2 to 6 inclusive, following delivery or operation. The questionnaire is a 13-category self-rating scoring system. Insomnia was added as an extra category. Patients discharged before the end of the observation week were asked to complete the questionnaire at home.

The distribution of scores was compared between the post-partum and surgical groups, using the χ^2 test of association. The numbers of patients experiencing each symptom were also calculated (as a percentage of the total number of patients in the group), on a daily basis in order to compare patterns of dysphoria between the groups throughout the week.

In-patients experiencing the blues were asked if they could identify the cause of their distress, how they were helped, whether or not they felt they could have been helped more, and by whom.

Results

A total of 110 women were asked to participate in the study; 87 completed sets of forms were returned giving a response

TABLE I
Comparison of score distributions between post-partum and post-operative groups

Symptom	*Major surgery*	*Minor surgery*	*d.f.*
Depression	1.42	1.38	2
Crying	0.50	13.56^{**-}	2
Anxiety	0.12	2.59	2
Tension	2.31	0.31	2
Restlessness	12.82^{**+}	1.35	2
Sleeplessness	0.83	8.47^{*-}	2
Tiredness	6.16^{*+}	8.92^{*+}	2
Dreaming	0.08	6.60^{*+}	2
Anorexia	47.22^{***+}	12.29^{**+}	2
Headache	0.60	0.86	1
Irritability	0.29	0.26	1
Lack concentration	25.02^{***+}	4.50^{*+}	1
Forgetfulness	5.33^{*+}	0.005	1
Confusion	0.18	0.35	1

*$P<0.05$; **$P<0.01$; ***$P<0.001$.
+ Symptom more severe than in post-partum group.
− Symptom less severe than in post-partum group.

rate of 80%. Non-respondents comprised 30% of the post-partum group, 5% of the major surgery group and 30% of the minor surgery group. A possible reason for the higher response rate in women having major surgery is that most were in-patients for the study week, facilitating return of their questionnaires.

Patterns of symptoms throughout the week

In the post-partum group the number of patients experiencing symptoms peaked on days 3 and 4, whereas the numbers in the surgical groups declined progressively throughout the week and did not show a peak (Fig. 1).

On examining the numbers of patients experiencing individual symptoms, however, it may be seen that many of the individual symptoms in the major surgical group peaked in a similar fashion to those of the post-partum group. The peaking phenomena were not so evident in the female minor surgery groups (Fig. 2).

Summary of comments made by women experiencing the blues

The woman could rarely give convincing reasons for crying; many were puzzled by their tears as crying for them was an unusual reaction. Some blamed their distress on somatic causes such as pain with perineal stitches or post-operative pain. Problems with the baby upset many post-partum patients and may have triggered the blues. Some women cried because of pleasant events; their tears came easily for a variety of reasons, not just because of unpleasant depressing experiences. Many women described a feeling of tearfulness building up through the day which culminated in an uncontrolled bout of crying at the arrival of visitors or some other event, such as news that the baby would have to undergo phototherapy because of neonatal jaundice.

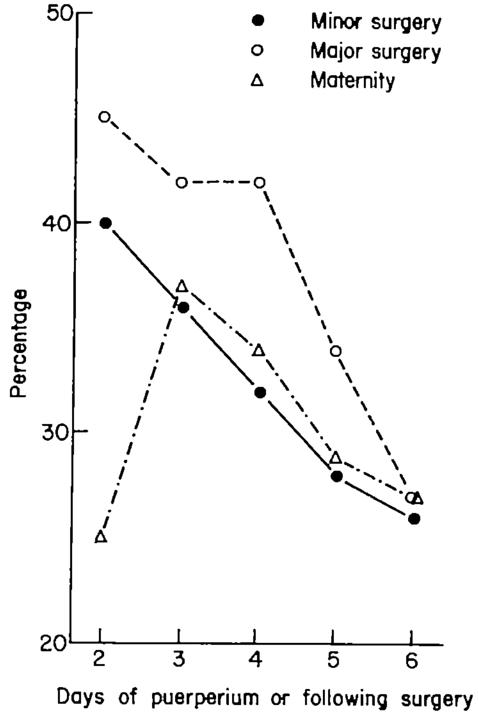

FIG. 1 Percentages of patients experiencing symptoms.

During her distress, every woman who experienced the blues wanted only her husband (or other close relative) with her, or else wanted to be left alone. Some said they felt foolish, as they could give no reason for their tears, and because of this preferred to shut themselves away to cry in private. No-one felt that the nurses or midwives could help, although in several cases they had tried.

Violent dreams featured in a few women's distress. These dreams involved danger and injury to themselves and their families.

Women in the post-partum and major surgical groups made very similar comments regarding their distress. No comment was peculiar to one group. No attempt was made to elicit comments from women discharged home before the onset of their distress, but a few nevertheless supplied comments that reflected those made by the women in the other groups.

Discussion

The present study has demonstrated that the dysphoria called the maternity blues is not unique to the puerperium. A similar type of dysphoria appears to occur in women following surgery; indeed there was slightly more dysphoria in the major surgical group than after childbirth. The only important difference between post-operative dysphoria and post-partum dysphoria appears to be in

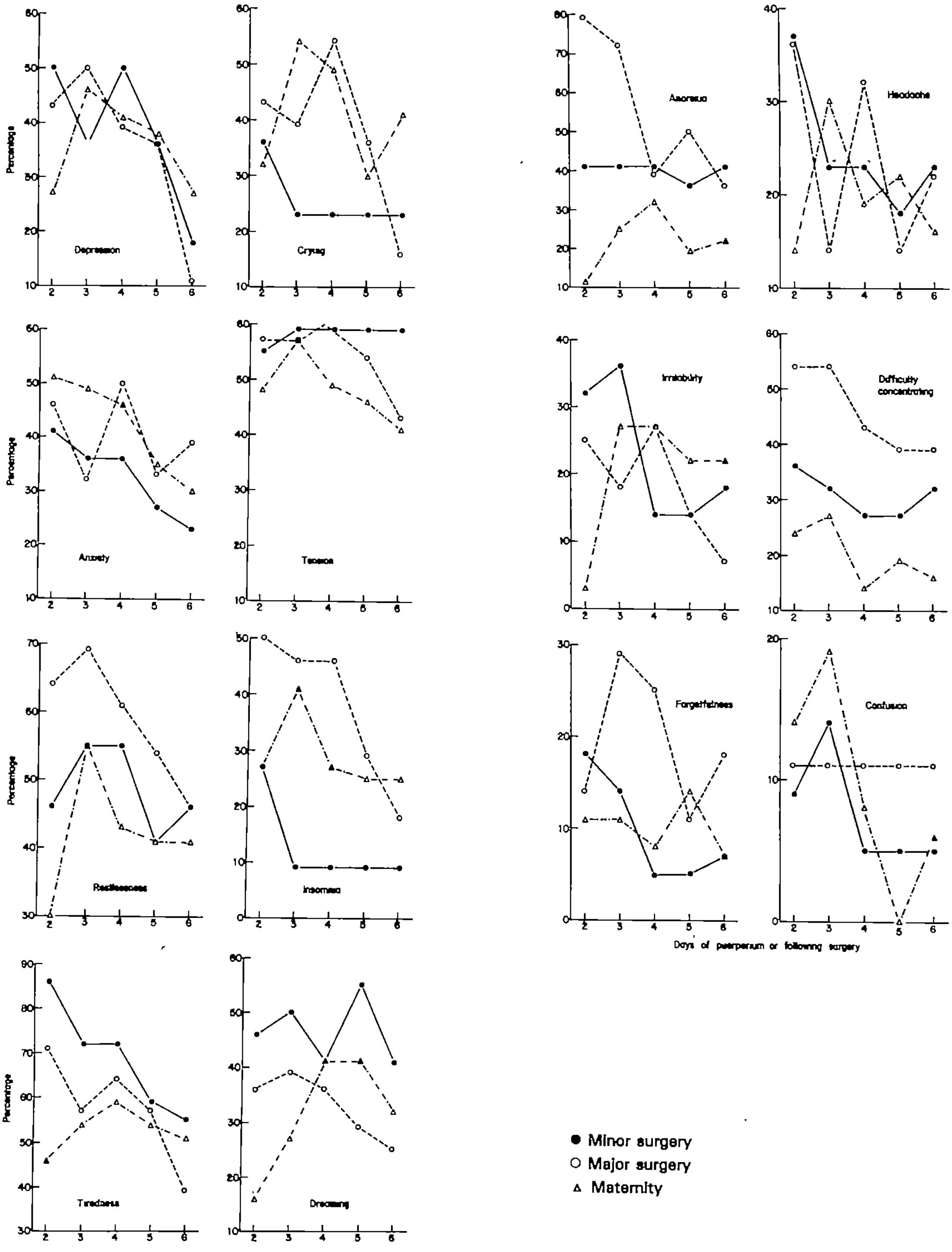

FIG. 2 Percentages of patients experiencing individual symptoms.

the timing. In this study it was found that post-partum dysphoria showed peaking on days 3 and 4, whereas post-operative dysphoria tended to be worse immediately after the operation and progressively diminished. This finding is consistent with those of Pitt (1973), Stein (1980), and more recently Kendell *et al* (1984). Kendell found that mood changes after Caesarean section peaked on day 5 in a similar fashion to those following vaginal delivery, but found no peaking following hysterectomy; instead, hysterectomy mood change ratings fell progressively from day 2 onwards. However, looking at individual symptoms in patients undergoing a wider spectrum of surgical operations, this present study shows that several symptoms (particularly crying and depression in women undergoing major surgery) peak in a similar way to the post-partum group, and indeed, many post-operative patients indicated by their comments that their blues occurred suddenly and unexpectedly at or around post-operative day 4. Differing patterns may perhaps be partially explained by the general physical discomfort experienced in the early post-operative days, particularly following major surgery, or alternatively by ward procedures. For example, surgical insomnia scores start at a high level and then decline through the rest of the week, whereas post-partum insomnia scores peak on day 3. This probably reflects the ward practice of 'rooming in' the mother with her baby from the second or third night onwards, thus making more difficult a good night's sleep for the mother.

In his study of 37 post-partum patients Stein (1980) found the peaking symptoms to be depression, crying, irritability, restlessness, dreaming and headache, whereas exhaustion, poor concentration and anorexia started at a relatively high level and declined throughout the rest of the week. This present study has identified symptom patterns similar in many respects to those described by Stein.

The cause of the blues remains uncertain, despite extensive biochemical investigation. An underlying assumption of these investigations is that the blues is a condition peculiar to the early puerperium, and that biochemical changes occurring after childbirth are therefore likely to be responsible. The present study suggests, however, that the blues is not unique to the puerperium and hence it is unlikely that the large drop in sex hormones is responsible. Cortisol is known to rise in pregnancy and post-operatively and has been implicated in mood changes. If post-operative dysphoria is precipitated by an increased output of cortisol, then it could be expected that major surgery, which causes greater adrenal stress, would result in a higher incidence of dysphoria than minor surgery, and this has been found to be the case.

Endorphins have also been implicated in mood changes (Koob & Bloom, 1983), and have been shown to increase during surgery (Cohen *et al*, 1981), and during pregnancy, and labour (Thomas *et al*, 1982). Newnham *et al* (1984), however, found no correlation between the post-partum blues and endorphin levels.

Whatever the precise cause of the post-partum blues, it is probable that the dysphoria is a reaction to a period of acute stress. Yalom *et al* (1968) mentions the similarity of the post-partum blues to the 'end phenomenon' – a dysphoric reaction noted after periods of intense stress experienced during, for example, paratroop training.

As this is an exploratory study, no attempt was made to control for the type of operation, delivery, length of stay in hospital or medication given, and this has probably affected the results to some extent. Nevertheless, the results of this study strongly suggest that the transient dysphoria commonly seen in the early puerperium and known as the maternity blues is also experienced by women a few days after surgery. In the search for the cause of the maternity blues, it may therefore be helpful to study the same parameters in post-operative dysphoria.

Newly delivered women and their families are often aware of the possibility of the maternity blues, but patients undergoing surgery are not. In view of the puzzlement and concern caused by the blues, it may be helpful to warn women undergoing surgery of the possibility of post-operative dysphoria.

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The General Health Questionnaire and the Detection of Emotional Disorder by General Practitioners

A Replicated Study

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This paper reports on a study of psychiatric morbidity in 920 patients attending 18 General Practitioners in Lewisham, South East London. Two indices of morbidity were computed on the basis of the patients responses to the General Health Questionnaire (GHQ) and the GPs' own estimates. Indices of the GPs' detection ability were found by comparing questionnaire and GP rating. The results are compared to those obtained by other workers in a sample of Manchester GPs. The GHQ gave a prevalence estimate of 42.9% which is comparable to that of 39.6% obtained in the Manchester study. In addition, the variation in caseness across sociodemographic groups was consistent with the Manchester findings. The GPs' estimates of morbidity, while much lower than those obtained in Manchester, have similar sociodemographic profiles. The results support the use of the GHQ as a screening device in epidemiological studies, and suggest the need for further investigation of the individual GP's performance as a case detector.

Over recent years the focus for epidemiological research in psychiatry has shifted from hospital to community, with resultant problems for definitions of caseness (Williams *et al*, 1980). This advance has produced a wealth of evidence which suggests that the bulk of psychiatric morbidity is seen at the primary care level and that only a small proportion of this is passed on to the psychiatric services. Statistics pertaining to this large group vary considerably depending on the case finding methods used (Hankin & Oktay, 1979). The reported rates of psychiatric illness show a great variation between individual physicians. Shepherd *et al* (1966), for example, showed a nine-fold variation of rates of reporting between GPs in their sample of London GPs, and Marks *et al* (1979) found that an average of 31.1% of patients were identified as 'psychiatric' with a range 3–77%. Importantly, however, less variation in morbidity is seen if screening tests or interview methods are used, suggesting that the variation of reported rates represents a large degree of observer bias. Furthermore, no correlation is found between the level of disorder reported by the GP and the level predicted by the screening questionnaire (Goldberg & Huxley, 1980).

This variation and discrepancy between GP and questionnaire measures has been shown to hold in other countries, for example Australia (Chancellor *et al*, 1977) and the USA (Hesbacher *et al*, 1975; Goldberg *et al*, 1982).

Studies investigating the sources of this inter-practitioner variation in reporting have identified factors in both the patient and the doctor. Shepherd *et al* (1966) explained 33% of the variance in terms of sociodemographic factors, in particular the number of patients joining and leaving the practice lists. Another important source was the doctors' attitudes, in particular those measured on a sub-section of the attitude questionnaire labelled 'psychosomatic score'. This score reflected the GP's assessment of the importance of psychological factors in the aetiology of listed diseases (e.g. asthma, peptic ulcer, cancer). Doctors who thought such factors important tended to report high numbers of psychiatric cases. Marks *et al* (1979) identified a number of patient stereotypes that alert the GP to the possibility of psychiatric illness, notably the separated and widowed and individuals seen by the GP several times previously. They also found the interview style and personality of the practitioners to be factors of importance in detection ability. The Australian study of Chancellor *et al* (1977) identified sociodemographic variables which were congruent with questionnaire measures (separated, widowed and divorced) while women over 45 years of age were over-diagnosed and men in the same age range under-diagnosed. The US study of Goldberg *et al* (1980) was an attempt to replicate Marks *et al* (1979) and found overall similar results with regard to doctor factors, however, there were some specific differences which were thought to be due to the new measures introduced into the US study.

In the detection studies mentioned above (with the exception of Shepherd *et al* (1966)) the criterion against which the GP's performance was judged was the patient's score on the General Health Questionnaire (GHQ) (Goldberg, 1972).

The GHQ was designed as a screening instrument for non-psychotic disorders and has, to date, been used in a wide range of studies. Its validity in general practice populations appears to be well established (Goldberg, 1978). However, the performance of GPs as case detectors relative to the GHQ requires further investigation. The Manchester study of Marks *et al* (1979) identified several patient factors that affected case detection by GPs. It is not known whether the factors generalise to other GPs in different parts of the UK. That is, do patient stereotypes underlying detection represent rigid stereotypes or do they vary among different groups of GPs?

The present study is part of a larger investigation designed to evaluate a community psychiatric clinic, the Mental Health Advice Centre (Bouras & Brough, 1982). One facet of this investigation was to estimate the prevalence of minor psychiatric morbidity and the detection abilities of a selection of GPs who refer patients to the clinic. It is this aspect of the investigation that will be reported in this paper.

The aims of the present report are: to report on the probable prevalence of emotional disorders as measured by the GHQ in general practice; to compare the detection of emotional disorders in two groups of GPs in different parts of the UK; and to assess whether sociodemographic variables associated with case detection represent rigid stereotypes.

Method

The survey took place in Lewisham, South East London. Thirteen General Practices were approached whose surgeries lay within the geographically defined catchment area of the Mental Health Advice Centre (MHAC). Eight practices agreed to take part. No attempt was made to obtain a random sample of GPs. The practices were chosen solely on the basis of their location as the study was designed to provide a description of the practices whose patients were potential referrals to the MHAC.

The design represents a partial replication of the investigation reported by Marks *et al* (1979).

The survey was carried out between October and December 1983. Each practice was surveyed for a period of 1 week. Consecutive patients between the ages of 16 and 65 years who attended the surgeries to see their GPs were asked by the author to complete the 28-item GHQ before seeing the GP. Sociodemographic data was also collected from the patient. Patients who were not attending for 'illness' reasons (e.g. innoculations, insurance examinations, routine ante-natal visits) were not approached. The GP was asked to complete a simple form after seeing the patient on which s/he rated the patient's degree of emotional disturbance on a 6-point scale ranging from 0 - no disturbance to 5 - severe disturbance. This scale was identical to that used by Marks *et al* (1979). Each GP was instructed by the author in the use of the scale. The GPs were not aware of any patient's GHQ scores during the week of the study.

The two ratings obtained from the GHQ and GP scale were used to compute the four main dependent variables which were:

(a) Conspicuous psychiatric morbidity (Kessel, 1960). This refers to the percentage of patients rated by the GP as psychiatrically ill. On the 6-point scale it represents a rating from 2–5 inclusive.
This gave the index of GP caseness.

(b) Probable prevalence of psychiatric illness. This represents the proportion of patients who obtain a high score on the GHQ after a correction term has been applied. The correction term is given by the formula:

$$\text{Probable prevalence} = \frac{HP - fp}{s - fp}$$

Where:
$HP = \%$ with high scores; $fp =$ false positive rate $= 1 -$ specificity; $s =$ sensitivity.
In the case of GHQ-28, a cutting score of 4/5 was used. At this cutting score in general practice settings the questionnaire has a sensitivity of 85.6% and a specificity of 86.8% (Goldberg, 1978).

(c) Spearman's correlation coefficient (ϱ). This was computed by a comparison of the GP's ratings and the GHQ score. This statistic can be used as a measure of the accuracy of the GP's assessment, where accuracy refers to the overall ability of the doctor to make assessments of psychiatric disturbance which are congruent with the patient's symptom levels.

(d) Identification index. This is a measure of the ability to identify symptomatic patients as sick and ignores the ability to identify symptomatic patients as normal. As such it is an assessment of the physicians performance as a case detector it can be found using the formula:

$$\text{Identification Index} = \frac{\text{Number of patients identified as ill by GP and GHQ}}{\text{Number of high scorers (GHQ)} \times \text{sensitivity}}$$

In addition to these, 'bias' was calculated for each GP, this represents the tendency of the physician to make or avoid making psychiatric assessments. It is computed by comparing the conspicuous psychiatric morbidity to the probable prevalence:

$$\text{Bias} = \frac{\text{CPM}}{\text{Probable prevalence}}$$

This was not reported by Marks *et al* (1979) but a figure for their overall sample of GPs is available in Goldberg & Huxley (1980). Since the CPM is generally not correlated with probable prevalence it can be considered to be largely a measure of bias. Thus, in this report, as in Marks *et al* (1979), CPM will generally be used as the measure of bias.

Results

Eight General Practices took part. They consisted of 18 GPs–six female and twelve male – in three single-handed and five group practices.

In the specified age group 983 patients attended the surgeries. Of these 63 were missed by the researcher during the surgery time. Therefore, some information was obtained on 920 patients. Of the 920 patients, 44 did not complete the GHQ. Reasons for not completing the questionnaire were: refusal (21), language difficulties (9), handicapped (11). Three questionnaires were incomplete.

The low rate of refusal confirms the acceptability of the GHQ to patients (Goldberg, 1972).

GP ratings were obtained on 809 of these patients. Some of the patients given the GHQ attended a trainee in one of the practices who did not take part in the ratings. The GHQ ratings obtained on these patients where included in the computation of the overall probable prevalence.

The overall results for the Lewisham doctors are shown in Table I where they are compared to those reported in the Manchester study. It is important to note that the overall probable prevalence of 42.9% is comparable to that of 39.6% reported in Manchester. However, the Lewisham GPs consider only 19.3% of the patients to be cases while the Manchester GPs were more liberal in their estimation (31.1%). The present results confirm the widely noted observation that GPs vary considerably in their detection abilities and provide an unreliable index of morbidity in epidemiological studies. The use of a standardised technique, however, appears to give comparable results.

The most striking differences between the detection abilities of the two samples are the lower bias and identification indices of the Lewisham GPs. The accuracy of both samples is similar. It is likely that the low identification index is a secondary effect of low bias since for a given level of accuracy as bias decreases the number of symptomatic patients correctly identified as cases will correspondingly decrease.

Correlations between the variables are shown in Table II. They are generally higher than those obtained in the Manchester sample. While the most obviously different correlation is that between Rho and CPM, in the Lewisham sample this correlation does not attain statistical significance. If bias is used instead of CPM, the correlation rises to 0.42 but remains non-significant. As with the Manchester study there is no correlation between probable prevalence and CPM. In both groups of doctors there is a moderate correlation between accuracy and identification index and a high correlation between identification index and CPM. In the Lewisham sample if bias is used rather than CPM, this last correlation rises to 0.95.

These correlations then, are similar and the present results confirm the lack of agreement between estimates of morbidity provided by screening questionnaires and by physicians. In addition they suggest that while the Manchester and Lewisham doctors are different in their detection abilities the relationship between the detection indices is similar in both groups.

The remaining results will concentrate on the socio-demographic characteristics of the patients and their effect on the four main variables (summarised in Table IV). At each stage a comparison will be made with the Manchester data.

TABLE I
Overall results

	Mean	*s.d.*	*Range*
Present Study – 19 GPs – 920 attenders			
Conspicuous psychiatric morbidity	19.3%	12.0	0.00–36.6%
Probable prevalence	42.9%	14.00	10.7–62.6%
Proportion identified by both GP and GHQ	13.7%	8.2	0.00–30.0%
Accuracy (Spearmans correlation coefficient)	0.35	0.19	−0.06–0.73
Identification index	0.36	0.24	0.00–0.85
Bias	0.62	0.76	0.00–3.30
Marks et al (1979) – 91 GPs – 4098 attenders			
Conspicuous psychiatric morbidity	31.1%	14.2	3.00–77.0%
Probable prevalence	39.6%	11.4	15.00–64.0%
Proportion identified by both GP and GHQ	19.0%	8.9	26.00–50.2%
Accuracy (Spearmans correlation coefficient)	0.39	0.15	0.09–0.66
Identification index	0.54	0.19	0.08–1.05
Bias[1]	0.79	Not reported	

1. Reported in Goldberg & Huxley (1980)

TABLE II
Correlations of main variables – comparison of Manchester and Lewisham studies

Correlated variables	*Marks et al (1979)*	*Present study*
Probable prevalence/ Conspicuous psychiatric morbidity	-0.17^1	-0.16^*
Spearmans Rho/Conspicuous psychiatric morbidity	0.08	0.36^*
Spearmans Rho/probable prevalence	0.03	-0.17^*
Spearmans Rho/identification index	0.4	0.54^{**}
Identification index/ conspicuous psychiatric morbidity	0.72	0.89^{***}

$^*P$ – NS; $^{**}P<0.05$; $^{***}P<0.001$.
1. Reported in Goldberg & Huxley (1980)

Sex

The results on this variable are summarised in Fig. 1. As in the Manchester study doctors perceive more illness in female patients (20.4%) than male patients (16.5%). This does not reach statistical significance ($\chi^2=1.52$, $P<0.2$). Although female doctors report fewer cases than male doctors, these differences do not attain statistical significance.

The GHQ confirms the finding of the Manchester study that there is probably more psychiatric illness among female attenders ($\chi^2=4.05$, $P<0.05$). As with the Manchester study this is only true for those women attending male doctors ($\chi^2=5.98$, $P<0.02$). Marks *et al* (1979) explained this finding by the selective recruitment of symptomatic men to female doctors. However, this is not the whole picture in the present study. In the Lewisham practices there is a significantly higher probable prevalence in women consulting female doctors than those consulting male doctors ($\chi^2=4.48$, $P<0.05$). The Manchester study found no significant differences in probable prevalence between female patients consulting male doctors and patients of either sex consulting female doctors. Further to this, it was also found that in the Lewisham practices significantly more symptomatic males consult female doctors ($\chi^2=8.65$, $P<0.01$). Thus, more symptomatic people of both sexes consulted female than male GPs in the present sample ($\chi^2=9.75$, $P<0.01$).

This finding is highlighted by the additional observation that proportionately many more women consult female rather than male GPs (80.5% vs 57.5%) and consequently proportionately less men. Overall, 79% of consulting men see male GPs. This suggests a real recruitment of symptomatic males to female GPs and in this way confirms the Manchester finding.

A possible explanation for this is that symptomatic patients may be avoiding doctors with low bias. Table IV shows the relationship of the GPs bias to high and low prevalence situations. It appears that high prevalence is associated with a low bias which is contrary to that expected. However, all the female GPs have a low bias and high prevalence. When the female GPs are removed from the contingency table the relationship between bias and prevalence disappears ($\chi^2=2.1$, NS). Thus, the relationship between bias and prevalence for the whole sample of doctors is explained by the association of high prevalence and low bias in female GPs. This suggests that the recruitment of symptomatic patients to female GPs is due to the sex of the GP and not the GPs bias.

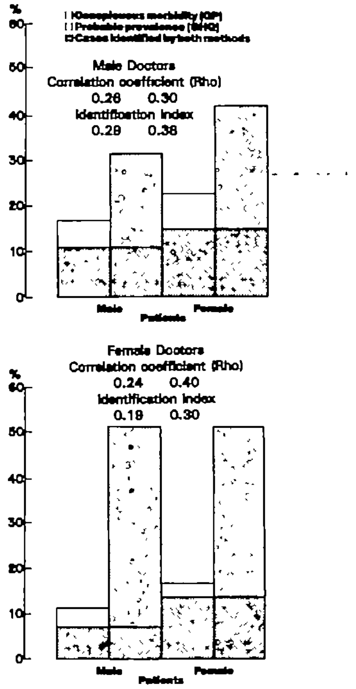

FIG. 1 The effect of sex of the doctor and sex of the patient on the four main variables.

The findings for Rho and identification index only partly confirm those of Marks *et al* (1979). As with the Manchester study identification index is lower for male patients than for female patients irrespective of the sex of the doctor. None of these figures attain statistical significance in the present sample. Contrary to the Manchester sample, overall accuracy is lower for male patients than female patients irrespective of the sex of the doctor. Again these findings do not reach statistical significance.

Age

GP cases show a peak in middle age ($\chi^2=19.43$, $P<0.01$). This is consistent with the probable prevalence, but this also shows a peak in younger age groups ($\chi^2=27.98$, $P<0.001$). This is similar to that found by Marks *et al* (1979). As with the Manchester study identification index reaches its highest values in middle age.

TABLE III
The effect of sociodemographic variables on the four main variables

	Conspicuous morbidity (GP): %	*Probable prevalence (GHQ): %*	*Case identified by both methods: %*	*Identification index: %*
Sex				
Male	16.5	38.5	10.0	0.26
Female	20.4	45.6	15.2	0.35
Age				
16–19 years	10.6	42.5	6.5	0.16
20–29 years	12.8	49.3	11.1	0.24
30–39 years	12.2	40.6	17.4	0.41
40–49 years	27.9	51.8	19.7	0.41
50–59 years	24.3	39.9	13.2	0.34
60–65 years	14.3	20.6	7.5	0.27
Employment status				
Full time	15.0	39.6	10.0	0.25
Part time	22.5	49.3	19.6	0.41
Unemployed	26.0	70.5	20.8	0.34
Retired	15.6	36.1	10.7	0.28
Student	13.9	32.1	11.1	0.35
Housewife	29.2	41.2	19.6	0.46
Marital status				
Single	18.3	48.5	13.4	0.30
Married	15.9	37.0	10.5	0.27
Separated	45.0	74.5	40.0	0.65
Divorced	24.1	42.9	17.9	0.44
Widowed	34.8	44.6	27.3	0.68
Age left school				
Before 15 years	28.7	43.8	17.8	0.42
15 years	24.0	59.4	19.9	0.38
16 years	16.2	46.0	11.1	0.25
After 16 years	14.3	33.2	10.4	0.29
Socioeconomic class				
I	25.0	42.0	15.0	0.34
II	15.5	33.9	10.6	0.28
III	17.1	43.1	12.8	0.31
IV	25.0	48.8	16.5	0.37
Presence of dependents				
Children under 5 years	18.1	45.7	16.4	0.37
Children 15–16 years	27.2	44.7	17.4	0.40
Elderly adults	23.5	47.4	23.5	0.51
Nationality				
British	19.6	43.8	13.5	0.32
Eire	26.9	65.4	25.0	0.45
West Indian	10.3	40.4	9.7	0.23
African	11.8	51.4	5.9	0.13
Indian	7.7	3.3	0.0	0.0
Other	20.0	27.6	16.7	0.49

In both the Manchester and Lewisham samples, the GHQ shows a tendency to identify more cases than the GP in all age groups. This is especially marked in patients between 16–30 years. It is least marked in those patients over the age of 60 years.

Employment status

The rates for psychiatric illness is highest among the unemployed ($\chi^2=24.6$, $P<0.001$). However, this is not reflected in the GP ratings who apportion the highest caseness to housewives ($\chi^2=22.76$, $P<0.001$). Other occupational groups are not significantly different from chance expectancy on either index. In the Manchester sample the unemployed were the largest group on both indices.

Discrete differences are seen with regard to identification index for this sociodemographic group in the Manchester and Lewisham samples. In the present sample, identification index is highest for housewives and lowest for those in

TABLE IV

Relationship of bias and prevalence in total sample of GPs

		Bias	
		High	*Low*
Probable prevalence	High	2	9
	Low	5	2

$\chi^2 = 5.15$, d.f. = 1, $P < 0.05$.

TABLE V

GP/GHQ Caseness and employment status

	Males		*Females*	
	Conspicuous morbidity (%)	*Probable prevalence (%)*	*Conspicuous morbidity (%)*	*Probable prevalence (%)*
Full time	16.4	35.0	13.8	43.1
Part time	0^1	35.5^1	23.4	50.0
Unemployed	22.2	62.2	30.4	78.4
Retired	0	24.2	23.8	43.8
Student	8.3	51.9	16.7	20.8
Housewife	—	—	29.2	44.2
	$\chi^2 = 5.14$	$\chi^2 = 8.85$	$\chi^2 = 18.19$	$\chi^2 = 22.4$
	NS	NS	$P < 0.001$	$P < 0.001$

1. $n = 5$

full time employment have similar rank orderings in both samples.

If the data are broken down by sex (Table V) the higher female rates are not accounted for by the idea that housewives are more symptomatic than unemployed women and therefore inflate the female mean. A female patient giving her employment status as unemployed is much more likely to be a GHQ case than one who is a housewife (78.4% vs 44.2). This result is in keeping with Manchester study. The effect of employment status on GHQ caseness appears to be due to the high percentage of unemployed women who were symptomatic.

Marital status

The conspicuous morbidity is highest in those married but living apart from their spouses ($\chi^2 = 21.575$, $P < 0.01$). The rank order of the remaining groups is widowed, divorced, single and married. There is no significant difference between these last four groups. The GHQ while confirming the high rates of disorder among the separated ($\chi^2 = 19.623$, $P < 0.001$, produces a different rank order of separated, single, widowed, divorced and married. There is no significant difference in the last three groups. Thus the GPs, while correctly attributing high rates of disorder to people separated from their spouses tend to underdiagnose illness in single people. This is reflected in the high identification index found in the separated groups and the low identification index in single people and those married but living together.

These results are in line with the Manchester study in finding high rates among the separated group for GP and GHQ based indices.

Age left school

The conspicuous psychiatric morbidity falls from 28.7% in those who left school before 15 years of age to 14.3% in those who left school after the age of 16 years. This difference is greater than expected by chance ($\chi^2 = 16.106$, $P < 0.01$). The GHQ partly confirms this trend. The Manchester study used the variable of 'age left full time education' and found highest rates for CPM and probable prevalence in those with minimum education.

Socioeconomic class

This variable was not directly measured by Marks *et al* (1979). Rather, they used 'age left full time education' as an approximate indicator of social class. For the present study the Registrar General's classification was used, the social class of single women being based on their father's occupation. Subjects in social class V were too few to be included in the analysis. Conspicuous morbidity was highest in social classes I and IV (25% respectively) falling to 15.5% (Social class II) and 17.1% (Social class III). These differences were not significant ($\chi^2 = 3.43$, $P < 0.4$). The GHQ does not confirm this trend and again no significant differences were found ($\chi^2 = 6.41$, $P < 0.1$). No large differences were seen for identification index across social classes.

Dependents

This variable was not measured in the Manchester study. The presence of dependents did not affect the reporting of cases by the GPs. In each of the three classifications of dependents, there was no significant difference in conspicuous morbidity between those patients who had, and those who had no dependents. The GHQ reflected the GPs' judgements as there was no greater degree of GHQ caseness in those with dependents and those without.

The percentage of patients classified by both GHQ and GP as cases was highest in those who had elderly adults as dependents. In this group the proportion classified by both methods was identical to the CPM thus all reported cases were correctly identified. The identification was highest in those who had elderly adults as dependents.

National origin

This variable was not reported in the Manchester study. Conspicuous morbidity is highest among people born in the Republic of Ireland and lowest in those born in India. While probable prevalence is highest in the Irish, thus reflecting the CPM , it is also high among Africans who have a relatively low CPM. For neither CPM nor probable prevalence do the differences across national groups reach statistical significance.

The identification index is highest in those born in Eire and in those of miscellaneous national groups. The low values obtained in those of African and Indian origin may reflect the small number of these people in the sample ($n = 17$ and 13 respectively).

Discussion

Performance of the GHQ

One aim of the present paper is to report on probable prevalence of emotional disorders as measured by the GHQ and to compare this across two General Practice settings. This has been addressed by focusing on the number of probable cases detected and their sociodemographic composition. The study of Marks *et al* (1979) has been used for comparison as it was considered that the urban/semi-urban settings of Greater Manchester was comparable to that of Lewisham, and both studies have used a composite picture from a variety of general practices.

The GHQ was designed as a screening instrument and, as such, should properly be used as part of a two-stage procedure to select people for further interviewing (Goldberg, 1972). However, for certain purposes, such as for obtaining an estimate of psychiatric morbidity, it may be justifiable to use the GHQ alone. This one stage design should only be used in populations where the validity of the questionnaire is known and when the GHQ is used on new populations it should be revalidated (Goldberg, 1978). In general practice samples, the GHQ could be used alone as validity indices have been established and are generally acceptable (Goldberg, 1978). Clearly, whether a one-stage or two-stage design is used in such populations will depend on the purposes of the study.

Table VI shows the figures for probable prevalence obtained by use of the GHQ in several general practice studies and compares them to the conspicuous psychiatric morbidity as rated by the GP. The GHQ produces a wide variation in prevalence estimates but the estimates are obtained in a variety of settings and even when obtained in comparable settings (Goldberg *et al*, 1976; Marks *et al*, 1979; Skuse & Williams, 1984) the number of GPs from which the estimates were derived varied. It is of interest that Marks *et al* (1979), using several Manchester GPs obtained an almost identical figure to Schein (1977) in a sample of Philadelphia family practitioners. The lower figure for probable prevalence produced by Hoeper *et al* (1979) is probably due to the use of a different criterion of caseness than the other studies. Hoeper *et al* used the research diagnostic criteria (Spitzer *et al*, 1978) and Schedule for Affective Disorders and Schizophrenia (Endicott & Spitzer, 1978) while the other studies based their criterion on the Clinical Interview Schedule (Goldberg *et al*, 1970).

In the present study, by taking consecutive attenders at eight General Practices, an estimate of the prevalence of minor psychiatric morbidity was made and this proved to be comparable to that obtained in the Manchester study. The variations seen in GHQ cases across sociodemographic groups were remarkably consistent with the results obtained by Marks *et al* (1979). Marks *et al* compared their results to those of Shepherd *et al* (1966) and, with the exception of social class, showed them to be similar. Unfortunately social class was not measured directly in the Manchester study but instead, time spent in full-time education was used as an indirect measure. The present study used both variables and found that, while those people with a minimum education showed a high probable prevalence and conspicuous psychiatric morbidity there was no effect of social class. In the present sample age left school and socieconomic class were fairly strongly correlated ($\gamma = -0.509$) suggesting that education level provides a more sensitive index of morbidity than social class. The findings for social class obtained in this sample agree with those of Shepherd *et al* (1966).

The similarity of the results obtained for probable prevalence and sociodemographic distribution of cases in the Manchester and Lewisham samples serves to emphasise the reproducibility of results using the GHQ. It is not felt that use of different versions of the GHQ in the two studies had an effect

TABLE VI
Summary of data from studies using GHQ and GP ratings

Study	*Country*	*Number of physicians used*	*Version of GHQ used*	*Sample size*	*Probable prevalence: %*	*Conspicuous morbidity: %*
Marks *et al* (1979)	UK	91	60 item	4098	39.6	31
Goldberg *et al* (1976)	UK	1 practice	60 item	365	29.7	36.7
Skuse & Williams (1984)	UK	1	60 item	272	38.9	24.3
Schein (1977)	USA	27	30 item	4086	39.5	17.6
Hoeper *et al* (1979)	USA	Not reported	30 item	1072	21.2	Not reported
Chancellor *et al* (1977)	Australia	18	30 item	1301	51.7	14.3

on the results obtained as both have very similar validity profiles when used in general practice samples (Goldberg, 1978).

Performance of the General Practitioners

The most striking differences between the detection profiles of the Lewisham and Manchester GPs is the tendency of the Lewisham doctors to report less cases than their Manchester counterparts. The lower identification index of the Lewisham GPs is a secondary result of this lower bias as the accuracy in both groups of doctors is almost identical. The correlation between the detection indices are similar for both samples suggesting that the relationship between the indices remains stable in different groups of GPs with varying profiles. In both groups of doctors, accuracy is not predicted by either the GPs perception of the level of disorder or the estimate made by the GHQ. However, the ability to make accurate assessments is partly predicted by the ability to identify true positives and this later ability is strongly related to the tendency to make (or avoid making) psychiatric judgements. The differences in overall bias of the two groups suggests that the Lewisham doctors have a different conception of what constitutes a psychiatric case, adopting possibly more stringent criteria before reporting a patient as emotionally disturbed. This may reflect their different attitudes towards psychiatry and differences in their interview skills.

A surprising result is the finding concerning recruitment of symptomatic patients to female GPs. A study carried out in West Philadelphia found that high probable prevalence was associated with high bias (Schein, 1977). In addition the studies of Marks *et al* (1979) and Goldberg *et al* (1980) found doctors with a high bias to be 'interested in psychiatry', sensitive to verbal cues of psychological distress and to ask many questions of a psychiatric nature. It was thus expected that the patients with emotional distress would avoid GPs with low bias, however, the opposite was found. This contrary finding is explained by the low bias of the female GPs. It appears that symptomatic patients are attracted to women doctors in spite of their low bias. Perhaps these patients take their complaints to female GPs in the expectation that they may be more amenable despite them not recognising their psychological disorder. A further possibility is that these patients do not wish to be labelled as psychologically disordered preferring to receive treatment for their physical complaints. It is not possible to examine these possibilities with the present data.

As with the Manchester GPs, the Lewisham doctors, on the whole, confirmed the variations in probable prevalence as measured by the GHQ. In the examination of GP performance as case detector, the identification index provides the most important measures of detection as it is a measure of the ability to identify symptomatic patients as ill. Furthermore, computed in the manner used in this paper, it is not affected by prevalence.

It is likely that certain signals must alert GPs to the possibility of psychiatric illness. The signal indicators measured in the present study were the sociodemographic variables. High identification indices were obtained for women, middle-aged patients, housewives, the separated and widowed, patients with minimum education, the Irish and patients with elderly people as dependents. This profile can be seen to produce a positive stereotype almost identical with the perceived by the Manchester GPs. The construction of this stereotype is on the whole, justified by the high prevalence in these groups. Two notable exceptions are those of the widowed and of housewives. While the increased vigilance for widow(er)s may be understandable in terms of the attention paid to this group in the literature, for housewives the increased vigilance may have a more dubious basis perhaps relating to drug advertising and other media portraying this group as neurotic stereotypes (Stimson, 1975).

Other groups appear to constitute negative stereotypes for Lewisham GPs; these are men, young age-groups, single and married patients, those in full-time employment and patients of African and Indian origin. Again, these generally match the low identification groups found in the Manchester study.

The present study then, creates a similar picture for Lewisham and Manchester GPs' stereotypes. The positive stereotype is of the middle-aged housewife, with minimal education, separated from her husband, who may be looking after elderly relatives. This is in distinction to the negative stereotype of the young educated bachelor or recently married man in full-time employment.

Conclusion

The probable prevalence of emotional disorder and its sociodemographic distribution was found to be similar in Manchester and Lewisham general practices. The estimates of morbidity by the two groups of GPs however, are widely different with the Lewisham GPs providing a more conservative estimate. This reinforces the well-recognised finding that practitioners estimates of morbidity are an unreliable measure in epidemiological studies. However, the GHQ can provide more stable results. This is not to argue for abandoning a two-stage approach as suggested by Goldberg (1972), rather, it supports the use of the GHQ as a screening device for epidemiological studies.

The finding of a recruitment of symptomatic patients to female GPs requires further investigation. This finding has implications for the management of emotional disorders in general practice as it may mean that some patients with these disorders avoid situations where their disorder may be acknowledged.

The GPs' performances as case finders, as measured by identification index supports the contention that, when broad sociodemographic categories are used as measures of signal strength, the GPs use generally similar cues in their conception of case stereotypes. This suggests that stereotypes used by GPs may be generally rigid and may be generalisable across practitioner groups. The use of patient cues as indicators of GP caseness deserves further investigation as the cues utilised in the consultation will also be part of a GP's construction of a case and will be directly utilisable in increasing case detection by GPs. This may be usefully addressed by analysing the differences between GHQ and GP cases using patient variables. This requires a more detailed study than was undertaken here possibly involving analyses of doctor-patient interaction and follow-up of the patient using a two-stage design.

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Ageing and Recovery from Alcoholism

G. NORDSTRÖM and M. BERGLUND

Fifty-five dependent (DSM-III) male alcoholics, chosen for good social adjustment, were personally followed up about 20 years after their first admission to hospital. The conditions of all the alcoholics were evaluated with a multidimensional diagnostic schedule when first admitted. At the follow-up, 11 were abstainers, 11 reported a change to social drinking via abstinence, and 10 reported a change to social drinking by gradual stages. Improvement after the age of 45 was mainly due to gradual change to social drinking. This type of improvement was in half of the cases attributed to a decreased tolerance for alcohol. In the present sample, there were no indications of any relationship between premature ageing and gradual change to social drinking. It is hypothesised that physiological processes of normal ageing with concomitant changes in reactions to alcohol may be of importance for remission from alcoholism in middle-aged and older alcoholics. The relationships of personality, social factors and the degree of alcohol-dependence to patterns of successful long-term adjustment are discussed.

Though several long-term follow-up studies of alcoholics have shown that a substantial proportion of the survivors eventually improve (Ciompi & Eisert, 1971; Öjesjö, 1981; Vaillant, 1983), knowledge about how processes of improvement evolve in relation to age is still limited.

Data from some general population and panel studies have indicated that many young problem drinkers, though they may have suffered quite severe social consequences of their abuse, tend to improve in their late 20s or early 30s (Fillmore, 1975; Saunders & Kershaw, 1979; Fillmore & Midanik, 1984). This type of improvement has been attributed to increasing maturity (Saunders & Kershaw, 1979; Fillmore & Midanik, 1984).

Other studies have indicated that alcoholism rates decrease with increasing age. Åmark (1951) found that the number of alcoholics admitted to treatment facilities decreased after the age of 50. Drew (1968) put forward the suggestion that alcoholism may be a 'self-limiting disease', with a tendency to resolution after the age of 40. In a personal follow-up of aged alcoholics in Switzerland, Ciompi and Eisert (1971) found that more than one-third of the subjects over 65 years of age were abstainers or moderate drinkers, improvement sometimes occurring after decades of heavy drinking. In a general population survey, Cahalan & Room (1974) found that the frequencies of different alcoholism symptoms were lower in subjects in their 50s and 60s. From other epidemiological studies, however, the frequency of alcoholism has been reported to change very little with ageing (e.g. Öjesjö, 1980; Mulford, 1982).

Ciompi and Eisert, as well as Drew, hypothesised that different biological, psychological and social aspects of ageing might be responsible for the amelioration of drinking habits with age. In the present paper, which concerns a follow-up, after about 20 years, of hospital-treated male alcoholics chosen for good social adjustment, we address the issue of ageing and recovery from alcoholism. We investigate whether there are any specific patterns of alcohol-adjustment or processes and mechanisms of improvement in alcoholics who improve when older compared with those who improve when younger. The possible relationships of background characteristics to such patterns and processes is studied.

Method

The sample

The present sample of alcoholics constitutes a subsample of a series of 1312 alcoholics who were first admitted to the Department of Psychiatry, University Hospital, Lund, Sweden, between 1949 and 1969. During these years the senior psychiatrists at the department rated their in-patients at discharge using a multidimensional diagnostic schedule developed by Essen-Möller (1961, 1971; Essen-Möller & Wohlfarth, 1947). The schedule consisted of one symptomatic or syndromatic part and one aetiological part. Among the aetiological items was 'chronic alcohol intoxication'. Those who scored positive on this item were included in the study, if they were first admitted as in-patients during

1949–1969 and lived in the county. The series has been reported on in previous publications (Berglund, 1984*a*,*b*, 1985). In order to obtain a group where a large proportion of the subjects had solved their alcohol problems, health insurance records were used. It has been shown that health insurance data relate strongly to the overall adjustment of alcoholics (Berglund, 1980). There were 324 patients fulfilling the following criteria: (a) male sex; (b) born 1916 or later; (c) first admitted 1949–1967; and (d) living in the county of Scania on 1 January 1981.

(A further 141 patients who fulfilled criteria (a) to (c) had died before 1981 while living in Scania (71 cases violent death, 70 cases other causes of death).)

Female patients were excluded to obtain a more homogenous sample. Patients born before 1916 were retired because of old age when the sample was followed up by health insurance data on 1 January 1981 and thus were not eligible for the present study, since the number of sickness cash benefit days was used as an inclusion criterion (see below). Patients admitted after 1967 were excluded in order to ensure an observation time of at least 15 years at the personal follow-up. The geographical delimitation was set for practical reasons. Out of the 324 patients, 70 (22%) fulfilled our criteria for good social adjustment, i.e. having at most 30 sickness cash benefit days during 1979 and 1980 together and a sickness cash benefit of at least 50 SEK/day. The figure of 30 sickness cash benefit days was the estimated unpublished median value for the male population of Scania for the years 1979 and 1980 together. The official mean value was 36.5 days. A sickness cash benefit of less than 50 SEK/day indicates part-time employment. These 70 patients constitute the sample of the present study.

Initial ratings and record data

At discharge after their first admission, all the patients were rated with a multidimensional diagnostic schedule as described above. For the present analysis, the main emphasis is put on the composite item 'personality disturbance with lack of internal control of behaviour', consisting of the items 'easily led, dependent, immature', 'psychopathic behaviour' and 'uncommitted, cold, uninterested'. These items were the ones that were seen as reflecting, in psychodynamic terms, an ego and/or superego weakness. This type of personality disturbance was more frequent in the abstainers than in the social drinkers ($P<0.05$). Otherwise, there were no significant differences between the abstainers and the social drinkers on the initial ratings.

The subjects who were married at the time of their first admission, who were not known to temperance boards, and who had a stable work record (according to the hospital records) and no criminal record, were categorised as socially stable, the others as socially unstable. Social stability at first admission was more frequent in the social drinkers than in the abstainers ($P<0.05$).

Personal follow-up

The subjects were contacted by mail and asked, according to rules set by the research ethical committee of the Faculty of Medicine, University of Lund, if they were willing to participate in a follow-up interview. The personal follow-up was carried out in 1982 and 1983. The interviewer (GN) was blind to all data about the subjects except age and year of first admission. The subjects were not informed in any way that the interviewer knew anything about the nature of the problems for which they were originally admitted. The interview was partly structured and partly open-ended. Sixty patients (86%) agreed to participate. Independent corroboration of the subjects' reports was obtained in 83% of the cases. The lost subjects differed from the ones who were interviewed, with a higher frequency of initial ratings indicating psychopathic behaviour ($P<0.001$), while available data indicated that outcome was about the same (Nordström & Berglund, 1986*a*). The length of the follow-up period was 21 ± 4 years. The mean age when first admitted was 32 ± 7 years and the mean age at follow-up 53 ± 7 years. The follow-up methodology and the interview technique, as well as details about different kinds of corroboration of the subjects' reports, are reported in detail elsewhere (Nordström & Berglund, 1987*a*).

The presence and severity of psychopathological symptoms at follow-up were assessed with the Comprehensive Psychopathological Rating Scale (CPRS) (Åsberg *et al*, 1978). The Drug Taking Evaluation Scale (DTES) (Holsten & Waal, 1980) was used to assess different aspects of psychosocial functioning at the time of the follow-up. Three of the four subscales were used (social functioning, social belonging, psychic status). On each subscale a global rating is made on a 9-step scale. Scale steps 1–3 indicate normal area, 4–6 symptom area and 7–9 illness area. The extent of the interview information obtained was in some cases too limited to permit reliable rating on the CPRS or the DTES. All patients were also asked about the main cause of improvement. In the present paper, only answers categorised as implying a decreased tolerance for alcohol are used in the analysis. The issue of causes of improvement has been dealt with in detail elsewhere (Nordström & Berglund, 1986*b*).

Classification of alcoholism

For the classification of alcoholism, both interview and record data were used. The classification was performed blindly with regard to initial ratings. Alcohol-dependence was assessed according to DSM-III criteria (American Psychiatric Association, 1980). For the retrospective year-by-year categorisation of drinking patterns, the Problem Drinking Scale (PDS), developed by Vaillant (1983), was used. This scale consists of 16 equally weighted items covering dependence symptoms as well as social and medical consequences of abuse. Subjects who had reached a cumulated score of 4 or more on the PDS were categorised as abusers for every year in which at least one score on the scale was noted, and as abstainers (if drinking no alcohol) or social drinkers if there was no score during the year. The mean number of drinks (one drink equal to 0.5 fl. oz) per month in the social drinkers was 25, range 5–53. Beer was the main alcoholic beverage consumed. Abusers with a more severe drinking pattern (> 3 abuse days per month, or > 2 weeks of continuous abuse per year) were categorised as

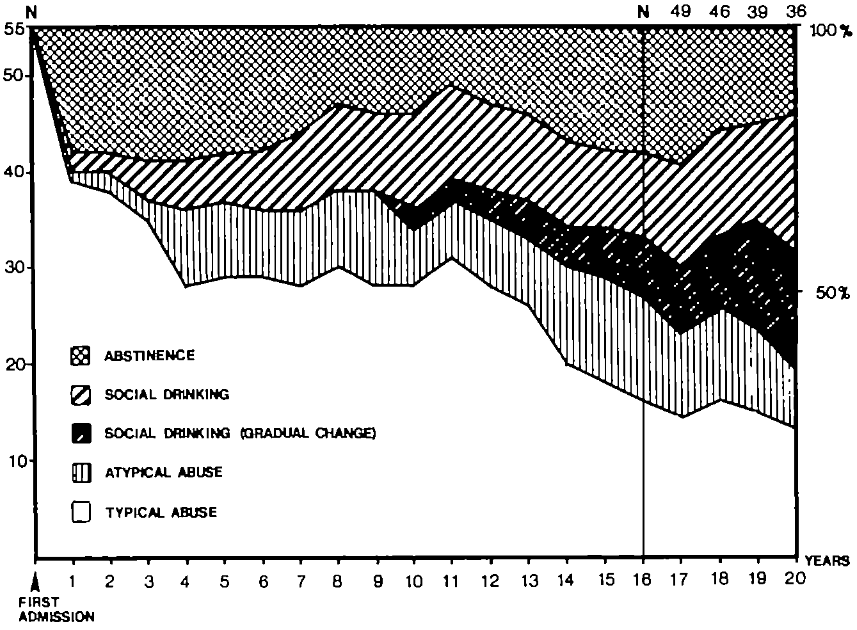

FIG. 1 Composite drinking patterns during the 16–20 years following first admission for the 55 subjects who had a lifetime diagnosis of Alcohol Dependence (DSM-III). All subjects were followed for at least 16 years after first admission. For years 17–20, percentages have been calculated for the subjects who were followed.

'typical abusers', and those with a less severe drinking pattern as 'atypical abusers' (Nordström & Berglund, 1987*b*).

In the present sample there were 55 subjects with a lifetime diagnosis of alcohol-dependence. Out of these, 13 were typical abusers at follow-up, 10 were atypical abusers, 21 social drinkers and 11 abstainers. The individual courses in atypical abuse (Nordström & Berglund, 1987*b*) and in social drinking and abstinence (Nordström & Berglund, 1987*a*) are reported elsewhere. Among the social drinkers, 11 reported a change by way of abstinence periods (mean duration 3 years), whereas 10 reported a change by gradual stages from problem drinking to social drinking. The present analysis is confined to the typical abusers, the social drinkers and the abstainers. The subjects who had never been alcohol-dependent were excluded.

Abstinence or social drinking were regarded as 'stable' when there was neither any relapse into typical abuse nor any longstanding relapse into atypical abuse. In three cases sobriety was regarded as stable although there were one or two isolated episodes of abuse of at most a week's duration, leading to a categorisation of the drinking pattern for the year concerned as atypical abuse. For another subject, a period of atypical abuse of a few months' duration, after a year of social drinking, was disregarded. Further, a month's relapse into typical abuse after 7 years of sobriety in one of the abstainers was disregarded concerning the question of stability of improvement.

Statistics

Differences between proportions were tested with the χ^2 test, or, if numbers were small, with the Fisher test (Siegel, 1956). Differences in distributions were tested with the Mann–Whitney *U* test (Siegel, 1956).

Results

Drinking patterns over intervening years

Figure 1 shows the composite course of drinking patterns during the 16–20 years following the first admission for the 55 subjects who had a lifetime diagnosis of Alcohol Dependence (DSM-III). The rate of sobriety (abstinence or social drinking) in the first year after first admission was 27%, changing very little during the first decade. After that, there was a gradual increase of the proportion of subjects attaining sobriety. The rate of improvement during the second decade was about 2–3% per year.

TABLE I

Initial ratings and record data related to drinking pattern at follow-up

	Drinking pattern at follow-up			
	Social drinking via gradual change	*Social drinking via abstinence*	*Abstinence*	*Typical abuse*
Number	10	11	11	13
Age when first admitted (median)	35	33	36	31
Heredity for alcoholism or personality disorder	4	6	6	7
Delirium tremens	0	3	3	2
Personality disturbance with lack of internal control of behaviour (A)	3	3^{1}	$8^{1,2}$	4^{2}
Social instability (B)	6	3^{1}	$9^{1,2}$	5^{2}
A+B	2	0^{3}	6^{3}	3
A or B	5	6	5	6
Neither A nor B	3	5^{1}	0^{1}	4
PDS score when first admitted (calculated retrospectively)	6.8±2.9	7.5±2.1	7.5±2.5	7.2±2.3

1. Comparison between abstainers and social drinkers via abstinence: Fisher test, *$P<0.05$.
2. Comparison between abstainers and typical abusers: Fisher test, *$P<0.05$.
3. Comparison between abstainers and social drinkers via abstinence: Fisher test, **$P<0.01$.

Abstinence was the most frequent type of favourable adjustment initially. However, 40% of those who were abstainers during some or all of the first 6 years after first admission were later to be found among the social drinkers. Gradual change to social drinking occurred later during the period and accounted for about 60% of the increase of sobriety rate seen during the second decade.

Initial characteristics related to drinking patterns at follow-up

Table 1 gives data about initial characteristics (initial ratings as well as record data) for the social drinkers with a gradual change, the social drinkers with a change by way of abstinence periods and the abstainers, compared with the typical abusers. Both personality disturbance with lack of internal control of behaviour, and social instability, were more frequent ($P<0.05$ for both) in the abstainers as compared with the social drinkers; also in comparison with the typical abusers. The social drinkers with a change by way of abstinence periods had the prognostically most favourable background characteristics in terms of personality disturbance and social stability.

Development and outcome characteristics related to drinking patterns at follow-up

Table II shows development and outcome data for the four outcome groups. There were no significant differences between the groups concerning age at follow-up.

The year when stable sobriety was attained differed significantly between the social drinkers with a change via abstinence (median 2 years after first admission) and those with a gradual change from abuse to social drinking (median 16 years) ($P<0.01$), and also between the social drinkers with a change via abstinence and the abstainers (median 10 years) ($P<0.05$). The social drinkers with a gradual change were older when stable sobriety was attained (median 51 years) than those with a change by way of abstinence periods (median 37 years) ($P<0.01$).

None of the social drinkers with a gradual change had ever had a delirium tremens, compared with five of the social drinkers with a change by way of abstinence periods ($P<0.05$). Five out of ten subjects with a gradual change from abuse to social drinking reported symptoms indicating a decreased tolerance for alcohol as mainly responsible for their improvement, whereas none of the subjects whose improvement had been related to initial or lasting abstinence did so ($P<0.01$).

As the follow-up, married status was more frequent in the social drinkers than in the abstainers or in the typical abusers, while the divorce rate was highest for the typical abusers. There were no differences between the three groups of subjects with favourable drinking patterns at follow-up concerning psychiatric symptoms, social functioning or social belonging. The abstainers and the social drinkers, taken together, scored lower than the typical abusers on the CPRS ($P<0.05$), and on the social functioning and psychic status subscales of the DTES ($P<0.01$) and the social belonging subscale of the DTES ($P<0.001$).

Discussion

Whether or not alcoholism is a 'self-limiting disease' (Drew, 1968) cannot be answered by this study. Further, the use of a clinical sample of male alcoholics limits the generalisability of our findings.

TABLE II

Development and outcome characteristics related to drinking pattern at the follow-up

	Drinking pattern at follow-up			
	Social drinking via gradual change	*Social drinking via abstinence*	*Abstinence*	*Typical abuse*
Number	10	11	11	13
Age at follow-up (median)	58	55	56	52
Cumulated PDS score	9.1±2.6	8.7±1.2	9.4±0.8	10.0±3.2
Number of years after first admission that stable sobriety was attained (median)	16[4]	2[4]	10	—
Age when stable sobriety was attained (median)	51[4]	37[4]	45	—
Delirium tremens ever	0[3]	5[3]	3	2
Decreased tolerance for alcohol as main cause of improvement	5[3]	0[3]	0	—
Marital status at follow-up				
never married	3	1	5	2
married	6	9	3	4
divorced	1	0	2	6
widowed	0	1	1	1
Annual income (1000 SEK)	73±22	76±23	66±20	74±15
CPRS score[1]	8±10	10±7	9±13	15±7
DTES score[2]				
social functioning	1.3±0.5	1.6±0.5	1.8±0.7	2.6±1.3
social belonging	2.6±2.2	1.9±0.8	2.6±1.6	4.5±2.1
psychic status	3.1±1.7	2.9±1.0	2.9±1.3	4.2±1.6

1. $N=7$, 11, 8 and 11, respectively.
2. $N=10$, 11, 8 and 13, respectively.
3. Comparison between social drinkers via abstinence and social drinkers via gradual change: Fisher test, *$P<0.05$.
4. Comparison between social drinkers via abstinence and social drinkers via gradual change: Mann–Whitney U test, two tailed, **$P<0.01$.

The course of alcoholism in females has been reported to differ from that in males, with a more rapid progression (Stabenau, 1984; Hesselbrock *et al*, 1985). We do not know to what extent data on ageing and recovery obtained from male subjects are relevant also for females. The characteristics of our sample as regards age when first admitted prevents us from drawing conclusions concerning changes of drinking behaviour in younger age-groups. Differences between younger and older men as regards norms and expectancies concerning drinking behaviour have been hypothesised, indicating that in this connection the age variable may be strongly related to socio-cultural factors (Fillmore & Midanik, 1984).

Our sampling procedure also precludes us from studying the influence of physical illness on drinking behaviour, since the subjects who were investigated were selected on the basis of a low number of sickness cash benefit days. In an age-matched sample of alcoholics with a disability pension, which has been described in previous papers (Nordström & Berglund, 1986*a*, 1987*b*), four out of the five subjects who attained sobriety reported that physical or mental illness had been important in the improvement of their drinking habits.

The main finding of the present study was that the processes of improvement differed between older and younger alcoholics. Improvement in older subjects was related to a pattern of gradual change from abuse to social drinking. This type of improvement, which has been reported previously (Kendell, 1965; Bailey & Stewart, 1967; Knupfer, 1972), was responsible for most of the change to sobriety seen during the second decade after first admission and after the age of 45. The predominance of social drinking over abstinence in successful adjustment in older subjects is in agreement with the findings reported by Ciompi & Eisert (1971).

Animal studies have shown three main types of mechanism that might explain the changes in alcohol effects with ageing: a decreased rate of alcohol metabolism, possibly partly due to changes in hepatic functioning with increasing age; a decrease in body water, leading to higher blood alcohol levels; and neurochemical changes, with a subsequent increased sensitivity to ethanol of the aged central nervous system (Wood & Armbrecht, 1982. Bardo & Risner, 1985).

Though alcoholics with severe liver disease tend to decrease their alcohol consumption (Hermos, 1984), this does not seem to be a plausible explanation for improvement in the present sample. While subjects with pathological liver function blood tests in our sample were all typical abusers, there were no indications of liver damage in improved subjects. It has been reported that the decrease in body water with increasing age may contribute to a decreased alcohol consumption in man (Vogel-Sprott & Barrett, 1984). In the same study, however, evidence was found that the alcohol-induced impairment of behaviour when blood alcohol level was controlled increases with age, and it was suggested that this, too, may lead to a compensatory reduction of alcohol consumption.

In the present sample, the clinical state was the same in the social drinkers with a gradual change from abuse as in the social drinkers with an early improvement by way of abstinence periods or in the abstainers. There were no behavioural indications of premature ageing, with a subsequent decreased capacity for sustained heavy drinking. However, an increasing number of reports have shown that long-term sobriety produces a normalisation of alcoholics' brain-functioning, as measured by symptom check-lists (DeSoto *et al*, 1985), by neuropsychological tests (Berglund *et al*, 1977; Grant *et al*, 1984) by CT scan (Ron *et al*, 1982) or by regional CBF (Berglund *et al*, 1980. Ishikawa *et al*, 1986).

Thus, age-related alcohol-induced cognitive impairment cannot be discarded as a possible improvement-promoting factor in our sample. However, the preserved psychosocial functioning through years of abuse in our social drinkers with a change by gradual stages, could perhaps indicate that mechanisms related to normal ageing are more likely to have contributed to their improvement. Age-related changes in body water and in central nervous system sensitivity to alcohol, as referred to above, may be of importance in this respect. Another possible mechanism could be a decrease of the euphoric effects of alcohol due to changes in the levels of different neurotransmitters related to ageing (Winblad *et al*, 1978; Gottfries, 1980; Liljequist, 1979). The finding that five of the ten social drinkers with a gradual change, but no others, referred to symptoms indicating a decreased tolerance for alcohol as being important for their improvement could support the hypothesis that physiological processes of normal ageing may be involved in remission from alcoholism in middle-aged and older subjects.

It was observed that the background data for the social drinkers with a change via abstinence, who were those who improved earliest after their first admission and at the youngest age, were quite similar to the indicators of a 'good prognosis' described by, for instance, Davies *et al* (1956) and Vallance (1965), namely a good personality and social stability. The background characteristics for the social drinkers with a gradual change did not differ significantly from those of the other groups. Partly because of the low number of subjects, the difference between the two groups of social drinkers concerning the frequency of delirium tremens should be interpreted with caution. Possibly, the experience of a delirium may more strongly motivate for abstaining and be less compatible with a gradual cutting down of alcohol consumption.

To conclude, the findings from this long-term follow-up of socially well adjusted alcoholics indicate the existence of specific patterns and processes of improvement related to ageing. From the clinician's point of view, the results presented may perhaps be seen as implying that the prognosis for the middle-aged or older alcoholic who has managed to maintain good social adjustment is better than would be expected were only the duration of alcohol problems taken into consideration. Possibly, therefore, this group of alcoholics might be a suitable target group for intensified treatment efforts. On a general population level, suggestions of a similar kind have been put forward concerning the possible value of widespread information about the cerebral effects of alcohol in older people (Parker *et al*, 1982).

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Alcohol, Other Drugs and Tobacco Use and Anxiolytic Effectiveness

A Comparison of Anxious Patients and Psychiatric Nurses

S. TILLEY

Recent studies raise questions regarding use or abuse of alcohol by phobics, and about compatibility of behaviour therapy and benzodiazepine use. In this study 40 patients, referred with mainly anxiety-related problems to a nurse behaviour therapist, and a comparison group of 40 psychiatric nurses completed a self-administered questionnaire assessing use of alcohol, sedative drugs and tobacco, and their perceived anxiolytic effectiveness. The paper confirms earlier findings that the patients, in general, drank less than the general public. They also drank less than the nurses, but used more sedative drugs and were heavier smokers. Alcohol was an unreliable anxiolytic. A few patients, including two agoraphobics, drank heavily and did not enter treatment made conditional on stopping drinking. Implications for case management are discussed.

The complex relationship between alcohol and sedative-drug dependence and anxiety-based problems poses thorny theoretical and clinical issues. A brief review of these will put the present study in context.

A number of studies have noted alcohol and sedative abuse among patients with anxiety-based problems (Marks *et al*, 1966; Sim & Houghton, 1966; Quitkin *et al*, 1972; Woodruff *et al*, 1972; Smail *et al*, 1984; Samarasinghe *et al*, 1984; Bibb & Chambless, 1986). Others have revealed a surprisingly high incidence of phobias among alcoholics (Mullaney & Trippet, 1979; Smail *et al*, 1984; Bowen *et al*, 1984). One question raised by these reports is whether people with anxiety-based problems become dependent on alcohol or sedatives through increasing and unsuccessful use of these drugs to quell anxiety; or whether heavy use precipitates or 'fuels' anxiety, especially phobic anxiety (Quitkin *et al*, 1972; Klein, 1980; Stockwell *et al*, 1984; Samarasinghe *et al*, 1984; Bowen *et al*, 1984; Bibb & Chambless, 1986).

Closely related is the question of clinical management. Some writers have advised treating alcohol or sedative abuse first, citing remission of phobias with abstention (Stockwell *et al*, 1984; Ashton, 1984). Others argue that treatment of dependence will fail if anxiety is not treated first (Quitkin *et al*, 1972). Miller (1986) reviews the pitfalls of concurrent treatment.

Behaviour therapists in particular have highlighted these clinical issues. Hafner & Marks (1976) and Chambless *et al* (1979) suggested that sedatives do not increase, and may reduce, the effectiveness of behaviour therapy for phobias. Sartory (1983) reviewed arguments about state-dependent learning and attribution of effects to drugs, concluding that sedatives should be stopped before behaviour therapy.

How often are problems of dependence likely to be encountered by clinicians treating anxiety-based problems, and what are sensible guidelines for managing such problems, in the absence of expertise in treating dependence? Samarasinghe *et al* (1984) surveyed the drinking and sedative use of 106 outpatients referred for behaviour therapy. They found that few drank to excess, and most found these substances unreliable anxiolytics. Patients generally complied with instructions to refrain from alcohol or drugs before sessions. With one exception, the few whose use was problematic stopped it when asked to do so as a condition of having treatment.

The present study provides more data on the use and reported anxiolytic effectiveness of alcohol and sedatives among patients referred to a nurse specialising in behaviour therapy. The methodology of Samarasinghe *et al* (1984) was in part replicated, as was their clinical practice of asking patients whose use of alcohol or drugs was seen as problematic to stop it before treatment, with referral for help with dependence if necessary. Tobacco use was also assessed. The patients' use of these substances was compared to that of a group of psychiatric nurses, chosen for convenience and because their judgement may be involved in assessment and management of patients' use; as well as to that of the patients reported in Samarasinghe *et al* (1984) and the general (Scottish) population.

Method

The questionnaire

This was based on the self-administered questionnaire used by Samarasinghe *et al* (1984). It had questions on usual frequency and quantity of alcohol and drug consumption; part use; and effects on anxiety, anticipatory anxiety and coping. In addition, patients were asked about tobacco use and anxiolytic effectiveness; and to report their previous week's drinking.

The respondents

Of 42 consecutive adult patients, 40 were asked to complete the questionnaire. All agreed. Twenty-six were male, 16 female; mean age was 37; 33 were out-patients, five in-patients, two day-patients; 18 were referred by psychiatrists, 15 by GPs, seven by others. Patients were retrospectively classified by the author into DSM-III categories where possible: agoraphobia with panic attack (ten); social phobia (eight); simple phobia (one); obsessive-compulsive disorder (five); marital problem (three); sexual dysfunction (two); intermittent explosive disorder (two); depressive neurosis (one); major depressive disorder (one); generalised anxiety (one); others (two); not classifiable (three). One patient denied having a problem.

Forty-two psychiatric nurses, matched for age (except two patients over 60 matched with nurses in their 50s) and sex, were asked to complete the same confidential questionnaire. Two declined.

Analysis

Results were analysed using χ^2 tests for independent samples or Fisher's exact probability test. The same tests were used for comparisons (see Discussion) with Samarasinghe *et al* (1984) and Dight (1976). In the latter comparison, percentages rather than numbers are compared to reduce distortion due to different sample sizes. Tests were one-tailed for sedative drug use, as patients were assumed to use more; and two-tailed for alcohol use, following Samarasinghe *et al* (1984) who found patients' drinking like that of the general population.

Numbers of responses reported are in some cases less than 40 due to incomplete questionnaires; similarly, percentages may be of the smaller numbers.

Results

Alcohol

Alcohol consumption is reported in units, where one unit equals 9 g alcohol.

On the whole, the patients drank less and less frequently than the nurses. In the previous week 27 patients (69%) reported drinking no alcohol; six (15%) drank 1–6 units; three (8%) 7–30 units; one (3%) 31–100 units; and two (5%) over 100 units. Ten nurses (26%) had drunk no alcohol; eight (21%) 1–6 units; 14 (36%) 7–30 units; seven (18%) 31–100 units; and none over 100 units. The difference between nurses and patients having a drink in the previous week was significant ($\chi^2 = 17.3$; d.f. = 1; $P < 0.001$).

Four patients (10%) reported usually drinking 6–7 days a week; seven (18%) 2–5 days; 15 (36%) once a week or less; and 14 (35%) were 'teetotal'. Among nurses, two (5%) usually drank 6–7 days a week; 16 (40%) 2–5 days; 19 (48%) once a week or less; three (8%) were 'teetotal'. Significantly fewer patients drank at least once a week ($\chi^2 = 8.5$; d.f. = 1; $P < 0.01$).

The amount drunk on a typical drinking day, for those nurses and patients who drank, was not significantly different. The heaviest drinkers were two agoraphobic patients, both of whom had received treatment for drinking problems. No female nurses, but 13% of female patients drank within a range said to involve a 'risk of harm' to health (36–64 units per week; Health Education Council, 1983). The two agoraphobics (9%) drank amounts indicating they 'may well be dependent' on alcohol. No male patients, but 13% of male nurses drank in the range involving a 'risk of harm' (51–95 units per week).

There were no significant differences in how often patients and nurses reported using alcohol for anxiety, nor in how effective they found it. Only three patients (8%) and no nurses always used alcohol when anxious; most in both groups never did so. The results were similar for anticipatory anxiety. Three patients (27%) and none of the nurses who took alcohol when anxious found it always helpful. Most found it sometimes helpful. Again, the results were similar for anticipatory anxiety.

Other drugs

Thirteen patients (33%) took minor tranquillisers; 15 (38%) antidepressants; two (5%) hypnotics. One nurse (3%) took minor tranquillisers; one (3%) antidepressants; one (3%) both. Altogether 24 patients (60%) and three nurses (8%) took one or a combination of these drugs ($\chi^2 = 24.65$; d.f. = 1; $P < 0.01$). Of the 16 patients (40%) not taking prescribed drugs, three (8%) used cannabis.

Four patients (11%) always took drugs other than alcohol when anxious; 14 (38%) sometimes; 19 (51%) never. Two nurses (5%) sometimes did so; the rest never. Six of those patients who took these drugs for anxiety (33%) always found them helpful; 11 (61%) sometimes; one (6%) never. One nurse found them helpful.

Tobacco

Twenty-two (55%) of the patients smoked, 14 (35%) of the nurses. Significantly more patients (nine; 23%) than nurses (none) smoked over 200 cigarettes a week ($\chi^2 = 5.18$; d.f. = 1; $P < 0.05$).

Seventeen patients and 11 nurses always or often smoked when anxious; six patients and eight nurses always or often found smoking when anxious made them feel better. There were no significant differences between nurses and patients in their use of tobacco for anxiety or its effectiveness.

Discussion

How valid is such a questionnaire? Self-report has strong face validity in assessment of perceived anxiolytic effect. While stringent checks on the accuracy of self-report of alcohol and drug use may not be feasible in the out-patient setting described here, the use of such measures has support from workers experienced in assessment of alcohol problems (Sobell & Sobell, 1975; Bernhardt *et al*, 1984), and is justified in the context of this study. Moreover, in further assessment and treatment of the patients, no grounds were discovered to doubt the reported level of drinking. The case of the nurses may be different. Nurses might be expected to under-report alcohol or drug use which could be seen as problematic, for fear of arousing professional concern. The validity of the nurses' self-report is also questionable given the finding that their estimates of usual drinking and last week's drinking differed significantly more often than did the patients', with last week's higher than usual ($\chi^2=8.23$; d.f. = 1; $P<0.01$). However, the fact that the nurses' drinking was still higher than that of the general population suggests that gross underestimation is less likely.

The reliability of the questionnaire is reinforced through comparison of the results for the patients in this study (referred to as the Dumfries patients) and the 106 patients reported on in Samarasinghe *et al* (1984) (referred to as the London patients). Both groups of patients were treated by nurse therapists with no expertise in managing dependence. Age and sex ratio were similar. Referred problems were broadly similar, with fewer phobics and obsessive-compulsive patients in Dumfries (60% compared to 78%; $\chi^2=4.97$; d.f. = 1; $P<0.05$).

The two groups were similar in the usual frequency of drinking and amount drunk on a typical drinking day; and in use and perceived effectiveness of alcohol for anxiety. Use of drugs and effectiveness were also similar.

One difference was notable. While there were no significant differences in drinking among different diagnostic groups in London, in Dumfries two patients, both agoraphobics, drank notably more than the rest – both 6–7 days a week, 114 and 119 units the previous week. Agoraphobics drank significantly more than other diagnostic groups in Dumfries (with 100 units as criterion, Fisher's exact $P<0.01$). This finding coincides with others (Bibb & Chambless, 1986) and indicates that agoraphobics may merit particular screening for alcohol dependence.

How does the drinking of the Dumfries patients and nurses compare with that of the general population? Table I summarises data on them and on people of like age and sex in a larger Scottish sample (from Dight, 1976). The categories are Dight's regular drinkers who had had a drink in the previous week; occasional drinkers who had not; and non-drinkers. The age groups compared are 20–40 as these contained most patients and nurses.

TABLE I

Proportions of drinkers and non-drinkers among Dumfries patients, nurses and Dight's sample (percentages)

	Dight	*Dumfries patients*	*Dumfries nurses*
Males 20–40			
Regular drinkers	84	30*	100*
Non-drinkers	2	10**	0****
Females 20–30			
Regular drinkers	58	57****	57****
Non-drinkers	6	14****	14****
Females 30–40			
Regular drinkers	56	14*	72***
Non-drinkers	3	72**	0****

*$P<0.001$; **$P<0.02$; ***$P<0.05$; ****NS

Compared to Dight's sample, a smaller proportion of male patients and a larger proportion of male nurses were regular drinkers. Non-drinkers were more common among male patients than among male nurses or Dight's sample. There were similar proportions of regular drinkers and non-drinkers among females in their 20s in all three groups. Of females in their 30s, however, a smaller proportion of patients and a larger proportion of nurses were regular drinkers than in Dight's sample; while non-drinkers were more common among female patients than among nurses or Dight's sample.

This study thus confirms the finding of Samarasinghe *et al* (1984) that most patients referred to nurse therapists for behaviour therapy drink less, not more, than the general population, and that they do not in general find alcohol or sedative drugs reliable as anxiolytics. Few fit the picture (Quitkin *et al*, 1972) of anxious people pursuing anxiolytic effect to the point of dependence. Those few raise important case management and theoretical issues.

Case management

In following the case management guidelines suggested by Samarasinghe *et al* (1984) the author asked four (10%) of the Dumfries patients to stop or reduce use of alcohol or drugs prior to behavioural treatment: the two agoraphobics; a social phobic who regularly drank heavily to allay anxiety; and an obsessional who used 10 mg lorazepam per day and feared worse symptoms if she stopped. The social phobic complied. The obsessional refused to reduce her drugs but agreed to refrain from use before treatment sessions. The two agoraphobics felt unable to comply with the request that they reduce drinking or at least agree to abstain before treatment and homework sessions, despite compliance being a precondition of treatment. The issue of dependence was then discussed and referral for help with this, with the prospect of later re-assessment, was offered. Neither was subsequently re-referred for behavioural treatment.

Thus two (5%) of the patients did not enter behavioural treatment for their anxiety-related problems when these guidelines were followed. The application of these guidelines could be construed as successful. A few problems of dependence were highlighted, though the most severe were already known and had been the focus of at least partially unsuccessful treatment. A few patients modified their use of these substances in ways that literature on anxiety-'fuelling', state dependent learning and attribution (Sartory, 1983; Stockwell *et al*, 1984) suggests may be helpful. Difficulties of trying to implement behavioural treatment with patients experiencing problems of dependence were, perhaps, avoided. The uncertainty on this point is important. The arguments of the above studies, though well-founded theoretically, have yet to be conclusively validated in the clinical setting. Following these management guidelines precludes, for example, single case studies of behaviour therapy given during periods of alcohol or sedative use and abstention, which might further understanding of the relationship of substance use and treatment efficacy. Two questions are thus raised: (a) given the current state of knowledge are therapists justified in requiring patients who are not asking for help with their use of alcohol or sedatives to change it in order to have behaviour therapy? and (b) if heavy users of these substances are screened from behaviour therapy, how can the claim that such use works to the detriment of that therapy be refuted?

Finally, while the emphasis of this paper has been on alcohol and sedative use, the high proportion of smokers among the anxious patients should be noted. Further study and consideration of appropriate intervention is needed.

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Point of View

Brief Psychoses: Do They Contribute to the Good Prognosis and Equal Prevalence of Schizophrenia in Developing Countries?

JANICE STEVENS

A high incidence of brief schizophrenia-like psychosis in Africa and other parts of the developing world has been repeatedly documented by many psychiatrists working in these regions (Dembovitz, 1945; Carothers, 1953; Jilek & Jilek-Aall, 1970; German, 1972; Leighton *et al*, 1963). This report does not deny that typical schizophrenia occurs in these countries, but the common culturally based schizophrenia-like psychosis may be misdiagnosed as schizophrenia, and thus inflate prevalence and improve prognosis data.

Most prevalence and outcome studies of schizophrenia in developing countries are based on individuals who are brought to out-patient clinics and hospitals. Although the number of these patients cannot be the basis for establishing prevalence throughout a region, it is possible to examine and compare clinical phenomenology and patient mix with similar data from Europe or North America, recognising that in general, only those with the most severe illnesses or who live reasonably close to the facility are likely to come to the hospital or clinic in less developed countries. Such a survey was undertaken by the author during a three-month tour in 1984 as Visiting Senior Lecturer in Psychiatry at the Medical School of the University of Zimbabwe.

The distribution of psychiatric diagnoses entering the mental hospitals in Zimbabwe is both similar to and different from that in the USA and Europe. In Harare, Zimbabwe's largest city, 40–50% of all admissions were diagnosed schizophrenia in 1983–1984. However, if Research Diagnostic Criteria (Spitzer & Endicott, 1978), requiring at least two weeks of illness for diagnosis of schizophrenia, are applied, more than half of these patients fail to meet the duration criterion. Instead, the condition would be diagnosed brief reactive psychosis. If the six months of continuous illness required by DSM-III (American Psychiatric Association, 1980) for diagnosis of schizophrenia is used, only 10–15% of admissions meet the criteria. Only 5–6% of admissions were diagnosed affective disorders, nearly all of manic type with no history of depressive episodes. Ten percent were diagnosed alcohol-related psychoses, 5–8% puerperal and the remainder accounted for by a variety of organic brain syndromes, including pellagra, severe anaemia, general paresis, cerebral malaria and alcoholic psychosis. Notable by their rarity or absence were the senile and presenile psychoses, major depressive psychoses, and eating disorders. Common were two syndromes that are relatively unusual causes of hospital admission in North America and Europe, namely brief reactive psychoses and psychoses of essentially similar form attributed to cannabis.

This report does not attempt to review the abundant literature on phenomenology of psychiatric illness of Africa or other developing areas, but is focused on the questions of the contribution of acute schizophreniform psychoses to prevalence and prognosis data. These are questions of great importance for those concerned with the aetiology and thus the epidemiology of schizophrenia.

Brief reactive psychoses

Frequency of acute psychoses as a cause of admission to hospital has been reported by a number of psychiatrists working in Africa and in other areas of the developing world (Bert, 1951; Gelfand, 1956; Collomb, 1967; Lambo, 1965). The acute brief psychoses occur in otherwise healthy young men and women of any age, but predominantly in young adults. They are characterised by relatively abrupt onset of extreme restlessness, insomnia, mental confusion, auditory and visual hallucinations, delusions of being controlled by others, incoherent rambling, pressured speech, disrobing, destructive and combative behaviour such as tearing or burning clothes, destroying property, and attacking or beating relatives. In Zimbabwe, patients so afflicted nearly always have a strong belief in supernatural forces controlling their lives and in the power of the traditional healer (*n'anga*) to control these forces. Often, a trip to the traditional healer for explanation of some untoward event directly preceded onset of the psychosis. Prior to use of electroconvulsive treatment and neuroleptics, these psychoses lasted 1–4 weeks (Dembovitz, 1945). With modern treatment, the course is generally half this length.

With or without treatment, recovery is usually complete.

Case histories

Case 1

A young teacher was brought to the psychiatric hospital by the police after smashing several plate-glass windows in local shops and breaking the windows of cars parked on the street. He then took off all his clothes and walked or ran nearly 10 km through the town stark naked. When captured, he was intensely aggressive, talked incoherently and claimed voices were talking to him and directing his actions. Following recovery, which required several weeks of treatment with high dose, high potency neuroleptics, he told the following story.

Approximately one month prior to onset of his running amok, he consulted a local *n'anga* to determine whether the future of his job was secure. The *n'anga* told him his workmates were jealous of him and might try to harm him. A few weeks later he found a 'flash card' in his office. It read, 'To die'. He knew at once that his colleagues planned to murder him, probably by bewitchment. This episode would not leave his mind. Hallucinated voices began to echo his thoughts; and his state of wild excitement ensued. He also admitted that prior to all these events he had been smoking 'dakha' (marijuana, cannabis) regularly.

Case 2

A 65-year-old widowed African woman was well until three days before admission to the psychiatric unit at Harare Hospital when she developed sleeplessness and restlessness and 'talked nonsense'. Five days before admission she had gone to the traditional healer because of headache. She was told that evil spirits had entered her head. Poultices soaked with a brew of herbs were applied to her scalp. She became increasingly restless and was brought to the hospital by police. On examination, this thin, elderly woman was found lying on the floor of the nurse's office, talking incessantly and irrelevantly, and unresponsive to commands. There were large patches of raw peeling skin on her head where the *n'anga*'s application of caustic herbal poultices had succeeded in removing her hair and scalding her scalp. She responded quickly to modest doses of chlorpromazine. When she recovered she told us that shortly before her illness she had received word that all her daughter's clothes and money had been stolen. In her view, this could not be an accident. Anxiety over the event, plus her fear of bewitchment, evidently triggered a typical brief psychosis.

Case 3

A 20-year-old unmarried woman was brought to hospital by relatives because of severe assaultive behaviour of abrupt onset. On admission she was excited, disoriented, evidently hallucinated, and combative. She was placed in isolation and treated with chlorpromazine, and recovered rapidly. Her sister then told us that the patient's father had by mistake taken her menstrual cloths to the *n'anga*, thinking they belonged to his (unwanted) second wife. The purpose of his visit was to have the *n'anga* injure this wife by charms applied to her used cloths. When the daughter discovered that the father had taken her cloths she was terrified (menstrual cloths are carefully tied up in a bag after use and no one is allowed to touch them for fear they may be used to bewitch the owner). Her typical brief psychosis ensued.

Discussion

Brought to hospital in handcuffs or tied up with rope, these patients, although initially violent, generally respond rapidly to sedation and seclusion. All symptoms usually disappear within 2–14 days. The patient typically claims amnesia for the entire period of onset and excitement. Less commonly the presentation is of extreme social withdrawal, mutism and negativism, also associated with auditory and visual hallucinations and feelings of being controlled by outside forces. In contrast to schizophrenia, which the episodes often resemble clinically, the brief psychoses are nearly always preceded by a definite environmental stress – usually a conflict arising from domestic strife, employment problems, accidents, illness or death in the family. In addition to such stressful social, economic and domestic events, prolonged or unusual physical illness in a close relative or friend is also a common reason for seeking explanations and help from the traditional healer. Thus most patients have consulted a traditional practitioner at some point proximate to their entry to hospital, and have been told either that their ancestral spirits are angry over some breach of rites, or that they have been bewitched.

We have been interested in a possible infectious origin for schizophrenia or some schizophrenias (Stevens *et al*, 1984). The better prognosis reported for schizophrenia in developing countries suggested that these brief schizophrenia-like psychoses might represent an encephalopathy that is quickly overcome by individuals with high immunity due to residence from infancy in relatively unsanitary environments. A similar proposal has been made to explain the more benign course of adult poliomyelitis and comparative rarity of multiple sclerosis in countries where sanitation is poor and both children and adults develop a high degree of immunity to many infections (Poskanzer *et al*, 1963).

To investigate whether some of these cases represented toxic psychoses, infection, or manifestations of psychomotor epilepsy, examinations were made of cerebrospinal fluid, electroencephalogram, urine screen for toxic agents, and peripheral blood smear for malaria or sleeping sickness in a number of typical cases. These studies were generally negative in the absence of a history or clinical signs of intoxication or infection. Rather than an infectious or toxic precipitant, it appeared, as also noted by Lambo (1965) and many others, that some untoward event in their lives coupled with a strong belief in and fear of easily angered spirits or bewitchment set the stage for the brief psychosis. Similar states were described in Europe at the turn of this century (Meynert, 1889) and have occurred, sometimes in epidemic proportions, among many ethnic groups undergoing cultural change.

In Zimbabwe, and elsewhere in Africa, the belief in witchcraft and fear of ancestor retribution are kept alive by the traditional healer, whose business it is to diagnose and treat all the disorders that are believed to arise from these two causes. The traditional healing sector in Zimbabwe is represented by more than 40 000 indigenous practitioners – diviners, healers, and herbalists – who are consulted by an estimated 90% of the African population. These practitioners learn their trade from an elder kinsman, or are 'called' to their profession during a dream or a state of possession. Many report that they become healers during or after experiencing a personal psychosis – itself considered evidence of spirit possession. Most diviners and healers have little or only a few years of education and many cannot read or write (Chavunduka, 1978).

The traditional healer is not only the first source of consultation for most patients with mental illness and epilepsy, but is also consulted by a majority of individuals or families when Western medicine fails to effect a rapid cure for a variety of ailments. Even when Western treatment is successful in relieving symptoms, it seldom explains remote causality, and it is this aspect of disease or misfortune that most interests the affected individuals or their families. For it is the traditional healer and not the Western doctor who can answer the patient's and family's all-important questions: '*Why* did this happen to *me*?' and '*Who* is doing it?' With their generally frightening answers to these questions, traditional practitioners appear to play an important role in maintaining the mental set of anxiety and fear of the supernatural that underlies symptoms of the brief psychoses.

In a recent study of 100 consecutive consultations by traditional practitioners in Harare and adjoining townships, Mavi *et al* (1983) reported that 29% had gynaecological, sexual, fertility, or VD problems, 8% had diarrhoea and/or vomiting, 8% were diagnosed 'depressed fontanelle' (a widely recognised result of bewitchment in infants), seven patients had swollen limbs or stomach, five were brought for 'madness', three for epilepsy, and most of the remainder with a variety of other physical ailments. Nine came for help because of recent bad luck or misfortune. Both cause and treatment were discovered by divination. There was little relationship between the complaint, the diagnosis of cause, and the treatment. Each case was judged according to context and no general rules or principles emerged. In the 100 cited cases, bewitchment was designated as cause of the problem in 19 cases, offended ancestral spirits in 16, and possession (by evil spirits) in 11. A majority (nearly 70%) were given herbal medicines to drink, inhale or rub on the skin; a quarter were required to perform some ceremony or pay a recompense of cows, cloth or cash to offended individuals or spirits. Charms to prevent further damage were sold to ten clients.

But the traditional healer is much more than a medicine man. He or she is the principal means of communication between the living and their dead ancestors. In most African religions, it is the ancestors who alone can protect the living from all the forms of ill luck that surround their lives. Witchcraft or sorcery, the only other traditionally recognised source of misfortune, can also be detected and counteracted only by the traditional healer. As the principal arbiter between the material and spirit world, the *n'anga* is in a position not only to both arouse and dispel fears but also to foster and perpetuate a thinking style that projects the cause of both good and evil events on mystical forces external to the self. Paranoid and violent manifestations of illness are therefore common, while melancholy, self-blame and guilt are rare – perhaps because the causal agent remains externalised and blame is directed outward rather than internalised (Field, 1960; Murphy *et al*, 1967; Jilek & Jilek-Aall, 1970; Schiefflein, 1982). Belief that the locus of control for one's fate resides in outside forces that can only be discovered and dispelled by the traditional healer has important implications for understanding thought patterns of the society at large as well as for analysis of the psychoses.

The International Pilot Study of Schizophrenia (IPSS) (World Health Organization, 1979) reported in 1979 that 36% of patients in Ibadan and 27% in Agra recovered from their psychosis in less than a month. The IPSS used the Present State Examination and CATEGO program (Wing *et al*, 1974) to establish diagnoses. This diagnostic system does not include duration-of-illness criteria but relies exclusively on the cross-sectional clinical picture. Since many patients with brief psychoses meet CATEGO criteria for schizophrenia, it is likely that a proportion of the individuals diagnosed as having schizophrenia would be classified as a brief reactive psychosis, schizophreniform disorder, or atypical psychosis under the six-month time criterion set by

DSM-III. These diagnoses have a better prognosis than DSM-III schizophrenia (Coryell & Tsuang, 1986). Inclusion of brief or schizophreniform psychoses in a study examining recovery rates in schizophrenia will generate a more favourable prognosis where these are common than is the case in countries where brief and schizophreniform psychoses are less frequently seen.

Although the prevalence of schizophrenia is often stated to be similar worldwide (Sartorius *et al*, 1986), prevalence as well as prognosis in developing countries may be greatly influenced by the inclusion of brief schizophreniform psychoses in the schizophrenias. A number of investigators have reported lower prevalence rates for schizophrenia in several developing countries (Fortes & Mayer, 1969; Torrey, 1980; Dale, 1981; Torrey *et al*, 1984; Halldin *et al*, 1984; Sikanerty & Eaton, 1984). Before definitive conclusions can be drawn, however, there is a need for prevalence studies based on modern criteria, including duration and house-to-house surveys rather than clinic or hospital-based statistics.

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Brief Reports

A Case of Gilles de la Tourette's Syndrome in Saudi Arabia

An occurrence of Gilles de la Tourette's syndrome in a 13-year-old Saudi female patient is documented, and treatment is described. It is thought that the improvement noted may be attributed to the hospital environment and reduced contact with the family.

Gilles de la Tourette's syndrome (GTS) is a condition that begins in childhood and is characterised by multiple motor tics and compulsive utterances that may progress to shouting of obscenities (coprolalia) (Fernando, 1976).

To the author's knowledge, no cases of the syndrome have been reported before from the Middle East: the following case report documents an occurrence, notes some cultural factors and describes the treatment of a patient in Saudi Arabia.

Case report

A 13-year-old Saudi female patient presented to the Psychiatric out-patient clinic at Riyadh Armed Forces Hospital (RAFH) with symptoms of compulsive uttering of obscenities, facial tics (blinking), other body tics and spitting. Her condition started seven years previously when she became extremely frightened one night by a group of cockroaches in a dark toilet. She spent the night crying, her sleep was restless and later she started to avoid going to the toilet to the extent of urinating and defaecating on herself. Four days later she exhibited facial tics which then progressed to multiple tics of neck, shoulders and hands. This was followed by utterances that later developed into true obscenities with a sexual or swearing content. The obscenities were reported by the patient's parents to be compulsive and worse in the presence of others.

The patient was brought up by very religious parents, and was the fourth youngest of 12 siblings. All family members rebuked her behaviour, and moreover her two older brothers were physically violent towards her. Her schoolmates used to laugh at her, calling her a "lizard". The teachers punished the patient because of her obscenities by making her stand facing the class. She was frightened to the extent of being incontinent in front of the class, and eventually she was expelled from school due to her obscene speech. Her family confined her to home, and did not allow her to talk to or play with her friends; hence she became almost isolated.

She had received treatment twice as an in-patient in two different psychiatric hospitals, but with little success. She was prescribed haloperidol: this only helped the compulsive utterances to some extent, and there were severe extra-pyramidal side-effects.

Following the initial assessment at RAFH Psychiatric Out-patient Clinic, the patient was admitted to the Psychiatric Unit of the hospital for a period of three months. During the first three weeks she received no medication, but exploratory psychotherapeutic sessions and regular meetings with the family were arranged to identify the psychodynamics and the factors of family pathology behind the condition. Physical examination and routine investigations were carried out, and showed the patient to be in good physical health, with no abnormality detected. Her IQ was found to be 'above average'. During this assessment period her behaviour was observed by our nursing staff and occupational therapist. Five forms of behaviour were identified, facial tics being the severest symptom encountered, followed respectively by utterances and obscenities; tics of neck and shoulder; spitting; and compulsive touching of the floor. All symptoms stopped completely during sleep.

At the end of the assessment period the following treatment strategy was formulated. Firstly, haloperidol was given. On a dose of 1 mg daily the patient developed severe extra-pyramidal side-effects; the dose was reduced to 0.5 mg daily, as this was found to be the maximum dose tolerated by her. Secondly, the psychotherapeutic sessions were continued, in an attempt to promote personality maturation, and so were the family meetings, in which counselling was the main theme. Thirdly, the patient attended the Occupational Therapy Department, where she was involved in different activities such as cooking, sewing and swimming.

At the end of the first week following initiation of treatment, there was a dramatic improvement of the patient's condition. This improvement was maintained throughout the remainder of the admission period, except in weeks 3 and 6 after initiation of treatment, when the patient's condition tended to be worse. The response of the symptoms to treatment appeared to be in reverse order to their sequence of onset; that is, the obsessional touching, which was the last symptom to develop, disappeared first, followed by stoppage of obscenities, then decrease of tics. The facial tics were the most resistant.

Despite the improvement in the patient's condition, the family insisted on her discharge, and although they promised to bring her as a day patient they never did. An out-patient appointment was later made, but the patient did not attend and unfortunately her condition could not be followed up.

Discussion

The results of the behavioural monitoring of this case may have important implications in terms of the aetiology of GTS, which still remains uncertain (Fernando, 1976). Nevertheless, it is known that the condition can be understood in terms of organic, hereditary, developmental, emotional and mixed models (Corbett *et al*, 1969; Enoch & Trethowan, 1979). Although it is well recognised that chlorpromazine, haloperidol and pimozide drugs have a beneficial effect in the treatment of GTS (Boris, 1968; Enoch & Trethowan, 1979; Ross & Moldofsky, 1978; Shapiro *et al*, 1983; Woodrow, 1974), the early and remarkable response of our patient to haloperidol is doubtful. This is because of the small dose of haloperidol administered and the worsening of the patient's condition at weeks 3 and 6, while she was still on haloperidol. In fact, the patient's deterioration was attributed to her contact with her family during week-end leaves, which were only granted at weeks 3 and 6, and hence reflects the bad effect of the home environment. Furthermore, there was no evidence of organic dysfunction, as indicated by the patient's good physical health and normal IQ. Therefore, the patient's improvement may be explained by the effect of the hospital environment, in terms of the better understanding and reaction of staff to the patient's symptoms, and the patient's enjoyment of the occupational therapy programme.

There is evidence in the literature that a disturbed parent–child relationship underlies the psychogenesis of the syndrome (Enoch & Trethowan, 1979). In our case this is confirmed by the results of the psychotherapeutic sessions and the family meetings, that showed the patient's inability to grow up psychologically at home, and her use of the GTS symptoms to seek the attention of her parents. The parents had 12 children, and therefore found it difficult to give attention to all of them. Moreover, the parents had always been much more attentive to the male children – a cultural phenomenon – and had applied a strict religious attitude for discipline at home.

Thus the patient acted as a scapegoat for the anxieties existing within the family by becoming the object of their perfectionist demands and punitive attitudes. The family dynamics appear to have played an important role in the maintenance and severity of this patient's illness.

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Sexual Aggressivity and Androgens

Male sex hormones are ascribed a great importance in the literature from the point of view of sexual activity and aggressive behaviour. Two cases of sexually motivated murder are described, committed by delinquents with low levels of testosterone in plasma: one a patient with Klinefelter's syndrome; the other a man after castration. Even a decreased level of androgens, either primary or secondary, is not an absolute prevention of sexually aggressive behaviour.

Male sex hormones are considered to have great importance for sexual activity, as well as for aggressive behaviour. For example, Ehrenkranz *et al* (1974) studied male prisoners and divided them into three groups: those with chronic aggressive behaviour; those socially dominant but not aggressive; and typical inmates who were neither physically aggressive nor socially dominant. The testosterone level in plasma of these men correlated significantly positively with the extent of their aggressiveness. Schiavi *et al* (1984), studying XYY, XXY and a control group of men, found a significantly positive relationship between plasma testosterone level and evidence of criminal conviction. A proportionate increase in testosterone has been noted when subjects have been divided into non-delinquents, delinquents without violent convictions, and delinquents with violent convictions.

Case reports

We had the opportunity to examine two men with considerably subnormal levels of androgens, who committed violent crimes.

Case 1

A 21-year-old worker strangled and six times stabbed a 23-year-old female worker whom he did not know closely, at their working place.

The father of the defendant is a driver, the mother is a worker. Birth and early childhood development were normal. He was brought up by a dominant mother in the company of an older brother. His performance at school was poor. He passed only seven years of school. He worked as an unqualified worker. So far he has no criminal record.

He has been interested in arms and knives since his early childhood, and played with them frequently. He further admitted sadomasochistic masturbation fantasies. Before the murder he used to touch unknown women in crowds, and he wrote obscene letters to women. He has not formed any partner relationship. He admitted that he felt sexual excitation while stabbing his victim. In physical examination, highly hypoplastic testes, with volume of about 2 ml, were found. Klinefelter's syndrome was thus assumed, and was verified by genetic examination (47, XXY). The gonadotropine levels of the man were high (FSH 26.3 U/l, laboratory standard 0–7; LH 26 U/l, laboratory standard 5–20), the level of testosterone was low (3.25 ng/ml, laboratory standard 3.5–9.5) and the steroid hormone binding globulin (SHBG) was normal (19.1 ng/ml, laboratory standard 9–21). The intellect of the delinquent was slightly low (IQ 86 according to the Wechsler–Bellevue test). The EEG recording was abnormal, with irritation phenomena most pronounced in both temporal regions. The results of a neurological examination were normal.

Case 2

In a lonely place in the woods, a 34-year-old worker murdered a 64-year-old woman, strangling her with a stick and stabbing her with a knife in the chest and in the genital region. As she was dying and after she was dead, he attempted sexual intercourse with her; because of insufficient erection he did not succeed.

The offender comes from a worker's family with a bad reputation. The older brother was repeatedly convicted of rioting. The defendant passed eight classes of a special school for retarded children. He was imprisoned three times.

Earlier he repeatedly dreamt that he attacked a woman, she defended herself and he overwhelmed her. Often he had ejaculation during this dream. Since the age of 17, he repeatedly made sexual attacks on unknown women. In most cases ejaculation occurred, without coitus. When 25 years old, he was caught in this criminal activity. After his imprisonment he was, at own demand and with the agreement of his mother, castrated. Since the operation, he has only exceptionally experienced sexual fantasies and erections; ejaculation has not occurred at all.

In repeated endocrinological examinations after the murder, strongly subnormal levels of testosterone in plasma were found (0.48–0.56 ng/ml, laboratory standard 3.5–9.5).

Discussion

Case 1

Delayed sexual development has been indicated in men suffering from Klinefelter's syndrome (Raboch *et al*, 1979). Androgen level has been found to be low from the beginning of adolescence (Topper *et al*, 1982). The sexual activity of these men in adulthood is usually low (Raboch *et al*, 1979). Abnormal EEG is often found (Nielsen, 1970), as well as low intellect (Swanson & Stipes, 1969). The most typical pattern of behaviour in 47, XXY men is a tendency toward generalised passivity and lack of ambition, combined with a minor proclivity to sudden outbursts of aggression or violence (Kolodny *et al*, 1979). They often have criminal anamnesis (Nielsen, 1970). Sexual deviations also occur (Bandman & Breit, 1984). Our case can be considered as a typical example of Klinefelter's syndrome. Only the fact that he committed a sexually motivated murder can be considered extraordinary.

Case 2

Castration, as a protective measure with dangerous sexual delinquents, is carried out in some countries (Bremer, 1959), but it is also criticised (Heim & Hursch, 1979). With most men it leads to pronounced decrease in sexual activity, but some retain the capacity for coitus for a long time after the operation (Bremer, 1959). With sexual deviants, castration strongly decreases their aggressivity, and only about 10% recidivism rate has been described (Freund, 1980). The present case demonstrates that in rare cases a sexual aggressive crime can be repeated after castration.

Conclusion

A decreased testosterone level, either occurring from the beginning of sexual development (Klinefelter's syndrome) or acquired in adulthood (castration), cannot be considered as an absolute measure preventing sexual aggressive behaviour. Other factors, such as psychological, social and organic pathological ones, are also important in influencing aggressive interactions.

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Erotomania and Senile Dementia of Alzheimer Type

A case of erotomania in a 75-year-old female with senile dementia of Alzheimer type (SDAT) is described. This case demonstrates the presence of an elaborately systematised delusion in SDAT, and the influence of the progression of SDAT on this delusion.

Erotomania, or de Clérambault's syndrome, consists of a delusional conviction of an amorous relationship with a socially unobtainable person with whom the subject has had little personal contact (de Clérambault, 1942). The subject is usually female, and the target of her delusion is frequently unaware of her beliefs and has done nothing to encourage them. The onset is abrupt and the course chronic. The target of the delusion remains constant (Enoch & Trethowan, 1979).

Primary and secondary forms of erotomania may exist. In primary erotomania, the delusion occurs in the absence of associated psychiatric illnesses. Reports of such cases have been rare, and the existence of this form has been questioned (Ellis & Mellsop, 1985). Secondary erotomania, or erotomania in the presence of a pre-existing psychiatric illness, has been associated with schizophrenia, schizoaffective disorder (Evans *et al*, 1982), paraphrenia (Teoh, 1972), major depression (Raskin &

Sullivan, 1974), bipolar affective disorder (Remington & Book, 1984), mental retardation (Greyson & Akhtar, 1977), cortisone psychosis, psychomotor epilepsy, meningioma and alcoholism (Doust & Christie, 1978). We report a case of secondary erotomania in a woman with senile dementia of Alzheimer type (SDAT). This case clearly demonstrates a systematised erotomanic delusion occurring in the course of SDAT. The age of onset of this delusion (75 years) is intriguing, and its relationship to the severity of SDAT is of theoretical importance.

Case presentation

Ms H was a 75-year-old widowed retired white female at the time of her enrolment in a research project investigating SDAT (Berg *et al*, 1982). Educated to the twelfth grade, she had married in her twenties and had worked 27 years as a telephone operator until her retirement at age 55. She was widowed at the age of 53. Following her retirement she remained highly functional until the age of 73, when she insidiously developed progressive memory loss, apathy, inactivity, amotivation, and loss of interest in hobbies. She was diagnosed as SDAT and referred to a research project studying healthy aging and SDAT. She fulfilled the strict inclusion and exclusion research criteria for SDAT, and was rated on our five-point Clinical Dementia Rating (CDR: absent, questionable, mild, moderate, severe) as mild SDAT (Berg *et al*, 1982; Hughes *et al*, 1982).

Past medical history was unremarkable. Past psychiatric history was notable for depressive symptoms in her early forties following menopause which lasted approximately one year. These symptoms were not treated and probably did not fulfil criteria for major depression. She had never demonstrated psychotic symptoms. Physical examination was remarkable only for obesity. Neurological examination demonstrated mild bilateral cogwheel rigidity at the elbows, without other Parkinsonian signs. Appropriate laboratory tests and CT scan were unremarkable.

Ms H's erotomanic symptoms began abruptly at the age of 75, after she met a 19-year-old maintenance man sent to repair her apartment's plumbing. In the days immediately following his visit, Ms H informed her family that this man was going to marry her. She referred to him as her "boyfriend", and announced their impending marriage. Her family had previously known the young man, describing him "trustworthy and reputable". Nevertheless, they investigated his contact with their mother, and found no evidence of improper advances or misleading statements. Moreover, he was unaware of Ms H's romantic beliefs concerning him. Over the next two years, Ms H talked frequently about her boyfriend and their impending marriage. She signed letters and cheques using his last name rather than her own. She would frequently tell her daughter of alleged communications between them. For example, she told her daughter, "He said he'd be out in the field today, but would be here for supper." (Actually, the only contacts the young man had with Ms H subsequent to his repairing her plumbing were brief, polite greetings he gave her outside the apartment complex.) Occasionally, Ms H would point out the window and state, "There my boyfriend is, ploughing out yonder", though her family would look and see no-one. Whenever Ms H's family attempted to dissuade her of her delusion, she merely giggled and persisted in her false belief.

Ms H had been free of all medication during the early course of her delusion and SDAT. During the two-year period of this delusion, she was treated by her family physician with trials of nortriptyline, ritalin, B12, and ergot alkaloids, with no apparent influence on her beliefs. Antipsychotics were refused. The patient had fluctuating depressive symptoms which did not qualify as a major depression. The delusion did not fluctuate with such symptoms.

Ms H had other concurrent psychotic symptoms. These included auditory and visual hallucinations involving her parents. She also spoke to her television, believing the actors were talking to her. During this two-year period, the SDAT progressed. By the time her SDAT progressed to a CDR moderate (77 years old), she no longer talked of her "boyfriend". Since then she has become aboulic, incontinent, wheelchair-bound, without spontaneous speech and severely demented. Auditory and visual hallucinations persist, although they no longer involve the young maintenance man.

Discussion

Delusions have been reported in a variety of organic brain syndromes, including SDAT. Our research indicates that many subjects with SDAT develop delusions during the course of their illness, although systematised delusions are less common.

SDAT is associated with major degenerative and biochemical changes (Blass, 1985). This case demonstrates that a particular type of elaborate systematised delusion may be initiated by such changes. Following the onset of erotomania, other delusions and hallucinations developed. These psychotic symptoms were episodic and not systematised. They, however, persisted as the dementia became moderately severe, whereas, after two years, the systematised erotomanic delusion gradually disappeared. It may be that Ms H's intellectual processes had deteriorated to the point that they could no longer maintain a systematised delusion, though they remained capable of generating fleeting psychotic symptoms.

To the best of our knowledge this case constitutes the oldest age of onset for erotomania, and the first case of its association with SDAT in the anglophonic literature. Apparently, age is no barrier to romantic delusions.

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Capgras syndrome, de Clérambault's syndrome, and *folie à deux*

A woman with Capgras and de Clérambault's syndromes transmitted persecutory, erotic, and somatic delusions to her son in a *folie à deux*. Capgras and de Clérambault's syndromes appear together more commonly than is apparent in the literature. Review of these cases shows a strong association with severe affective disorder.

The Capgras and de Clérambault syndromes have found their way into the literature with every conceivable variant, and their occurrence together in the presence of a shared paranoid disorder (*folie à deux*) is unusual. The Capgras syndrome was elaborated in 1923 by Capgras and Réboul-Lachaux as *'l'illusion des sosies'*, the belief that persons close to the subject have been replaced by identical doubles (Enoch & Trethowan, 1979). In de Clérambault's paradigm of erotomania, the patient has the "conviction of being in amourous communication with a person of much higher rank, who has been the first to fall in love and was the first to make advances". There are many extrapolations from this 'fundamental postulate'. De Clérambault attempted to separate this syndrome from other erotic paranoid states, but not all of his own cases clearly illustrated this (Enoch & Trethowan, 1979), and the paradigm has been established solely on its content, without regard to course of phenomenology. Many cases show the presence of a severe affective disorder.

Folie à deux, although described by others, was named by La Sègue and Falret in 1877 (Enoch & Trethowan, 1979). Special circumstances permit an active or primary agent to create a delusion and gradually impose it on an associate(s), who modifies and integrates the material. The individuals live a close-knit existence, isolated from outside influences, and the delusion is more readily accepted when it is kept within the limits of possibility. Virtually any theme or category of delusion can be found, including persecutory, grandiose, religious, depressive and somatic as well as erotic. Dewhurst & Todd (1956) set out three criteria for the diagnosis of *folie à deux*: positive evidence that the partners have been intimately associated; a high degree of similarity in the general motif and delusional content; and the presence of unequivocal evidence that the partners accept, support and share each other's delusional ideas.

Case report

Mrs A, a 39-year-old, unemployed, university-educated woman, was born in a displaced persons' camp and migrated to Canada in 1953. Several operations corrected a moderate scoliosis diagnosed at nine years. Although a loner most of her life, the patient married at 22 and her only son, B, was born in 1973. She felt the marriage functioned poorly, as her husband promoted her 'bouts of jealousy' by making passes at other women.

While she may have been psychotic the year of her son's birth, psychiatric admissions began two years later. Treatment with lithium, fluphenazine and antidepressants was initiated, but she was non-compliant. In late 1981 she was admitted to hospial with grandiose, erotic, and somatic delusions (the somatic delusions concerning body odour, irritants forcing her to shave her head, unusual ideas about her corrected scoliosis, leg, and clitoris). Her agitation remitted rapidly but the delusions were unaffected by neuroleptics.

B's poor school attendance was the reason for the current presentation. Mrs A changed his school because of perceived rumours of her sexual misconduct. She sought treatment for him for numerous ailments, using the appointments to keep him out of school. Many of these are conditions from which she suffers. It was impossible to determine whether any particular physical symptom originated in Mrs A or her son.

Mrs A calls him Darwin, or some Russianised variant so that others will not know his identity; B has himself taken the name of Carl as an alternate. She believes that strangers, posing as her parents, will not tell her the 'truth' about her origins, so that she suspects her 'real' ones are dead, and has thought of investigating further. Her 'mother' and 'sister' have impersonated her; she believes that a double of herself was seen in a different city.

Her first love, at 11 years, was another girl in hospital; the first male love, at 15 years, 'proposed marriage' but his parents intervened, spiriting him off to marry someone else. Several other men have 'proposed' to her. She recently imagined she was the object of affection of a Royal Canadian Mounted Police officer. She writes sexually suggestive letters and calls him daily. Mrs A published a book of verse abounding in themes of loneliness and sexuality in a vanity press.

The patient exhibited a generally euphoric distractible air, thought disorder, rapid pressured speech, derailment, neologisms, and clang associations, without hallucinations. The cognitive, physical and laboratory examination were essentially normal, as was the EEG.

B repeated his mother's persecutory and erotic delusions and was suggestive to somatic symptoms but did not maintain her Capgras syndrome or the somatic delusions with respect to her body. He was able to name only minor physical differences between the two of them, and knew that both he and his mother have the same birthmark on their chests. He was unwanted and although his milestones were normal, Mrs A wondered if he was 'slow'. He was extremely passive, with little reaction to events. He has not had any serious illnesses nor ever required admission to hospital. The boy was bright and friendly without thought disorder, depression or cognitive disturbance; affective range and expression were preserved. Physical examination was normal. His Wechsler Intelligence Scale for Children – Revised (WISC-R) Full Scale IQ was 111 (performance 95, verbal 123). His educational attainment was relatively good, considering his frequent school absences. Children's Aid has been extensively involved with trying to assure school attendance and compliance with treatment, including individual psychotherapy for him, to encourage separation and independence.

Discussion

While Mrs A satisfied the diagnostic criteria for a schizophrenic disorder, paranoid type, the preservation of affect, grandiosity, elation and lack of negative symptoms made schizoaffective or atypical bipolar affective (manic type) disorder, strong alternative possibilities; as she was not examined in an acute state, it was difficult to determine exactly how her delusions were related to mood.

Mrs A's disorder arose soon after B's birth, and she had several periods in hospital before he reached the age of three. They were abandoned to an isolated, insular and impoverished life. B, while somewhat passive, did not maintain all of his mother's delusions. Interestingly, he showed that he had incorporated her behaviour by responding to and adopting substitute, alternative names. He maintained some separateness by an identification with his father, aspiring to the same profession. In view of B's ability to appreciate that some of his mother's beliefs are untenable, and his intelligence and capacity to form relationships outside the symbiotic bond with his mother, as well as their mutual affection, immediate separation was not advised. Working with school authorities and Children's Aid, attempts were made to strengthen contact with reality by individual therapy with the son and an alliance with his mother. Her cooperation with these measures has been variable, as has her own compliance with neuroleptic treatment; contact has been intermittently maintained.

Most reports of *folie à deux* show a pattern of dominance and submission. Nine-tenths occur in families, with the mother–child combination the second most common. The inducer or primary agent must be in close proximity, be a figure of authority or identification, and be in the early or less severe stages of psychotic decompensation. Children of psychotic parents have several types of disturbance, and the conditions favouring these include a symbiotic relationship; lower than average intelligence in the child; a close identification with the sick parent; suggestibility, especially in relation to bodily feelings; passivity or submissiveness; and a marked involvement in the psychotic manifestations of the parent. The 'parapsychotic reactions' mirror the parent's illness closely, disappearing when the child is permanently separated. The conviction with which the delusion is held varies between children, and in any particular instance depends on the presence or absence of the deluded parent or reality-oriented figures (Anthony, 1969). Social circumstances, particularly poverty and isolation, play a role in shaping these influences.

The Capgras syndrome frequently has an erotic colouration. When a spouse is the object of the delusion, the patient may believe he is committing adultery with the imposter (the Alceme complex). While Sims and co-workers (Sims & White, 1973; Sims & Reddie, 1976) reported patients with affective disorder who showed both the Capgras symptom and de Clérambault's syndrome, several more cases can be discerned than have been recognised in the literature. Krafft-Ebing (case 56) in 1905 reported on a manic woman who, in addition to the Capgras syndrome, had nihilistic, dysmorphic (broken nose) and erotic delusions (pregnancy, proposals of marriage made to her). Brill (1912) discussed a man with either schizophrenia or mania who had both Capgras and Frégoli delusions along with de Clérambault's syndrome. De Clérambault's syndrome is recorded by Evans *et al* (1982) with the Frégoli symptom and by Picard *et al* (case 2) (1958) with the Capgras symptom in probable affective disorder. Seeman (case 1) (1971), using the term 'phantom lover' syndrome, and Pande (1981), the incubus syndrome, report cases of Frégoli and Capgras symptoms respectively with schizophrenia.

Shraberg and Weitzel (1980) alluded to erotomania co-existing with Capgras symptom in epilepsy. Among the patients reported by Christodoulou and his colleagues, erotomania was seen with Capgras, Frégoli and intermetamorphosis symptoms.

The two delusions of substitution and of erotic pursuit discussed above are uncommon but not rare. Individually they have been reported along with diverse delusional themes; their own association seems to occur with grandiosity, multiple delusions and possibly with the manic phase of bipolar affective disorder.

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De Clérambault's Syndrome in Organic Affective Disorder

Two Cases

Two patients with erotomania (de Clérambault's syndrome) associated with epilepsy and head trauma are presented. The delusion appeared during periods of mania or hypomania and partially remitted during depression or euthymia. Erotomania may be seen in a wide variety of psychotic conditions, including those that are organically based.

Erotic delusions include several related delusional disorders (Enoch & Trethowan, 1979). In the 1920s, de Clérambault described the most well known of these, pure erotomania. This syndrome is characterised by the 'fundamental postulate' of a woman having the conviction of being in amorous communication with a person of a higher social rank, who ws the first to fall in love and to make advances to her. Derived themes include beliefs that the object can never find happiness without her, his overt attachments or marriage are invalid, contact is made indirectly by means of his immense resources, his overt attachments or marriage are invalid, contact is made indirectly by means of his immense resources, his conduct may be outwardly paradoxical, and their relationship is universally acknowledged and approved. There is a precise sudden onset, strong affective colouration, tendency toward action, and a lack of themes not related to the principal object of the delusion (Enoch & Trethowan, 1979). Secondary or mixed forms of erotomania have shown a wide variety of other delusional themes, greater elaboration, and multiple or even non-existent love objects.

Erotomania has been classified as a variant of schizophrenia, as a paranoid disorder, as a separate disorder (*'les psychoses passionnelles*', passionate psychoses), as a non-specific delusion occurring in conjunction with a variety of psychoses, or even as an acceptable occurrence without psychopathological implications. DSM-III (American Psychiatric Association, 1980) restricts the delusions of the paranoid disorders to persecution or jealousy, thus excluding erotomania from this category. This nosological confusion is due in part to incomplete descriptions of pre-morbid history, personality and clinical phenomenology of many early cases, as well as to the lack of conformity of the derivative themes (Ellis & Mellsop, 1985). However, a mental disorder cannot be characterised by its content or cross-sectional picture alone; classification also depends on its form, associated disturbances of mood, course, outcome, aetiology, and response to treatment. We present two cases of erotomania in patients with organic affective syndromes.

Case descriptions

Case 1

Mrs A, a 39-year-old right-handed housewife, was evaluated after incarceration for harassment of a teenage woman. One year before evaluation, she began to exhibit severe mood cycles. At times she was depressed and manifested fatigue, withdrawal from contact with her family, anhedonia, poor appetite, worsening of mood during the day, crying, decreased memory and concentration, suicidal ideation and slowing of thoughts and movements. At other times she exhibited an exuberant mood, swam nude in view of others, impulsively attempted to buy a car and a house, and engaged in many futile and destructive projects.

Following one episode of depression she 'came to life' when B became a Big Sister to her daughter, claiming she and B had a special relationship sanctioned by God. Mrs A believed others were jealous of them; she and B could communicate their unbounded love without speech, and B was able to transmit thoughts into her mind. Mrs A offered her money and gifts. A crisis ensued when B ran away from her in a panicked state, after what seemed to be a homosexual advance. The young woman's father moved to cut off contact, but the patient tried to see her many times, telephoned, and sent letters and presents to the house. Violation of a restraining order resulted in a short incarceration.

The patient had been diagnosed as having epilepsy when at age six she had a generalised convulsion preceded by an abdominal aura; recurrent seizures occurred with fever and menstrual periods. The presumed aetiology was head trauma at two years of age, although three paternal second-degree relatives had epilepsy. She was treated with phenobarbital until 15 years of age, and thereafter with phenytoin. At the time the delusion began, she had been without medication for two years. Mrs A often had the sensation of numbing, with fading of external sounds, or had olfactory hallucinations; she denied formed auditory hallucinations.

Mrs A's development was normal, but she never developed friendships with others. She performed poorly in school, testing in the dull normal range, but managed to graduate from high school. She worked intermittently in clerical positions. She had been married for 14 years and had two children. She had regular pre-menstrual mood changes, including depression, withdrawal, irritability, and aggressiveness. Libido had vanished almost entirely, and she admitted to neither heterosexual nor homosexual activity. Her maternal grandmother had a mental illness of unknown type that required hospitalisation, and remitted in late life; no other family member had psychiatric disturbance.

On examination, she was alert and anxious and she had angry outbursts against her mother when the latter described the patient's behaviour. Language function was normal, though her verbal output was tangential. There was mild impairment in both verbal and non-verbal memory. Visual-spatial testing showed severe deficits and she made errors on frontal systems tasks. Wechsler Adult Intelligence Scale - Revised (WAIS-R) scores were in the low average range (Verbal IQ = 83, Performance IQ = 83). Psychomotor speed was normal. A right hemiparesis and exaggerated right-sided muscle stretch reflexes were noted. EEG demonstrated slow wave activity in the left temporal region. CT scan was normal. The patient refused treatment and her delusions persisted.

Case 2

Mr C was a 27-year-old right-handed male brought to hospital by his college roommates because of threatening and aggressive behaviour. The patient had a history of many episodes of head trauma with loss of consciousness. As a child he had suffered physical and sexual abuse in foster homes; 11 years prior to admission he sustained a non-penetrating gunshot wound to the head; three years prior to admission he was in a motorcycle accident, with two weeks of unconsciousness; and he had been in many fights in which he sustained brief loss of consciousness. Mr C had frequently used alcohol and a variety of illicit drugs (LSD, marijuana, phencyclidine, opiates) but was not intoxicated at the time of admission. He had previously been arrested for shoplifting, disturbing the peace, and possessing of illegal drugs.

When examined, the patient was expansive and grandiose. He stated that he would someday be "king of the world". His speech was pressured, intrusive, and tangential, and he made inappropriate sexual comments and inquiries. He exhibited flight of ideas and admitted to prior, but denied current, auditory hallucinations. His affect was angry and labile. Mr C had a history of many hospital admissions in the past for similar behaviour and had been treated with a variety of neuroleptic and antidepressant agents and lithium carbonate. Of these, only lithium carbonate had ameliorated his behavioural disorder. The patient had experienced brief episodes of depression, with tearfulness, hopelessness and suicidal thoughts, but he had made no attempts to kill or injure himself. The patient was adopted; no information concerning his biological family was available.

The patient had a persistent delusion that a woman acquaintance was in love with him but would not acknowledge it and appeared to reject him. He had made many attempts to contact her and had been ordered by the court to refrain from bothering her. He remained convinced that she drove by his apartment many times each day, and called him by telephone but would not converse when he answered. The delusion had persisted for several months prior to admission, and resolved during his stay in hospital.

Psychological assessment revealed normal language, memory, visuospatial abilities, abstraction, and concentration. His IQ was in the superior range. A neurological examination was normal.

The patient was treated with lithium carbonate (300 mg three times daily). His manic behaviour began to subside, but he left the hospital prematurely and was lost to follow-up.

Discussion

In the first case, a woman with bipolar affective disorder and partial complex seizures originating in the left temporal lobe, manifested a homosexual erotomanic state with the features of de Clérambault's syndrome. The delusion began during a hypomanic episode but did not continue into the depressive phase of her illness. In the second patient, de Clérambault's syndrome made its appearance during hypomanic episodes following repeated head trauma. Both patients met DSM–III criteria for organic affective disorder and manifested de Clérambault's syndrome during episodes of mania or hypomania.

There are prominent symptoms of affective disorder in many previously published reports of erotomania. Multiple episodes, a waxing and waning course, hypersexual behaviour or promiscuity, and grandiosity are common. Thymoleptic treatments have ameliorated the erotomania and the associated affective symptoms in many cases. Most patients have demonstrated an accentuation of their delusions with hypomania, and partial or complete remission with euthymia. The patients reported here also showed an association between mania and de Clérambault's syndrome that did not persist into the depressive phases of their illnesses.

Few cases of erotomania with organic aetiologies have been reported. One of the cases of '*érotomanie associée*' (de Clérambault, 1913) was a young man with probable frontal lobe injury who developed the delusions during an episode of elation. Of the six cases reported by Schachter (1977), case 1 had head trauma (with fracture of the right temporal bone) and case 6 exhibited symptoms consistent with epilepsy. Among eight patients described by Lovett Doust & Christie (1978), the delusions were linked in case 2 to prolonged ingestion of oral contraceptives, in case 3 to cortisone treatment, in cases 6 and 8 to alcoholism, in case 4 to left temporal lobe epilepsy, and in case 7 to a deep epileptogenic focus due to a sphenoid wing meningioma (laterality unspecified).

Psychiatric symptoms found in the idiopathic psychoses may be duplicated by mental syndromes with an identified organic basis. Organic delusional disorders are common and may be the sole manifestation of brain dysfunction. A large number of metabolic disturbances and structural disorders, particularly affecting the limbic and subcortical areas, have been implicated in producing organic delusional syndromes (Cummings, 1985). In addition to causing psychosis *per se*, the delusional content may be affected by the site of the lesion. Among patients with organic delusional disorders, most of those with Capgras symptoms have had lesions involving the right hemisphere, whereas a majority of patients with Schneiderian first-rank symptoms have had lesions involving the left hemisphere (Cummings, 1985). Too few cases of erotomania associated with focal brain disturbances have been reported to determine if the laterality of the lesion is significant; but like most other organic delusional disorders, the associated brain injuries have usually involved basal or temporal limbic structures. Epilepsy with partial complex seizures and temporal lobe foci appears to be particularly common among patients with focal lesions and erotomania.

Delusions may also accompany organic affective disorders, and patients with secondary mania commonly manifest mood-congruent grandiose delusional syndromes. Secondary mania has previously been reported in disorders similar to those noted in the patients reported here, including post-traumatic conditions, epilepsy, neoplasms, cerebrovascular disorders, and degenerative conditions (Cummings, 1985). The two patients described here, and de Clérambault's (1913) patient who manifested erotomania during a period of elation following head trauma represent the only reported cases of secondary mania with de Clérambault's syndrome.

The two patients reported here and the brief review of the association of erotomania with organic delusional disorders bear on the nosological position of de Clérambault's syndrome. The occurrence of erotomania in patients with secondary mania and in patients with neurological disorders without prominent affective symptoms has been documented. These observations strengthen the conclusion of Ellis & Mellsop (1985) that de Clérambault's syndrome is an aetiologically heterogeneous disorder and, like Schneiderian first-rank symptoms, may be seen in a variety of psychotic conditions. Specific treatment of the behavioural disturbance depends on the investigation and recognition of the underlying aetiology for the delusional disorder. In patients with affective disorder, erotomania is most likely to occur during manic periods, and the possibility of an organic aetiology should be considered in all patients with de Clérambault's syndrome.

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Incidence Worldwide of Schizophrenia

SIR: The critical comment by Stevens & Wyatt (*Journal*, July 1987, **151**, 131–132) on the preliminary report of the WHO Collaborative Study on determinants of outcome of severe mental disorders (*Psychological Medicine*, November 1986, **16**, 909–928) contains a number of factual errors and inaccuracies.

(a) Nowhere in the report is "worldwide similarity of schizophrenic incidence" claimed (as Stevens & Wyatt suggest); rather, the findings of the study are interpreted by us as "suggestive evidence for a more or less uniform occurrence of a 'core' of schizophrenic manifestations in different populations" (p 927).

(b) The case-finding period of the study was two years, not one.

(c) The inclusion criteria were not "either a broad (ICD–9 or CATEGO S, P, O) or narrowly defined (CATEGO S+, Schneiderian, nuclear) schizophrenia", but "the presence of a clinical diagnosis of schizophrenia (ICD 295), paranoid psychosis (ICD 297), reactive psychosis, paranoid and unspecified (ICD 298.3, 298.4, 298.8), unspecified psychosis (298.9), or one of the CATEGO classes S, P and O" (p 915).

(d) Table 6 of our paper contains data on the percentage distribution by age group of the cases in each of the 13 study sites; there is no way of inferring from such a table "large differences in age-specific incidence rates in the samples", as Stevens & Wyatt do.

(e) Figure 2 of our paper refers to *all* the patients in the study, and not to the cases in developing countries only.

(f) The reference to "inclusion of alcohol and drug-related psychoses by the authors" is misleading; in fact, we included patients with paranoid and hallucinatory illnesses associated with alcohol and drug use (ICD 291.3, 291.5 and 292.1) without evidence of organic features, provided that their symptomatology fell into one of the above-mentioned CATEGO classes. There were altogether 12 such cases (out of 1379). Brief confusional states, for example, were excluded.

Such misunderstandings aside, Stevens & Wyatt contest the significance of the findings on two grounds. First, they question the validity of the ICD–9/CATEGO diagnostic criteria employed, mainly for their lack of an in-built minimum duration requirement that would automatically restrict the diagnosis of schizophrenia to cases in which the psychotic symptoms tend to persist (as required in the DSM–III and, to a lesser degree, RDC). Secondly, they query the validity of the conclusion that, among the study subjects identified by the ICD–9/CATEGO criteria in the developing countries, there is a relative excess of patients who recover or show a milder course of the illness than symptomatologically similar patients in the developed countries.

The first point concerns the debatable issue of the diagnostic definition of schizophrenia: should it exclude, by introducing an arbitrary cut-off point, cases of recent onset that otherwise meet widely accepted descriptive criteria of the syndrome? While such *a priori* exclusion might be justified in selecting homogeneous patient samples for genetic or other biological studies, it would be totally inappropriate for epidemiological research which must take heed of the entire range of variation, temporal and descriptive, of the condition in a given population.

In designing the study, we explicitly adopted a stage-wise diagnostic approach which, at the level of primary screening, started with a 'broad' set of criteria aiming to minimise the chance of erroneously rejecting 'true' cases. Once the patients meeting the 'broad' criteria were fully assessed, more stringent CATEGO classification rules could be applied, including those defining the class S+ which is characterised by Schneiderian first-rank phenomena. The CATEGO classes are based on an assessment of symptomatology, and course is not part of their definition. However, the pattern of course and the duration of illness episodes were meticulously evaluated on the basis of previous history and follow-up data, and can be plotted for each level of the symptomatological definition. We consider this separation of the phenomenological from the temporal axis to be an asset, rather than a liability, in this kind of research, as it provides an empirical framework in which alternative diagnostic concepts can be tested.

Stevens & Wyatt doubt the validity of the diagnostic procedures employed in the study, and suggest that the study population may represent a heterogeneous collection of disorders. In particular, they suspect that the patient samples in the developing countries contain a high proportion of acute, transient psychoses which may be aetiologically different from the rest of the disorders classified as schizophrenia. In the WHO patient sample there was, indeed, considerable heterogeneity as regards course and outcome, but surprisingly little in the presenting symptomatology. This is clearly illustrated by the PSE symptom profiles (Figs 5–7 of our paper), for both the 'broad' definition of the disorder and the S+ patients. Insofar as the brief transient psychoses are symptomatologically different from schizophrenia of an acute onset, very few such cases would have slipped into the study through the PSE/CATEGO 'sieve'. However, similarity of the symptom profiles apart, there are other reasons to think that the disorders identified in the different centres belong to one category, whatever diagnostic label we put on it. In all study areas the age and sex-specific distribution of onsets followed similar patterns, characterised by an earlier onset in men than in women, while the aggregate incidence rates were very close to one another in the two sexes. This was true for both the 'broad' and the 'restrictive' definition of the cases, which reinforces our conclusion that the 'restrictive' CATEGO class S+ simply picks out the more florid schizophrenic illnesses, and does not identify a special syndrome.

The first contact incidence of schizophrenic disorders meeting the above criteria could be reliably estimated in 8 out of the 13 study areas. Two of the 8 areas were in the North of India: the city of Chandigarh, and a rural area near Chandigarh. The differences between the rates of the 'broadly' defined schizophrenic illnesses were statistically highly significant, and this is clearly stated in the report. However, the differences between the rates of occurrence of S+ type cases were not significant. The largest difference in the 'broad' rates was that between the rural area in India (4.8 new cases per 10 000 population at risk per year) and Honolulu, Hawaii (1.6 cases), a threefold difference. Although statistically significant, a difference of this magnitude in the observed rates of a low-incidence disorder such as schizophrenia is epidemiologically almost negligible.

We agree with Torrey (*Journal,* July 1987, **151,** 132–133) that none of the study sites which provided the incidence data belong to the few areas in the world where exceptionally high, or exceptionally low, prevalence of schizophrenia has been reported or suspected. We have no reasons to dispute the existence of pockets of unusual population frequency of schizophrenia, and incidence surveys in such areas would be highly desirable. Our conjecture, however, is that the incidence rates found in the WHO study represent a fair approximation to the typical, or modal, frequency of occurrence of schizophrenic disorders in most parts of the world (without being "worldwide").

As to the more favourable course and outcome of schizophrenic disorders in the developing countries, the new data do indeed support the earlier conclusions of the International Pilot Study of Schizophrenia. Table 11 of our paper shows that, independently of any differences concerning the proportion of acute onset schizophrenia (which has a more favourable prognosis) in the developing countries and in the developed countries, Third World patients with an insidious, or gradual, onset of their illnesses had a milder pattern of course than patients exhibiting the same type of onset in the developed countries. To us, this represents strong evidence that the phenomenon exists, although at this stage it cannot be explained by any specific environmental factor.

In forthcoming publications, we intend to provide much more detail on the findings outlined in the preliminary report.

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Depressive Disorder in Primary Care

SIR: Blacker & Clare (*Journal*, June 1987, **150**, 737–751) state that "only a proportion, probably less than 10%, of depressed patients in primary care are referred to psychiatric services".

A large proportion of the psychiatric literature is devoted to research into biological aspects of depressive illness and to clinical trials of antidepressants. Fifteen to twenty years ago, patients with moderate to severe depression were often directly referred to a psychiatrist before antidepressant treatment had been prescribed. During that time, and under those conditions, much pioneering work was carried out into the biochemistry of depressive illness. The situation today is somewhat different: patients are often referred to specialist research units, often within the university setting, only after a general practitioner has failed to obtain an adequate response with one or more antidepressants for periods of one to two months. Since Katona *et al* (1986) have pointed out that previous antidepressant treatment can affect biological variates for up to three weeks after withdrawal from the drug, researchers are investigating not the biological characteristics of depressive illness but the biological legacy of previous antidepressant treatments. In addition, they are also looking at a highly selected group of patients who are often half-way through their self-limiting episode.

The majority, if not all, of new antidepressant drug trials are carried out under the supervision of a psychiatrist in either an in or out-patient setting. Conclusions are therefore drawn from a population of patients who may have psychotic features or suicidal ideation and, as described above, are by definition often relatively treatment-resistant. While this traditional testing ground provides a stringent test of a new antidepressant, I feel that putative antidepressants should be tested in the general practice arena where one finds the full spectrum of depressive disorders.

I concede that such trials would be difficult to carry out. Indeed, my own preliminary survey has indicated a marked disinclination of general practitioners to test new drugs. The possibility of destroying the doctor/family relationship at the first sign of an adverse reaction was the most common reason.

It should be emphasised, as Blacker & Clare have done, that the situation may be somewhat different in the USA where one may bypass the general practitioner and contact a mental health professional directly. For this reason, biological and clinical trial data obtained in the USA in the last ten years or so may be more relevant than that obtained in the UK.

Perhaps closer collaboration between psychiatrists and general practitioners and their respective Royal Colleges could once again ensure that pioneering research is carried out in the UK.

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Khat-induced Paranoid Psychosis

SIR: As my article of 1945 was perhaps the first on the subject of khat-induced psychosis (Carothers, 1945), and since the course of the illness in the two cases there described differed markedly in one respect from that described by Dhadphale & Arap Mengech (*Journal*, June 1987, **150**, 876), I would like to add a comment to this discussion. The latter writers saw no relapses in any of their 15 patients, whereas one of my patients suffered two relapses requiring readmission within a year, and the other had three such relapses within 13 months. In explanation of this discrepancy, I can only surmise that by the time of the later article some supervision after discharge was maintained in Kenya: a supervision which was not available there in 1945.

However this may be, it seemed to me then (and does so still) that, since the great majority of khat chewers suffer no obvious ill-effects, these florid psychoses occur in people who are mentally vulnerable and who do require some organised support when discharged from hospital.

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HIV and Informed Consent

SIR: I read Davies's letter (*Journal*, June 1987, **150**, 881–882) with interest. As the natural history of human immunodeficiency virus (HIV) infection unfolds, so the neuropsychiatric complications are recognised with increasing frequency. Psychosis may accompany HIV infection (Thomas *et al*, 1985; Rundell *et al*, 1986; Jones **et** *al*, 1987) or may be the first presentation of the acquired immunodeficiency syndrome (AIDS) (Thomas & Szabadi, in press). Indeed, HIV dementia may be the first and only manifestation of HIV infection (Navia & Price, 1987).

In psychotic patients who come from high-risk groups (promiscuous homosexuals, bisexuals, drug addicts, prostitutes, and haemophiliacs), HIV infection must be considered in the differential diagnosis. A clinical dilemma is posed when such individuals are unable to give informed consent to HIV testing (or understand the pre-test counselling, etc.) by virtue of their disturbed mental state.

Venepuncture without consent on an informal patient is a battery and a form of trespass on that person. Should those psychotic individuals who are unable to give consent or are irrationally refusing (as a result of their mental disorder) be detained under Section 2 of the 1983 Mental Health Act for the purpose of assessment and further investigation of their mental illness?

I have had personal experience of two psychotic patients who were HIV positive; it was necessary to detain both under the Mental Health Act, as one had tried to throw himself in front of passing vehicles and the other had threatened to cut his mother's throat. It is envisaged, however, that there will be some psychotic individuals (from high-risk groups) who are not thought to be a risk to themselves or others but who are unable to give informed consent. In these individuals it would seem inappropriate to recommend detention under the Mental Health Act for performing a blood test. In such cases, would a doctor be ethically justified in removing some blood from a co-operative patient for HIV screening without obtaining informed consent?

The advantage of knowing HIV status is apparent. Treatments for HIV infection are available, but as yet are only palliative. The infected individuals can be identified and appropriate precautions taken, if not already being practised. A negative test should lead to further investigation of the aetiology of the psychosis.

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Behavioural Psychotherapy in General Practice (Marks, *Journal*, May 1987, 150, 593–597) – A Response

SIR: It is widely believed (by several people, in fact) that psychiatry is a highly specialised business. This notion is based upon quite false assumptions that I need hardly trouble you with here. Literally hundreds (possibly thousands) of Girl Guides, St John's Ambulancemen, swimming bath attendants and the like have found it quite easy to acquire what at first sight appear to be complex skills without any time-wasting basic science training.

In fact, you can take it from me that psychiatry is beset by unnecessary and restrictive mystique, whereas within days (possibly hours) all that needs to be known about the subject can be imparted to thousands (if not millions) of quite ordinary people with little knowledge of anything particular already in place, so to speak.

We have recently shown, for example, that the therapeutic silence in group therapy is a skill mastered easily by cohorts of bus drivers although, admittedly, they may have been a taciturn bunch to begin with. Our classes in writing benzodiazepine prescriptions, too, have clearly demonstrated that, within just a few minutes, most cloakroom attendants have grasped a skill hitherto thought to require years of training.

It is true, of course, that we have had a bit more difficulty in training a group of unemployed geography supply teachers to recognise schizophrenic thought disorder, but this may well have been due to some confusion on our part about this strange condition.

The evidence for what I'm saying comes from my own long experience, you understand. But you can take it from me and my staff that there is really nothing to it and we could within weeks (or possibly days, in an emergency) turn out thousands (possibly millions) of. perfectly well-qualified psychiatrists busily and successfully treating just about anything you care to mention.

Frankly, I can't see what all the fuss is about.

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Female Sex Offenders

SIR: In O'Connor's interesting account of female sex offenders (*Journal*, May 1987, **150**, 615–620), one example stands out and cannot go unremarked.

Case 6 is that of a young woman and her 3-year-old daughter who, after parting from an alcoholic and violent husband and father, were living with an affectionate man whom the mother was about to marry. The "offence" consisted of the man on *one occasion* allowing the child to hold his penis, with the mother's agreement. Intercourse between the couple had at times occurred, as probably in many families, in the small child's presence.

The result of these events was, not that counselling was offered to the parents, but that the mother and her cohabitee were imprisoned for two and three years, respectively, thereby presumably leaving the child parentless. It seems the mother had been seen by a psychiatrist (not the author) at the time of the trial and "No psychiatric abnormality was found...".

O'Connor could have helped us greatly by indicating the steps taken by the first psychiatrist to support this family and prevent the Court from depriving a child, at a vulnerable developmental stage, of her parents, when these were apparently affectionate and cared both for her and each other. Some discussion of how the sexual behaviour within this family came to be discovered, and defined as an "offence", would also have been of interest.

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SIR: The woman involved (Mrs C) sometimes looked after a neighbour's children. Mrs C's child told these children what had happened and they told their mother, the neighbour. She in turn informed the NSPCC, and they informed the police and social services.

Mrs C and her co-defendant were both remanded on bail for the six months before the case came to Crown Court. The child was taken into foster care and Mrs C saw her twice weekly.

Mrs C, her co-defendant, and the child were assessed on one occasion while on bail by a child psychiatry team experienced in child sexual abuse cases. In their report they expressed a willingness to continue seeing them should the parents be at liberty, but they expressed serious reservations about the child being returned to Mrs C if her co-defendant was living in the same house until a long period had elapsed.

At Crown Court Mrs C and her co-defendant pleaded guilty and were both remanded in custody for the preparation of reports. At time of sentencing, the report of the child psychiatry team, a probation report, and a psychiatric report were all available to the Court.

I too was surprised by the sentence in this case.

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Comparative Trial of a New Antidepressant

SIR: The trial by Levine *et al* (*Journal*, May 1987, **150**, 653–655) had a "double blind" design. However, tremor was four times more common with fluoxetine than with imipramine ($P < 0.01$). Does it not occur to the authors that this may have undermined the double-blind design? A notional double-blind design may be subverted by side-effects. This possibility may be addressed empirically by inviting participants to guess the patients' drug status on each occasion when drug effects are being assessed; observed guesses can then be compared statistically with those to be expected by chance, thereby providing a measure of how "double-blind" the study is.

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SIR: It is theoretically possible to undermine the double-blind design of clinical trials when there is a significant difference in response between the trial and comparator drugs. This can be either a measurable or easily-detected difference in clinical efficacy or in the side-effect profile.

It has been an unfulfilled ambition of mine to carry out a clinical study when the former occurred in favour of the new compound. In this study a significant difference was elicited with one minor side-effect which did not influence compliance, and little attention was paid to this effect until the results were analysed.

Clearly it would be possible to attempt to break a double-blind design if the difference were obvious. As this is not the intention of double-blind studies I can see little advantage being gained by attempting to break the code in advance of completing the trial. If there is any advantage in doing so I am sure that there would be many such studies where this might be more easily accomplished.

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Lithium in the Treatment of Aggression in Mentally Handicapped Patients

SIR: The report of a double-blind trial of lithium in aggressive mentally handicapped patients (Craft *et al, Journal,* May 1987, **150,** 685–689) is important. A paper published seven years ago was similar in content (Dale, 1980). Both report on patients who had been in hospital for varying lengths of time (Craft, 0–49 years; Dale, 2–42 years), the average stay being 12 years in each. My study was a retrospective one, while that of Craft *et al* was prospective. The methods of analysing the findings are very different, but the results remarkably similar.

Lithium therapy must now be seen as having a definite place in this difficult group of patients. If given earlier in the condition instead of as a treatment of last resort, as it is now, then the necessity of long-term admission may be obviated. This would be of benefit to patient, relatives, and staff.

Lithium is a relatively simple and safe form of treatment in skilled hands, which could have wider use in this clinical context. The economic advantages which would accrue from its early use have some relevance too.

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DALE, P. G. (1980) Lithium therapy in aggressive mentally subnormal patients. *British Journal of Psychiatry,* 137, 469–474.

Compensation Psychosis

SIR: White *et al* (*Journal,* May 1987, **150,** 692–694) report the case of an episode of paranoid psychosis in a patient who had suffered concussive head injury and had become involved in litigation for compensation; his delusions centered around the latter. While post-traumatic psychoses are rare, paranoid ideation occurs frequently in the course of prolonged litigation.

In the introduction to their paper the authors mention a number of old and recent aetiological issues which deserve attention. Quoting White (1981), they state that "there was no statistical difference between those victims in whom the accident had given rise to litigation and in whom the case was still proceeding and those victims where compensation was not an issue". This may be correct as far as statistical evidence goes. However, the most important factor should not be overlooked, i.e. the responsibility for the accident and its consequences. The majority of accidents in which compensation is not an issue are those occurring in the home, in sports, and on the road (in the latter case the accident victim being the responsible party). The neurotic disability process does not start with litigation for compensation, which is a much later development; it starts as soon as the victim realises that someone else (employer, property owner, driver, etc.) is at fault, followed by the attending physician's mistaken belief that he is helping his patient by ordering rest from work and activity. Compensation is but one significant factor in the development and maintenance of post-traumatic disorders. Other equally important factors subsumed under the heading of 'secondary gains' from disease are attention, protection, and over-solicitousness on the part of the patient's environment that constitute a serious obstacle to response to treatment, spontaneous recovery, and rehabilitation. Occupational and marital dissatisfaction, latent before the traumatic event of the accident, may frequently manifest themselves as symptoms of the neurotic disorder.

In analogy with the notion of iatrogenic disorders I have coined the term 'nomogenic disorders' (from the Greek *nomos* – the law) (Tyndel, 1974) to describe those psychopathological disorders in which the law and its application is playing an aetiological role in the development, enhancement, and perpetuation of these disorders. The case reported by White *et al* represents a typical example.

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Tuberous Sclerosis and Psychosis

SIR: This year the *Journal* has published two case reports of psychotic behaviour in tuberous sclerosis (TS) (Lawlor & Maurer, *Journal,* March 1987, **150,** 396–397; Clarke *et al, Journal,* May 1987, **150,** 702–703). These suggest that psychosis in tuberous sclerosis is a rare manifestation of a rare disease. We draw attention to our study of the psychiatric status of 90 children with tuberous sclerosis (Hunt & Dennis, 1987). At five years of age, over 50% showed psychotic behaviour (predominantly autistic), 59% were hyperkinetic, and 13% were severely aggressive. Psychosis therefore appears to be a common rather than a rare manifestation of the severe effects of the TS gene.

Furthermore, a conservative estimate of the prevalence of TS, for which there is no absolute marker, is 1 in 15 400 for children under 5 (Hunt & Lindenbaum, 1984). The disorder is thus not as rare as previously thought.

Our study of TS leads us to propose that profound language delay, severe impairment of social interactions, hyperkinesis, and sleep disturbance constitute a behavioural phenotype. Although not pathognomonic, this occurs so frequently in tuberous sclerosis that we consider that it should be included among the diagnostic criteria for the disorder.

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Amnesia: Organic and Psychogenic

SIR: In his review of amnesia (*Journal*, April 1987, **150**, 428–442) Kopelman fails to deal with the questions of whether individuals who say they have lost their memory, in the absence of any organic cause, are truly amnesic, and of how genuine amnesia may be distinguished from feigned amnesia. He seems to assume that all those suffering from what are often referred to as fugues and hysterical amnesias have genuinely lost their memory.

The question of psychogenic amnesia seems to have attracted little interest in recent years, and it is significant that many of the publications quoted by Kopelman are more than 40 years old. In the Podola case (Furneaux, 1960) the genuineness of claimed amnesia was rigorously probed during several days of a highly publicised trial, on the issue of fitness to plead. Several psychiatrists and other doctors gave evidence; some of them provided the unedifying spectacle, to which psychiatry seems uniquely prone, of disagreeing with one another about the most basic symptoms of the condition about which they were giving expert testimony. Leigh stated that a patient with hysterical amnesia for the whole of his life would not be able to recognise common objects or tie his shoe laces, but Stafford-Clark said that this was entirely wrong. In fact, Podola had made a number of apparent blunders tending to show that he did remember things he claimed to have forgotten, and the jury found his amnesia to be feigned.

The views of the late Sir Charles Symonds (quoted by Merskey, 1979) are of interest:

> I suspect also that all so-called hysterical fugues are examples of malingering. Forty years ago a young man was brought to this hospital (the National Hospital, Queen Square) as an out-patient to B Room, where I was working, with loss of memory for a period of a week. I had a heavy load of patients and it was a hot afternoon. I did not want to admit him and could not face the prospect of a prolonged psychotherapeutic session. I said to him "I'm quite sure you can remember if you try: here are paper and pencil. Write me out your story, and I will do what I can to help you". With that I put him into the side room, closed the door and went on with my work. At the end of my session I had forgotten about him, but as I was about to leave there was a tap on the door of the side room and he emerged with two sheets of close written foolscap.
>
> His story was this. He worked in a shop in Birmingham, had recently married, and took £10 from the till to cover the expenses of his honeymoon. He had, as he said, "borrowed" it. On his return home he found that a previous misdemeanour had been discovered, and that he had been sacked. So there he was with no prospect of repaying the £10 before his theft would be discovered. He concluded that the only way out was suicide and that the right way would be to jump off the cliffs at Lands End. So he made his way down to Lands End but there was no suitable cliff. It was dark and cold. He went to the nearest police station and declared that he had lost his memory and knew neither his name nor address. He was then transferred to hospital at Penzance and subsequently referred to Queen Square.
>
> Since then I must have seen half a dozen cases of so-called hysterical fugue in private practice and have adopted the following plan. I have said to the patient "I know from experience that your pretended loss of memory is the result of some intolerable emotional situation. If you will tell me the whole story I promise absolutely to respect your confidence, will give you all the help that I can and will say to your doctors and relatives that I cured you by hypnotism". This approach has never failed, and I have been told some dramatic stories".

It is not uncommon for loss of memory to be claimed for the period immediately surrounding the commission of an offence, such as shoplifting. In these cases it usually becomes clear that the person concerned does have some, possibly vague, recollection of events, and such clouding of memory as is present can often be explained by the effects of drugs or alcohol, or both. Classical 'hysterical amnesia' seems to be (like other dramatic 'hysterical' symptoms) even rarer now than in the past, and I have not seen a case for many years. But was it not the case that when such patients arrived at the casualty department, all identifying articles had usually been carefully removed?

I have never been able to satisfy myself of the genuineness of claimed psychogenic amnesia and suspect that many psychiatrists share this view, even though they might not feel as confident as Symonds in dealing with such patients. Do many psychiatrists now believe that genuine psychogenic amnesia exists, and if so, is that belief sustained by anything more than credulity?

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Physical Examinations by Psychiatric Trainees

SIR: Rigby & Oswald (*Journal*, April 1987, **150**, 533–535) draw attention to the unsatisfactory physical examinations recorded by psychiatric trainees. How much such shortcomings contribute to missed physical morbidity is uncertain.

Psychiatric trainees, and maybe their seniors, also pay scant attention to aspects of the clinical method which yield more information than physical examination. Hampton *et al* looked at the relative importance of history, examination, and investigations in making a diagnosis in medical out-patients. In 87% of patients, reading the referring letter and taking a history sufficed. Examination only made a significant contribution in 7%.

I have reviewed the case notes of 20 patients randomly selected from those admitted to this hospital in 1986. In one case an incomplete systems review of physical symptoms was recorded. The biological symptoms of depression were the only physical complaints mentioned in the other notes. In all cases a physical examination and a coherent history, from patient or relative, were recorded.

Hampton *et al* state that their findings cannot be directly applied to other settings, but it seems unlikely that physical examination could produce more information than questions about physical symptoms in the patients seen by psychiatrists. The arguments of Oswald & Rigby that all relevant data should be recorded apply equally to examination and history-taking.

It may be a council of perfection, but should not psychiatrists be encouraged to ask about and record physical symptoms? The skill of taking a medical history should be as enthusiastically preserved as that of performing a physical examination.

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Reference

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Depression, Dementia, and Disability in the Elderly

SIR: The studies of Good *et al* (*Journal*, April 1987, **150**, 463–470) and Griffiths *et al* (*Journal*, April 1987, **150**, 482–493) require some comment, since acceptance by the *Journal* may lead some readers to suppose that they represent a significant contribution to psychiatric epidemiology. Good epidemiological research is founded on well-defined samples, appropriate methods, and interesting questions; these studies are seriously inadequate in every respect.

Sampling: Both studies are based on data obtained from a sample of 200 old people registered with a group practice, so these subjects are not "community elderly" as asserted by Good *et al*. In the first place, elderly people registered with a GP are likely to be more alert and healthy than those who are not so registered (Murphy *et al*, in press). Secondly, although it is unclear just how the subjects were recruited, according to Griffiths *et al* only a proportion were randomly selected from the practice list. The remainder (we are not told how many) were enrolled into the study when they attended the health centre, which introduces a serious bias. Many elderly people with psychiatric disorders are unknown to the health services, and those that present usually do so with additional physical or behavioural problems. It is hardly surprising, therefore, that the authors should have found an association between their measures of dementia, depression, and disability. Thirdly, their subjects were all able to get to the health centre for assessment; this obstacle will have excluded many of those with moderate and severe depression or dementia as understood by psychogeriatricians. Findings based on this peculiar sample cannot be extended to the elderly population in general.

Methods: The authors used a questionnaire derived by them from the Hamilton Rating Scale for Depression (HRSD) which they describe as the "most appropriate in community studies in the elderly". They cite Kearns *et al* (1982) in support of this, but in fact Kearns *et al* were at pains to point out that their observations on depression rating scales in in-patient samples could not be extended to out-patient or community settings. The HRSD is valid only as a measure of severity in established cases of depression; its usefulness with non-cases remains to be demonstrated, particularly in the elderly. There is much emphasis on somatisation and psychomotor symptoms in the HRSD, and positive responses to these items may well be related more to physical illness than to depressed mood in this age group.

The authors seem to have eschewed any sort of validation – they comment on the "confused" classification of depression in psychiatry, and assert that "depression and dementia here refer to categories defined according to . . . rating scales and not to clinical diagnoses". However, they quote "prevalence" figures, and seek to impress upon us the clinical relevance and "retrospective justification" of their factors and clusters; these might have been more convincing had they provided us with some initial validation of their cut-off criteria. I was particularly struck by their comment that "a score of 0–13 [on the HRSD] would include all normals but would not exclude all depressed patients – have they not heard of false positives? It is a pity that no psychiatrists were involved in their study.

Aims: The declared aims of Griffiths *et al* were to determine the association between depression, dementia, and disability, and to identify patients at risk in the community. The first aim has been thwarted by their inadequate sampling and methodology; the second seems to have been abandoned, since there is nothing in their paper that relates to it.

Good *et al* are particularly coy about the purpose of their study, and no underlying hypothesis is discernible. Implicit in their introduction is the potentially interesting notion that normal subjects and those with depression have a common structure to their symptoms, but they have not tested this useful null hypothesis with separate analyses of normal and depressed groups. Rather, all we are given is a description of the symptom structure in the group as a whole, quite unrelated to any clinical or operational diagnoses.

Had the authors clarified their aims at the outset they might have chosen their sample and methods more appropriately, and their fine display of multivariate techniques would have been to some purpose. As it is, their efforts merely demonstrate yet again that in epidemiological research at least you can't make a silk purse out of a sow's ear – not even with cubic polynomials.

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Murphy, E., Smith, E. R., Lindesay, J. & Slattery, J. Excess mortality in late life depression. *British Journal of Psychiatry* (In press).

Sir: We were interested in the comments made by Lindesay, but consider that most of the points he raises would be answered by a more careful reading of our original papers. He reiterates reservations about the sample which we had been at pains to point out in our presentation. His criticisms underline the epidemiological problems we discussed.

We avoided arguing from the particular to the general – the subjects were 'elderly in the community', not "community elderly" as Lindesay alleges we asserted – there is a semantic difference. We emphasised that our sample was a 'good' group, described in terms of disability, and suitable for comparison with groups such as the housebound. Nowhere do we purport that the sample was random.

Our selection of the HRSD was based on a wide examination of the literature, and was not predicated on Kearns *et al* (1982) alone.

The identification of patients at risk is implicit in the final paragraphs of the discussion of Griffiths *et al* (*Journal*, April 1987, **150**, 482–493).

In Good *et al* (*Journal*, April 1987, **150**, 463–470) we stated that separate results were not presented for subsets of the sample, although the factor structure of the 'normal' subjects was similar to that of the whole sample; it would have been of little value to present a statistically non-significant analysis of the small depressed group.

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Asperger's Syndrome

SIR: There is controversy about the validity of Asperger's syndrome (AS) (Schopler, 1985; Volkmar *et al*, 1985; Wing, 1986). Although first described in 1944, interest in the condition was revived by Wing (1981). Individuals with AS are described as having: pedantic, lengthy speech; stereotyped speech; impaired non-verbal communication; Aprosodic speech; peculiar social interaction; lack of empathy for others; repetitive activities; resistance to change; clumsy or stereotyped motor movements; and hypertrophied skills or circumscribed interests. A number of investigators have conceptualised AS as part of an 'autistic spectrum', and note that no behavioural distinction can be made between AS and higher-level autism (Schopler, 1985).

We recently re-read "Children with circumscribed interest patterns" (Robinson & Vitale, 1954). This paper is of interest not only because of the lucid clinical case descriptions presented, but also in terms of the discussion following the paper by Kanner. None of the patients described fit DSM–III criteria for autism as applied by us to case report material. Furthermore, Robinson & Vitale, as well as Kanner, see these children as being different from autistics. The defining features of the children described include pursuit of special interests restricted to certain areas which have special value to them. There is an avoidance of involvement in other conceptual, social, or interpersonal areas.

We applied the above descriptive criteria for AS to each of these patients. Cases 1 and 2 were positive for four criteria each, with insufficient data for the other six criteria. Case 3 was positive for five criteria, with insufficient data for five. Interestingly, case 1 exhibited severe phonic tics (Kerbeshian & Burd, 1986). With the data available, we can only state that the cases as presented are not inconsistent with AS.

Kanner's comments may be of some general value in the controversy regarding the syndromic specificity of AS. He argues the value of an atheoretical description, and cautions against putting "the interpretive cart before the semeiologic horse". He notes that circumscribed interest patterns as a syndrome may also be found in children whom we today might describe as hyperlexic, in obsessive children, and in schizophrenic children, as well as in autistic children. Finally, Kanner lauds Robinson & Vitale for their attempts to single out specific clinical patterns as a basis for diagnostic categorisation.

It is with Kanner's thoughts in mind that we are concerned about the potential diagnostic reification of conceptualisations such as "good-prognosis autism" or "autistic spectrum". Whether AS represents a point of confluence of one or more primary syndromes, or whether it coheres as a primary syndrome itself, should be decided by studies based in data, and not by opinion. We should strive toward having the semeiologic horse pull the interpretive cart.

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Hepatitis B and Mental Handicap

SIR: At the recent symposium on hepatitis B held in London, a warning was issued that hepatitis B was the most serious form of viral hepatitis, and it was said that safe and effective vaccines were available which could prevent infection and so prevent most cases of liver cancer.

This is a timely reminder to all colleagues who work in the field of mental handicap to be alert towards hepatitis B virus, which is endemic among the mentally handicapped population, particularly now when the mentally handicapped live for much longer (Carter & Jancar, 1983) and a number of patients have been discharged from hospitals.

Between 1976 and 1980 all the patients in the seven hospitals and seven hostels for the mentally handicapped in the Bristol area were screened for markers of hepatitis B infection. Of 2239 patients, 123 (5.5%) were carriers of hepatitis B surface antigen (HBsAg) and a third of these were 'infectious' (negative for antibody to hepatitis B_e antigen). Patients with Down's syndrome were 18 times more likely to be 'infectious' carriers than those without, and male patients were six times more likely to be 'infectious' carriers than female patients.

The carrier rate decreased with age, but the proportion of carriers who were hepatitis B_e antigen positive were unaffected by age (Clarke *et al*, 1984).

It is therefore very important that monitoring of the hospital and community population of the mentally handicapped for hepatitis B is maintained and that vaccination of patients and staff takes place when appropriate.

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Nurses' Attitudes to Psychiatry in a General Hospital

SIR: Liaison psychiatry and community care appear to be having a field-day in academic psychiatric circles. But to be practically successful the ideas must percolate through to the professionals we work with: physicians, nurses and para-medical staff. Their acceptance of such ideas will be determined by the attitudes they already have towards psychiatry and psychiatrists. Hence the necessity for further clarification of these attitudes.

Thirty-five trained general nurses (SRNs) were surveyed using an attitude questionnaire (Townsend, 1978). A control group of 35 age and sex-matched nurses just beginning their training was also studied. Although the sample consisted predominantly of women, over two-thirds felt that women are no more likely to develop mental disturbances than men. Nearly nine out of ten supported the belief that adult mental disturbances can be traced to emotional experiences in childhood. Almost half genuinely thought that most suicides occur because of rejection in love. After doing an eight-week placement in psychiatry in a DGH Unit with rapid turnover and no long-stay provision, about 1 in 5 still felt that few inmates of mental hospitals ever leave hospital.

Regarding the work of the psychiatrist, two-thirds felt that we should show patients where their ideas are incorrect. After having participated repeatedly in multi-disciplinary ward rounds, a fifth continued to feel that the main job of the psychiatrist was to recommend hobbies and other ways for patients to occupy their minds. Curiously enough, two-fifths of the control group felt that a good psychiatrist acts like a father to his patients. Four-fifths of the trained group disagreed with this.

It was clear from the responses that among these nurses, a dichotomous view concerning physical and mental illness persisted. Over four-fifths of trained nurses saw little role of physical causes in bringing about mental disorder. A similar proportion also denied the role of poor diet in any manifestations of mental instability. This viewpoint is potentially dangerous, since the possibility of the elderly deprived/mentally ill presenting the mental symptoms because of subclinical vitamin deficiency is greater in a general hospital setting.

At a social level, a third of the nurses denied mental health the status of an important national problem. About half also felt that psychiatrists almost always have difficulty in telling whether or not a patient's mental disorder was curable.

In spite of all the methodological pitfalls of a questionnaire study, one message is clear. The resistance and mistrust towards psychiatry and psychiatrists is only marginally different now from what it was in the past. The days of total community care are imminent, and yet some of our colleagues are not aware of what psychiatry has to offer. While a review of the psychiatric training of general nurses is needed, a careful re-appraisal as to our own behaviour merits study.

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Phensedyl Abuse

SIR: Interest in the abuse of ephedrine (Whitehouse & Duncan, *Journal*, February 1987, **150**, 258–261) and pseudoephedrine (Pugh & Howie, *Journal*, December 1986, **149**, 798) prompts me to report the occurrence of phensedyl abuse in a group of young men in a small country town (Midsomer Norton) and the treatment of an addict.

Phensedyl is a proprietary cough linctus available without prescription. Each 5 ml contains 7.2 mg of ephedrine hydrochloride, 3.6 mg of promethazine hydrochloride, and 9 mg of codeine phosphate. There are no reports in the literature of dependence on phensedyl, although one can only echo the contention of Whitehouse & Duncan that abuse of substances containing ephedrine occurs unreported in the UK.

Case Report: A 31-year-old single man requested help in stopping drinking 2–3 bottles (200–300 ml) of Phensedyl per day. He had been drinking this for 11 years, along with about 10 friends, most of whom were still abusing it. He had also abused *Actified* and intravenous amphetamines, but was not currently doing either. He had been in prison for two short periods for stealing to pay for his habit and, although on both occasions he had not undergone serious withdrawal symptoms, he had increased his consumption since then. He had no other psychiatric symptoms. It was calculated that 200 ml of Phensedyl contained 288 mg of ephedrine hydrochloride, 144 mg of promethazine hydrochloride, and 360 mg of codeine phosphate; these amounts were prescribed in tablet form as a daily dose on a reducing regime, and the patient was seen and counselled regularly over a seven-month period. He remained drug-free for a further four months, but his course since then has been one of intermittent Phensedyl abuse and abstinence.

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Negative Interaction Between Lithium and ECT

Sir: We report a woman who developed a mild acute organic brain syndrome with normal serum lithium level, stopped lithium, recovered, and because of continuing depression was given ECT and then developed a severe confusional state.

Case Report: A 57-year-old woman with a history of recurrent unipolar depression was admitted to hospital following the death of her husband. Her father had suffered from depression, but had not sought treatment. He had taken to his bed for 10 years, mainly for depression, but eventually died of senile dementia, Alzheimer's type. There was no other family history of affective disorder. The patient had three children, but none proved to be supportive following the death of her husband.

She was admitted to hospital and treated initially with a variety of antidepressants, including dothiepin, mianserin, and Parnate, but could not tolerate amitriptyline or nortriptyline. Lithium was added to her regime, and although it initially seemed to help her depression she developed severe tremor, ataxia, and a mild confusional state. All medication was therefore stopped. She remained severely depressed and it was decided to give her a course of bilateral ECT.

After the first ECT she complained of mild myalgia and took to her bed, but recovered. ECT appeared to help her depression. However, after the second ECT she became severely confused, rambling, disorientated in time and place, and had a reversal of her sleep rhythm. She then had to be nursed in bed for about a month. She gradually recovered from her confusion and thereafter made a slow recovery. Her depression returned, but using small doses of dothiepin the depression gradually remitted over the next year. She is almost fully recovered from depression, but complains of a poor memory. There are no signs of any dementing illness.

The presence of an ECT-induced confusional state following an episode of lithium toxicity (but with normal serum level) has not been previously reported. While, in retrospect, it might have been more clinically prudent to have delayed ECT, the patient was in a state of very severe depression when this decision was made. Furthermore, we were unaware at the time that there might be any adverse reaction between ECT and lithium.

A few case reports suggest the possibility of such an adverse interaction. Hoenig (1977) reported a patient in whom ECT precipitated a severe encephalopathy, although the patient's serum lithium level was well below the toxic value. Remick (1978) reported a patient who was given ECT while on lithium therapy; after the fourth ECT she developed severe confusion and was incontinent. Six weeks after the resolution of her confusion she was once again treated with ECT, but this time with significant improvement of her depression and no confusion. In a third case (Weiner *et al*, 1980), lithium carbonate was introduced after a third ECT treatment and the patient developed a severe fluctuating confusional state.

In a retrospective controlled study of 25 patients who had both ECT and lithium, Small *et al* (1980) found that the lithium/ECT treatment group had more severe memory loss, atypical neurological findings, and a poorer response to ECT. These authors speculated that the patients on lithium might either be a more severely depressed group, or that there might be a true negative interaction between these two common methods of treatment.

Our patient and the patients reported by others suggest the latter, and we are presently investigating the possibility of a true negative interaction between lithium and ECT.

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Structural Brain Lesions in Functional Psychosis

SIR: Findings pertinent to cerebral and cerebellar pathology and functional psychosis seem to be limited and contradictory (Hamilton *et al*, 1983; Wexler, 1980). It has not yet been proven, however, whether there is a significant aetiological relationship between organic pathology and psychiatric illness in general. We present two case reports of functional psychosis: one patient with bipolar affective disorder who had radiological evidence of cerebral and cerebellar atrophy, and another with a schizophrenia-like disorder who had cerebellar atrophy. They were both illustrated by CT.

Case Reports: (i) A 49-year-old woman without any personal or family history of neuropsychiatric illness became depressed at the age of 38, with symptoms of sadness, hopelessness, psychomotor retardation, decreased appetite and sleep, and a serious suicidal attempt. She was admitted to a psychiatric clinic, but within a few days her depression changed to a manic state with increased psychomotor activity, euphoria, loquacity, and delusions of grandeur. She returned to her normal state in about three weeks, but experienced a similar depressive and a manic episode six years later. We examined the patient during her third depressive episode, at which time she was treated with tricyclic antidepressives and lithium. At six-month follow-up, the patient showed a significant deterioration and was noted to have a cerebellar syndrome with symptoms of dysmetria, dysarthria and cerebellar ataxia. Serum lithium level and blood chemistry were normal. EEG revealed nonspecific dysrhythmia, but CT of the brain revealed diffuse cortical atrophy, ventricular dilatation, and cerebellar vermian atrophy. During the following two months her condition gradually merged into a dementia syndrome which was documented by psychological tests. Later she developed a paranoid-hallucinatory and delusional state. Finally, her schizophreniform manifestations improved, but the dementia remained unchanged.

(ii) A 24-year-old female had symptoms of hearing voices and talking and acting as if they were real. She also had fears of being harmed by others, and gradually became withdrawn. Her initial symptoms had occurred at the age of 15, and she was diagnosed as having an early onset schizophrenic disorder with symptoms of auditory hallucinations, persecutory delusions, delusions of reference, lack of interpersonal relations with flatness of affect, and finally autism. In spite of antipsychotic therapy, the patient's condition worsened and she became severely disabled and dependent on others. Over the past year all psychiatric symptoms disappeared, but she revealed several neurological deficits, including left central facial paresis, left hemiparesis, horizontal nystagmus, ataxia, and dysequilibrium, all of which were preceded by headache, nausea, and vomiting. Currently the patient's neurological findings are the same, and the psychiatric condition is within normal limits. CT was carried out twice, and both times showed cerebellar vermian atrophy.

Heath *et al* (1979) found CT scan abnormalities in 50% of 264 patients with functional psychosis, 42 (32%) of whom had cerebellar vermian atrophy. Nasrallah *et al* (1981) reported 43 schizophrenic and 15 manic patients in whom CT findings were consistent with cerebellar atrophy. The main function of the cerebellum is thought to be largely motor, and especially related to co-ordination, tonus, and balance, but some of the clinical and neurophysiological studies revealed new aspects of the cerebellum in relation to autonomic, limbic, and higher cortical systems (Hamilton *et al*, 1983). Wexler (1980) pointed to right hemispheric dysfunction in manic depressive psychosis and left hemispheric dysfunction in schizophrenic psychosis.

We conclude that there is probably a sub-group of schizophrenic patients in which the symptomatology could be related to structural brain lesions, which is also true for bipolar affective disorder.

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Book Index Reviews

SIR: Book reviews rarely contain a reference to the index. Of the 137 books considered in Vol. **150** (January–June 1987) of the *Journal*, 9 indexes received some comment. Chick (p. 419), Jacoby (p. 421) and Smith (p. 134) used such expressions as "well-indexed", "comprehensive" and "excellent". Greer (p. 280) noted that "the omission of an index is a disadvantage", Kerr's "minor reservations relate(d) to the lack of an index" (p. 293), Rachman (p. 420) commented that "as there is no author index it was necessary to make a count", and Yach (p. 890) stated that "the index is somewhat rudimentary". Mayou (p. 573) "had to work hard turning from chapter to chapter and making the maximum use of the index". Wilkinson (p. 281), reviewing *Behaviour Therapy in Primary Care: A Practical Guide*, asks if "it is significant that the index contains no reference to illness, mental disorder, psychiatrist or psychiatry?"

With regard to the other 128 books, presumably some have adequate indexes, and some not. Is this information not worth recording by reviewers, especially if the book might be used for reference purposes?

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A HUNDRED YEARS AGO

The report of the Superintendent of the Richmond District Lunatic Asylum reveals an almost incredible condition of things. "The sanitary state of the institution is deplorable. The waterclosets throughout are in a sad state. In the North (male) House there are in one group of wards no waterclosets whatever that are accessible for day use, and the only accommodation for 120 men is one earth chair." Under such circumstances, it is not surprising to find that out of an average population of 1076 as many as 117 were under hospital treatment for diarrhoea and dysentery during the year, or to find that these diseases caused twenty-two deaths in the same period; while for the past ten years they have caused an average of twenty-six deaths annually. "Erysipelas is rarely absent from the house, and in some years has caused a considerable number of deaths." This state of things demands remedy, and the newly appointed superintendent deserves every support in his attempt to put matters on a satisfactory footing. The percentage of deaths on the daily average numbers was 14.8, and that of recoveries on admission was 30.

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Books Reconsidered

The Ego and the Mechanisms of Defence: Anna Freud*

This book was first published in English in 1937. In science fifty years is a long time, longer even than in politics, yet I can recommend psychiatrists to read this traditional psychoanalytic text for many reasons.

In the first place, for sentimental reasons. Anna Freud, daughter of the Master, is no ordinary analyst. Furthermore, her real contribution to psychoanalysis lay in her elucidation of the defences and in the very necessary extension of the work to children. Freudian developmental theory was largely retrospective and inferential, so that the work of Anna Freud was particularly valuable. This book is concerned with both these aspects of her work.

Another reason for reading this book is that the simplicity of the style and the immediacy of the clinical material give a powerful impression of the real nature of defence mechanisms. It is a pleasure to realise that subtle and rich descriptions of largely unconscious mental events can be described without jargon in clear, ordinary, yet striking language. Every psychologist or psychiatrist who has failed to resist the temptation to yield to jargon, however learned, in his reports or papers should read this book.

The content of the book, in my view, is still valuable. It sets out clearly the main defence mechanisms and is richly illustrated with clinical material. I know of no better guide to these unconscious processes. At this juncture, however, I can hear readers' objections that surely Freudian defences are old hat, that psychoanalysis has moved on, that modern analysts and psychiatry in general has outgrown the model of the mind implicit in the notion of defences.

This view of defences is, I think, mistaken. Thus, experimental psychology, which has always been so strongly opposed to psychoanalytic ideas, has recently become interested in unconscious processing (of information rather than affect), and concomitantly studies of subliminal perception are now more respectable in departments of psychology and there is a considerable body of experimental work relevant to defence mechanisms. I think this experimental evidence is important because it substantiates the psychoanalytic concept of defences.

Defences, given that they can be substantiated, are important for a variety of reasons. Firstly, for eclectic psychiatrists and psychologists they give valuable insights into behaviour. Notions such as denial and reaction formation are useful in unravelling presenting symptoms. Repression, too, is widely used even by workers who would refuse validity to anything psychoanalytic.

Furthermore, they are powerful in understanding complex attitudes and viewpoints in society, ones which often desperately require change. Thus to conceive of racism as projection helps us understand its protagonists and suggests methods of alteration. The violence of anti-vivisectionists, for example, and of football crowds, when seen as displacement becomes less strange. The savagery of those in favour of fierce punishment for sexual offenders and murderers, and the reaction-formation of those troubled by such conflicts, again makes sense.

Defence mechanisms, even for those who are cautious in accepting the concepts of psychoanalysis, have powerful explanatory force; they may be among the most long-lasting and useful ideas in psychoanalysis. Their lucid and elegant explication by Anna Freud seems to me well worthy of reading; this is a classic text.

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British Journal of Psychiatry (1987), **151**, 423–433

Book Reviews

Editor: Sidney Crown

Suicide Risk: The Formulation of Clinical Judgement. By John T. Maltsberger. New York: New York University Press. 1986. Pp 174. $32.00.

The bibliography in this book is far longer than the index and contains in excess of 150 quoted publications. This might not seem out of the ordinary except that these include Jones on von Kleists' suicide, Seneca's *Lucillium Epistolae Morales*, Williams' *A Streetcar Named Desire*, Goethe's *The Sorrows of Young Werther*, and Asberg on 5-HIAA in the cerebrospinal fluid. This book beckons to be read, and the reader will not be disappointed. It is short and to the point, containing many illuminating and resourceful references well-rooted in literature, history, psychoanalysis, and general human experience.

So often American authors use a language tantalisingly reminiscent of English but startlingly incomprehensible. This is also unfortunately true of many psychoanalytic tracts closer to home. However, this book admirably avoids these pitfalls and is clear and straightforward. It is divided into 5 sections with a particularly entertaining and comprehensive excursion through the vulnerability to suicide (and it is possible to write in an entertaining manner about such a subject while never being light-hearted or frivolous). The other sections include the suicide crisis, recognising the suicidal patient, the formulation of suicide risk, and the pitfalls in estimating suicide danger.

The author proves most illuminating when highlighting the basic psychodynamic elements of vulnerability to suicide and the suicide crisis itself, as illustrated by "poor Blanche DuBois who in order to maintain her mental balance had always depended on the kindness of strangers" or in patients who lose their equilibrium when their idealisation of a leader is interfered with, such as in the case of Albert Joffe, a devoted communist who before committing suicide wrote to Trotsky when Stalin excluded him from the party, "... that is why it is time to put an end to my life... I cannot go along with the state of affairs where the party tolerates in silence your [Trotskys'] exclusion".

Perhaps the weakest area in the book is the final chapter, entitled 'Pitfalls in estimating suicide danger', which leaves one feeling somewhat frustrated by the author's attempt to place the unsuccessfully prevented suicide firmly at the feet of the professionals who are unable or unwilling to fulfil his rigorous criteria for a full psychodynamic formulation. Maltsberger alludes to the psychoanalytic and other therapeutic treatment strategies which would be introduced once this assessment has taken place: "formulation is the basis as well of intelligent psychoanalytic psychotherapy of suicidal patients, but these matters are outside the scope of this book". I suspect that a book covering these aspects would be a rather different kettle of fish. In addition, if it contained controlled outcome studies of such psychoanalytic treatments it would also be very short.

Nevertheless, these are relatively minor quibbles in what is an intellectually satisfying and very readable tome. I recommend it most highly to psychiatric trainees and general psychiatrists alike, although I suspect that the majority of doctors in casualty departments who are in the front line of the initial assessment of suicide risk would find it a rather daunting prospect. The relatively high price may put it out of the reach of people who might wish to read it at their leisure.

David Roy, *Consultant Psychiatrist, Goodmayes Hospital, Essex*

Interventions for Children of Divorce: Custody, Access, and Psychotherapy. By William F. Hodges. Chichester: John Wiley & Sons. 1986. Pp 374. £38.50.

This book aims to provide principles and guidelines for professionals working with children of divorce. The author has reviewed a large number of studies, but he points out that these are mostly descriptive and largely methodologically inadequate. However, the intervention strategies are clearly illustrated with examples, and these provide a valuable resource.

The impact of divorce on children is different depending on the age of the child. Hodges argues that we need to consider the characteristics of the child and their circumstances in order to establish what one would expect the child to understand and cope with. The theoretical basis of this work is developmental, and strongly based in object relations and Piagetion theory. Within this model the impact and long-term adjustment to divorce is considered.

There are chapters on medication, custody, visitation, and single parenthood as any clinical intervention should be placed in the context of the effects of different ways families reach their present status. The chapter on mediation is particularly important, as the evidence reviewed suggests that it is superior to court-resolved battles for all concerned. It is also argued that whatever post-divorce arrangements are made for children should be monitored and evaluated. With the high rate of remarriage the author has also reviewed work in this area, as clinicians often find themselves working with such families.

The second half of the book presents various models of intervention, including individual, group, and family therapy. Each approach is carefully considered and illustrated. At the end of the book Hodges provides a list of things that are not known and need to be known when helping children of divorce: there are thus plenty of suggestions for future research.

This book is well written and easy to use as a reference book – in view of the price this seems the most likely use. The fact that the research and practices reported are from America should not deter the British reader.

NIGEL BEAIL, *Senior Clinical Psychologist, Princess Royal Community Health Centre, Huddersfield*

Attempted Suicide: A Practical Guide to its Nature and Management (2nd ed). By KEITH HAWTON and JOSE CATALAN. Oxford: Oxford University Press. 1987. Pp 211. £12.50.

The second edition of this readable book is a little larger than the first. Its main format remains the same, and it is still the most useful basic text on 'attempted suicide' which is currently available. It is valuable at several levels, from a basic guide to practical management to a good summary of recent research work.

The new additions to the text reflect various recent changes in the clinical scene. Problems concerning unemployment, child abuse, adolescence, the role of psychotropic drug prescription, and up-to-date epidemiological findings are discussed in detail. The new DHSS guidelines on the management of deliberate self-harm are also mentioned, although it would have been useful to see them in their entirety. One omission concerns the recommendations that multidisciplinary standing committees should be established in each major clinical centre, charged with the tasks of monitoring standards of care and establishing codes of clinical practice, as well as methods of education and stimulating research. One wonders how widely this idea has been implemented throughout the country.

The book closes with consideration of the way forward and the many problems which beset primary prevention of 'attempted suicide'. It is indeed a source of disappointment that no research has yet shown any form of intervention to be effective in reducing repetition, and we are still in the early stages of identifying key issues in primary prevention of 'attempted suicide'. The exhausted casualty officer, facing yet another overdose patient, may still feel justified in believing that punitive stomach washouts could be effective as a way of preventing further repetition. In due course further enquiry will surely prove him wrong, and any aspiring researcher will find within this book many good ideas which deserve further exploration.

H. G. MORGAN, *Professor of Mental Health, University of Bristol*

In-Patient Psychiatry: Diagnosis and Treatment (2nd edition). Edited by LLOYD I. SEDERER. London: Williams & Wilkins. 1986. Pp 399. £45.00.

At a time when community psychiatry is fashionable it is curiously anachronistic to find a book which is almost entirely devoted to in-patient psychiatry. Yet even day-patient care receives only a brief comment here. The narrow focus does enable certain aspects of in-patient care to be discussed in more detail than is found in general psychiatric textbooks, but the multi-author nature of the book has resulted in a considerable amount of repetition. Young psychiatric trainees are often puzzled by what actually happens in a psychiatric ward and may be uncertain about how they should spend the individual time that they have with their patients, what community and staff meetings are about, and what the aims of in-patient group psychotherapy should be. Some attempt is made here to provide the psychiatrist in training with advice and clear guidelines, and the chapters devoted to group therapy, the therapeutic milieu, and the family are particularly well-written.

Unfortunately, much of the detailed factual information one would also expect to find in such a text is only sketchily presented, and there is much that British psychiatrists would take issue with; for example, the chapter on schizophrenia uncritically presents the views of such workers as Wynne and Singer, and is heavily biased towards psychotherapeutic management. Somewhat paradoxically, another chapter, devoted to the use of the laboratory, dogmatically recommends a number of "diagnostic" biochemical tests such as the dexamethasone suppression test, again without discussing any of the controversy surrounding their use. Considering that the book is dealing with in-patient treatment, ECT is dealt with inadequately and cognitive therapy only briefly mentioned. One is left to wonder whether this is to be thought of exclusively as an out-patient technique! The chapter on alcoholism fails to discuss aims other than abstinence in any depth, and the drug addictions are notable by their absence altogether.

Overall, the book would be of limited value to those preparing for the membership examination, but the sections dealing with interpersonal and social aspects of in-patient care probably make it a worthwhile addition to a psychiatric library.

LINDA GASK, *Research Fellow, University Hospital of South Manchester*

Psychotherapy and the lonely patient. Edited by SAMUEL M. NATALE. New York: The Haworth Press. 1986. Pp 120. £19.95.

I would not recommend this multi-author book. It contains eight papers on the title subject, ranging from an intellectually flabby meditation on the relationship between religion and loneliness (Palontzian and Janigian) to an extremely sophisticated analysis of a therapist's errors in two sessions with a rather schizoid patient who was incidentally lonely, i.e. unable to make use of the objects in his environment.

There is no conceptual or editorial unity between the various papers and their approaches (cognitive, pastoral, theological, and dynamic, among others). There is therefore no consistent argument as to the status of 'loneliness', either as a primary or as a secondary problem, nor is there a consistent view as to whether it is an entity to be treated in its own right or as a management problem. In the two papers which present clinical material in any depth (Gerardi, and Richmond & Picken), the patients' loneliness derives for the most part from the denial of aspects of the self (rage, envy, and depression) which precludes contact with others in relation to whom they might experience them. In those patients where cultural alienation or social disruption (divorce or retirement, for instance) present management or counselling problems, there is no suggestion as to the extent to which psychopathology might have prevented the patients from making their own initiatives, nor as to how to understand and meet resistance to well-intentioned, commonsensical advice.

I am afraid that I find nothing in this book to advance my ideas as to how to help the many patients in my own practice who suffer from loneliness either as a result of their pathology or because of their extreme isolation.

R. R. N. CARVALHO, *Consultant Psychotherapist, St Mary's Hospital, London*

Medicine and the Behavioural Sciences: An Introduction for Students of the Health and Allied Professions. By MICHAEL PRITCHARD. London: Edward Arnold. 1986. Pp 352. £19.95.

In recent years the introduction of the behavioural sciences into the curriculum of medical education, as well as similar developments in allied health professions, has predictably led to the publication of numerous books in this area. Most of these examine the contribution of either psychology or sociology to medicine, and there are many fine examples of this kind of book. Michael Pritchard's book, however, is rather different on several grounds. Firstly, he is a doctor, and he impressively demonstrates that it is possible to bridge the contrasting worlds of medicine and the behavioural sciences. Secondly, he attempts to present both psychology and sociology in an integrated fashion, although psychology takes up the lion's share of the twenty chapters. The third unusual feature of the book is its structure.

Part 1 includes a general introduction to the behavioural sciences and an overview of psychology and sociology. Pritchard chooses to structure the next four parts according to what he regards as "four basic and integrative psychosocial perspectives". In Part 2 he presents the 'analytical/reductionist' perspective. Here he examines familiar psychological processes such as perception, learning, and motivation. There is also one sociological chapter, entitled "Social structure and the individual". Part 3 explores the 'developmental/historical' perspective, covering both individual and societal change. Part 4 addresses the issue of differences in behaviour both between individuals and between societies. There is a brief examination of social anthropology and separate chapters on personality and intelligence. Part 5 is entitled "Interactional perspective", and focuses on social psychology and clinical aspects of social interaction. Part 6 is a single chapter on "Psychosocial aspects of disease". Although this brief chapter by no means does justice to the considerable volume of research in this area, in fairness to Pritchard it should be pointed out that throughout the book he refers to clinical aspects of each subject.

I didn't find it easy to chart my way through Pritchard's unfamiliar framework, but perhaps the non-behavioural scientist would not be handicapped by this kind of limiting structural expectation. There is certainly a lot of interesting material which could be valuable to the health professional. I have some reservations, however, mainly about potential readership. There are undoubtedly many students and colleagues in the health professions who are thirsty for knowledge about psychosocial aspects of health, and I would happily recommend this book to them. For the majority who just want a basic introduction, however, I don't think this will be their cup of tea: it is likely to be too technical and in-depth for their tastes, and it lacks a central clinical focus. In fact, I regard the title as misleading. The book is basically about the behavioural sciences, and has little to do with medicine. I strongly agree with Pritchard's expressed aim to stretch readers beyond the obviously clinically relevant, but I suspect you have to first tempt them with what they are really interested in, namely illness and ill people.

ERIC BUTTON, *Lecturer in Clinical Psychology, University of Southampton*

Community Psychiatric Nursing: A Social Perspective. By SUE SIMMONS and CHARLIE BROOKER. London: William Heinemann. 1986. Pp 196. £8.95.

In a recent radio programme about medical matters an item of research, carried out by nurses, was presented. A video had been made of the research findings "rather than present them in a learned journal". "Nurses do not read such journals", was the rationale, and one wonders how that contributes to the frustrations and dissatisfactions so common in the profession and how the academic and intellectual potential of able nurses is realised.

This book is evidence that a contrary view has voice in the CPN profession at least. It is well written, well situated in a real practical world, and firmly based on research evidence which it reports cogently and coherently. It unashamedly claims a real place for CPNs in the development of new mental health services and does not waffle about sharing tasks and blurring boundaries with other disciplines. As such, the authors may have trodden on some toes, especially in Social Service departments!

However, it backs up its claim to an exciting future for CPN services by describing the role of the CPN from a wide variety of standpoints – individual case work, relationship with social networks, and service and organisational issues. It begins from the premise that socio-economic and other environmental factors are important to the development of mental ill health and sets the scene well for a readership inexperienced in these areas.

It is good to see a book by nurses and for nurses, which is prepared to embrace a wide range of mental health literature and interpret it so clearly. A must for all hospital libraries.

GILLIAN WALDRON, *Consultant Psychiatrist, Tower Hamlets Health Authority, London*

The Psychiatric Implications of Menstruation. Edited by JUDITH GOLD. Cambridge University Press. 1986. Pp 102. £15.00.

This fascinating little book, which forms a volume in the series *Progress in Psychiatry,* is made up of a series of short essays on the many aspects of menstruation and its disorders. The result is an immensely enjoyable book which is both informative and easy to read. The many contributors include psychiatrists, psychologists, and gynaecologists. The book begins with a valuable review of the many types of premenstrual depressions and discusses interactions that depressive illness can have in relation to menstruation. As a gynaecologist, I found this chapter of great value and I know that the discussion of this subject will be of great help to me in clinical practice. There follows a fascinating review by Ellisa Benedek of the use of premenstrual syndrome as a defence in law; this chapter includes descriptions of all cases where such a defence has been tested, both in the USA and in Britain.

The problem of stress in university students and its effect on menstruation is discussed, together with the role of examinations and athletics in the production of menstrual disorders.

Anorexia nervosa and its effects on menstruation is also included and the physical aspects of the problem are discussed, but I would have liked to have seen further discussions concerning the endocrine aspects of this disorder than were presented in this chapter.

A chapter entitled "Grief process after pregnancy loss", by Elizabeth Herz, was an extremely welcome inclusion as this section covered the problem of miscarriage and not stillbirth. Early pregnancy loss is rarely reviewed in standard textbooks, and such a good review of the subject was a pleasure to read. The book concludes with two chapters covering the psychological sequelae of infertility and the menopause. The latter reviews the many myths that abound concerning the end of menstruation and does much to sort fact from fiction in this subject.

Overall this is a useful little book from which I have learnt a great deal. It covers the interactive area between psychiatry and gynaecology, about which there is often little understanding and frequently much confusion. By reading this book I have become much better informed. I would recommend it most strongly not only to psychiatrists, at whom it is primarily aimed, but also to gynaecologists.

ANNE M. JEQUIER, *Senior Lecturer/Honorary Consultant in Obstetrics and Gynaecology, Queen's Medical Centre, Nottingham*

Premenstrual Syndrome: Current Findings and Future Directions. Edited by HOWARD J. OSOFSKY and SUSAN J. BLUEMENTHAL. Cambridge: Cambridge University Press. 1986. Pp 95. £15.00.

The premenstrual syndrome is a difficult subject both to understand and to treat. Its aetiology is not defined, its pathophysiological basis is not understood, and therefore its treatment must, to some extent at least, only be empirical. Indeed, there are some who do not think that the syndrome even exists. However, there cannot be many gynaecologists who face the apparently increasingly frequent problem in the clinics that would agree.

This book, which forms a volume in the *Progress in Psychiatry* series, addresses the whole problem of the premenstrual syndrome and the result is a most informative and useful review. It begins with an overview of the affective disorders and their relationship to premenstrual depression. The close association between the two is described, and the interaction between depressive illness and the premenstrual syndrome outlined in a most helpful way.

There follows an interesting chapter concerning the biology of premenstrual changes, which consists of a careful review of the 'hard' evidence that hormonal changes induce premenstrual syndrome. It would appear that the monamine oxidases may be of great importance.

A chapter concerned with menstrually related mood disorders includes useful methodology of visual analogue scales and other self-rating schemes. The authors also discuss in detail how premenstrual depression may be mimicked by many of the affective disorders that can occur episodically. The interaction between affective disorders and premenstrual depression is further explored. As a gynaecologist, I am sure that my management of patients who complain of menstrually-related depression will be considerably improved by my reading this chapter.

The evaluation of premenstrual syndrome, together with general advice on treatment of less severe forms of the disease, is clearly described in a good and well-balanced central chapter. The management of more severe forms of the syndrome is also fully reviewed, together with the treatment of any other co-existing psychiatric disease. It is pointed out that few controlled studies are available, and that there is scant evidence for the existence of any therapy of proven value for premenstrual disease. The volume concludes with a chapter concerned with cognitive approaches to the treatment of premenstrual depression and a chapter on research techniques used to study the premenstrual syndrome.

This is a very interesting and valuable little book. Although it is written exclusively by psychiatrists, it will be of considerable value to gynaecologists who probably see as much of this disorder as anyone. It was a pleasure to read such a concise and objective review of this difficult problem.

ANNE M. JEQUIER, *Senior Lecturer/Honorary Consultant in Obstetrics and Gynaecology, Queen's Medical Centre, Nottingham*

Masters of Madness: Social Origins of the American Psychiatric Profession. By CONSTANCE M. MCGOVERN. Hanover, NH: University Press of New England. 1986. Pp 262. £18.00.

Reading this book I experienced a strange feeling of déjà vu: it all seemed so familiar. And then it dawned on me that this was because the history of American psychiatry is essentially the history of English psychiatry told with an American accent.

The Association of Medical Officers of Asylums and Hospitals for the Insane was founded in England in 1841, whereas the American analogue, the Association of Medical Superintendents of American Institutions for the Insane (now the American Psychiatric Association), was founded three years later in 1844. The origins of these associations in both countries lay in the expressed wish of well-intentioned men to pool their experience with the earnest desire of improving the treatment of the insane.

There were differences in emphasis, of course, but the rise and the fall of the asylum systems in England and America ran roughly parallel courses, although it is in the context of the demise that the closest approximation can be seen. What is most remarkable is the sharp division of opinion which has arisen in both countries as to the desirability of closing the mental hospitals.

This division could not be better illustrated than in the quotations given at the beginning of Chapter 7, which are the declared views of two Presidents of the American Psychiatric Association. In 1958, Harry C. Soloman opined, "I do not see how any reasonable objective view of our mental hospitals can fail to conclude that they are bankrupt beyond remedy . . . [they] should be liquidated as rapidly as can be done". However, in 1984 John A. Talbott had occasion to write, "Our public facilities are deteriorating physically, clinically, and economically: our chronically ill are either 'transinstitutionalised' to nursing homes or deinstitutionalised to our cities' streets".

It seems a pity that this most readable and informative book should be marred by occasional lapses in scholarship. For example, there is not and never has been a "British Association of Medical Officers of Lunatic Asylums". Again, the author alleges that at the trial of Daniel M'Naghten (*sic*) the court decided that a person was insane if he could not distinguish right from wrong. The court decided no such thing; the so-called right/wrong test was incorporated in the McNaughton Rules which were formulated after the trial.

HENRY R. ROLLIN, *Emeritus Consultant, Horton Hospital, Epsom*

What is Epilepsy? The Clinical and Scientific Basis of Epilepsy. Edited by M. R. TRIMBLE and E. H. REYNOLDS, Edinburgh: Churchill Livingstone. 1986. Pp 350. £40.00.

There have been many books on epilepsy in recent years, including several involving one or other of the editors of this book. It is easy to justify the production of these volumes on the basis that epilepsy is a common problem, but non-specialists may be forgiven for wondering whether there is really sufficient new information to require the use of so much paper. The present volume has arisen from the symposium which took place in July 1985, "To review the present state of knowledge and to discuss its relationship to the larger, central question; what is epilepsy?". The latter is a question of philosophical interest, but is not of much importance in clinical practice, in which an entirely empirical approach is taken to all such questions. This book has, in fact, much to commend it. It consists of 24 review chapters, each well-written and representing an authoritative view. Even hoary old classics such as the classification of seizure disorders make interesting reading. The scope of the book ranges from a historical introduction, through discussions of problems of epilepsy in children, differential diagnosis from other paroxysmal neurological disorders, EEG, seizure monitoring and depth electrode studies, hysterical seizures, and other interactions of psychiatry with epilepsy, to a number of concluding chapters on monkey models of epilepsy and new advances in pharmacology, largely of an experimental nature. Thus, there is something of interest not only for clinicians, including paediatricians, psychiatrists and neurologists, but also for the basic scientists. Each chapter is complete in itself and the reader is spared transcripts of the discussions that no doubt occurred at the meeting until the last two chapters. I recommend that the reader delete these from his copy: there really cannot be any point in printing sentences such as "I think that is an important point... it is a very important issue". The book concludes with a brief index, but unfortunately this is woefully inadequate. In view of the discrete nature of each individual chapter this makes it extremely difficult for the reader to find particular subjects. Naturally, many are covered in different chapters in different ways, and the only thing to do is to read through the whole book! Nonetheless, this book is generally more useful, more readable, and better written than other recent volumes on epilepsy and it deserves to be widely read.

MICHAEL SWASH, *Consultant Neurologist, London Hospital*

Liaison Psychiatry: Mental Health Problems in the General Hospital. By JOAN GOMEZ. Beckenham: Croom Helm. 1987. Pp 276. £22.50 (hb), £10.95 (pb).

This textbook of general hospital psychiatry is an introductory one for trainee psychiatrists, medical students, and general physicians and surgeons. After four general chapters each area of medicine is covered in varying degrees of detail.

Unfortunately, the first chapter may alienate physicians and surgeons, especially those who do have a positive attitude to psychiatry and prescribe psychotropics in adequate doses. The medical student and trainee psychiatrist may be put off by the daunting list of 24 tasks that the liaison psychiatrist "could and should do", and the established psychiatrist may be irritated because this book teaches that psychiatry is a 'soft' subject (even though the author emphasises the need to establish 'hard' data), that the psychiatrist is the one who must find out why a patient harmed himself, and that memory impairment is a feature of depression. This is a pity, because the book does contain some useful advice arising out of the author's extensive clinical experience.

By repeatedly emphasising the role of the liaison psychiatrist (he can help all concerned in the care of the dying, yet has no panacea) Gomez gives the impression of a special area of clinical practice rather than a set of skills that all doctors should adopt. The assessment of patients is not sufficiently direct and the psychological treatments not presented in sufficient detail for physicians to use this as a handbook. The critical trainee physician and psychiatrist will be irked by the lack of research data, and references are quoted in a rather haphazard way. The prevalence of depression among patients prior to hysterectomy, in neurological units, and in those with irritable bowel syndrome is dealt with very superficially and a book of this nature requires more than a short paragraph for subjects such as hypochondriasis, and the relationship between life events, depression, and physical illness.

Some may find this a useful text, but the rapid development of research findings relating physical and psychiatric disease surely merits a more exciting textbook on this subject.

FRANCIS CREED, *Senior Lecturer and Honorary Consultant Psychiatrist, Manchester Royal Infirmary*

Treatment of Multiple Personality Disorder. Edited by BENNETT C. BRAUN. Washington: American Psychiatric Press. 1986. Pp 206. $17.50.

This book is a collection of chapters by different authors, devoted mainly to the treatment and prognosis of multiple personality disorder (MPD). It is evident that most of the authors have worked together and share the same views of the disorder, which they have conveniently categorised. They share a model of the aetiology of MPD which includes: (a) an inborn capacity to dissociate, usually identified by excellent response to hypnosis; (b) repeated exposure to an inconsistently stressful environment in which the subject has separated the good and the bad into two or more historical chains; and (c) an overwhelming traumatic episode to a vulnerable person which induces dissociation into the previously constructed partial memory and personality. Thirteen basic issues in psychotherapy for MPD are identified: developing trust; making and sharing the diagnosis: communicating with each personality state; contracting agreements about the limits of patient behaviour; gathering history; working with each personality state's problems; undertaking special procedures, such as hypnosis, which have been found particularly helpful in the treatment of MPD; developing communications between the different subpersonalities; achieving resolution and integration; developing new behaviours and coping skills; using social support systems; consolidating gains; and follow-up.

The accent is on individual psychotherapy, but there is also a chapter on the use of group psychotherapy as an adjunct to individual work, the development of social support systems, and the use of drugs. Different subpersonalities in the same individual react differently to medication. This is a fascinating area, which if extended could complement the classic study of 'the pharmacology of placebo'. There is a chapter on the diagnosis, commentary, treatments, and follow-up of 52 patients with MPD. The average number of personalities is 15.4, the commonest number being 5 personalities and the maximum in this study being 110 sub-personalities in one individual. The average period of treatment was 22 months. Relapse was much commoner in patients with 18 or more personalities, in whom 60% had partial or complete relapse. Patients with 17 or fewer personalities had an 8% relapse rate.

In North America, the diagnosis has become increasingly common – even in 1980 there were only 200 cases recorded in the literature, but since then there have been many series published, some of them in the hundreds. It is difficult to know how much of this increase presents a true increase in the affected population and how much is due to an increasing awareness among psychiatrists resulting in more accurate diagnosis. It may also be possible that now that the diagnosis has become fashionable, it may be over-diagnosed. Patients may also have learned to conceptualise their problems in ways that attract professional and lay interest. There seems to be an important relationship between child abuse and the later development of MPD, and it is of great interest that the increase in the diagnosis of MPD parallels the increased awareness of child abuse which has been a feature of the past 10 years. Many MPD patients have been previously diagnosed as borderline personality, normally considered difficult to help psychotherapeutically in contrast to the excellent results described in the volume under review. It may be that the authors' cases were easier ones, or it could be that the diagnosis of MPD and the specific psychotherapeutic strategies used with these patients is a more helpful way of viewing some cases of borderline personality.

Where there are very sudden changes in mood of patients with a 'personality disorder' and the appearance of 'psychotic' phenomena in patients whom we are fairly certain are not psychotic, we may find with further enquiry that the phenomena may be more readily understood in terms of MPD. It remains to be seen whether the experience of this syndrome in the UK will compare to North America, but if it does this collection of essays will be a helpful guide in its diagnosis and treatment.

OSCAR HILL, *Consultant Psychiatrist, Middlesex Hospital, London*

Handbook of Studies on Schizophrenia. Part I: Epidemiology, Aetiology and Clinical Features. Part II: Management and Research. Edited by GRAHAM D. BURROWS, TREVOR R. NORMAN and GERTRUDE RUBINSTEIN. Amsterdam: Elsevier Biomedical Press. 1986. Part I: Pp 246. $72.25. Dfl. 195.00. Part II: Pp 313. $250.00.

These two thin volumes are not conference proceedings, but contain a collection of invited contributions from authors from a number of countries, including Australia. Every conceivable aspect of schizophrenia is covered; there is an emphasis on biological aspects, but chapters on social and family factors, patient services, and even schizophrenic art are included. The standard in general is very high. Many of the chapters provide balanced, detailed, and up-to-date reviews which are exhaustively referenced. A minority are less than comprehensive, and one or two authors seem content to review mainly their own work. Most impressive are the ten or so chapters relating to aetiology and pathogenesis, where the cover is authoritative. The chapters on pharmacotherapy are wide ranging and informative, and those on family therapy and psychotherapy are clear and even-handed. Unfortunately, there is a relative neglect of the clinical features of schizophrenia: the three chapters devoted to this are individually of a high standard, but several important issues are alluded to rather than examined in detail. However, specialised topics such as schizophrenia in epilepsy, organic states, and the puerperium are given a great deal of attention.

This handbook is of general interest; it provides readable accounts of many important topics, and would thus make a valuable addition to a psychiatric library. It would also be of great assistance to anyone with research interests in schizophrenia. Finally, to those, like the reviewer, who have had to teach on schizophrenia at a postgraduate level it is probably indispensible.

P. J. MCKENNA, *Lecturer in Psychiatry, High Royds Hospital, West Yorkshire*

The Sociology of Mental Disorders (2nd ed.). By WILLIAM W. EATON. New York: Praeger. 1986. Pp 281.

Eaton, who is a psychiatric epidemiologist at John Hopkins University, has responded to the success of the first edition of his sociological primer in psychiatry by incorporating a number of important recent developments. These include DSM-III and the NIMH Epidemiologic Catchment Area Program; more surprisingly, he has added three literary first-person accounts of mental disorder, including an extract from Sylvia Plath's *The Bell Jar*. The social approach here begins by separating "bizarre behaviours" from the rest of human life, and then considering them as either disease or deviance. This is followed by considering the social epidemiology of mental disorders, in which it is rather surprising to find demoralisation as a major category. The remaining sections deal with social psychology, the family, collective behaviour, organisation of treatment, socio-economic stratification, and mental disorder in the modern world (with emphasis on the views of Marcuse, but no mention of Basaglia).

The limitations of present-day sociology in confronting the issue of psychiatric disorder are, on the whole, frankly acknowledged here. For instance, the conceptualisation of social supports and life events stressors is not complete because of the failure to separate the two clearly; there is an absence of intensive population-based family studies; and data on mental disorder have not yet been brought into a comprehensive theory of ethnic and class stratification. On the other hand, some yawning gaps are skated over – family theories of the aetiology of schizophrenia are discussed without reference to either the absence of control samples or the excess of perinatal damage in those who later become ill.

This is generally a very lucid and jargon-free account of the relevance of sociology to psychiatry, which is likely to be useful to trainees in any of the mental health professions, if supplemented by, for instance, *Ideas on Institutions.* The focus, though, does tend to be strongly more American, and Europeans will be surprised to read that "The role of left-fielder entails expectations about fielding fly balls", as they are unlikely to have any such expectations at all.

HUGH FREEMAN, *Editor, British Journal of Psychiatry*

Treatment of Affective Disorders in the Elderly. Edited by CHARLES A. SHAMOIAN. Cambridge: Cambridge University Press. 1986. Pp 84. $15.00.

There are two cuckoo eggs in this slim book; a survey of the dexamethasone suppression test and platelet monoamine oxidase in cognitive impairment and depression, and a retrospective case-note admission study examining the hypothesis that age of onset of depression is associated with increased risk of delusions, unconvincingly disclaiming referral artefact: both would have been better published (or not, perhaps) elsewhere. There are five better eggs, including a brief survey of controlled trials of antidepressant medication in elderly patients, a very useful review of the cardiovascular effects of tricyclic antidepressants, and a helpful synopsis of the pharmacological treatment of depression in patients with cardiovascular disease. The other reviews – of mood disorders in post-stroke patients, and of kidney function and lithium treatment in the elderly – are less useful, in that the former preaches to the converted, while the latter is rather sketchy. There is useful material in this book for those responsible for psychopharmacological treatment of the elderly in hospital.

A. J. D. MACDONALD, *Senior Lecturer in Psychogeriatrics, Hither Green Hospital, London*

Living with Teenagers. By MARTIN HERBERT. Oxford: Basil Blackwell. 1987. Pp 238. £6.95.

This is a sound guide to adolescence, primarily intended for parents and teachers, by an author who is well-informed and able to discuss the subject in a balanced, intelligible way. Herbert deals with the nature of adolescence, independence versus authority, emotional difficulties, anti-social behaviour, sexual activity, school and work, including school problems and unemployment, and ways of helping when problems arise. The author is a psychologist, and throughout the book there is a tendency towards the methods of clinical psychology – behavioural psychology, problem-solving techniques, and cognitive approaches; commendable, but rather dull and systematic, especially the various check-lists. However, social and psychodynamic aspects of adolescence are mentioned, and the book is enlivened by occasional cartoons reproduced from *Punch* and elsewhere. This is a steady, thorough and serious book and I would recommend it for a short-list of books on this theme for the general reader.

DEREK STEINBERG, *Consultant Psychiatrist, Bethlem Royal Hospital, Kent*

Epilepsy in Young People (Portsmouth Symposium, 1986). Edited by EUAN ROSS, DAVID CHADWICK and ROBERT CRAWFORD. Chichester: John Wiley. 1987. Pp 169. £21.95.

The editors present a sound and sensible guide to epilepsy in adolescents suitable for any in-coming houseman, even if the slightly patronising title suggests, rightly, that it is based on symposium proceedings. Chapters on pregnancy, when to start treatment, when to stop, and how to explain epilepsy to a child are particularly meritorious. A weakness is the lack of consideration of the management of behavioural problems associated with epilepsy, but there is a superb chapter by Gregory Stores on non-convulsive status epilepticus in childhood presenting with essentially behavioural manifestations. In the ensuing discussion, Michael Trimble includes the phenomena of psychotic patients with spike-wave discharges confined to the limbic system and scanty epileptic changes on surface electrodes. In the light of new investigative techniques discussed in the symposium, can we persuade the same team to tackle the vexed topic of prolonged states of coma, confusion, and catatonia in children?

E. M. R. CRITCHLEY, *Consultant Neurologist, Royal Preston Hospital, Lancs*

From Asthma to Thalassaemia: Medical Conditions in Childhood. Edited by SARAH CURTIS. London: British Agencies for Adoption and Fostering. 1986. Pp 159. £7.95.

This short book, designed for social workers and those involved in the adoption of children with medical conditions, has its main value in the list of references at the end of each chapter. Most of the introductions are too sketchy to be helpful. Exceptions to this are those on cardiac conditions and cerebral palsy, which could have been models for the other chapters. Conditions such as Huntingdon's chorea and schizophrenia sit rather uneasily alongside muscular dystrophy and malignant diseases. The general chapters were too general to make real connections with the more specialist chapters on particular diseases, and some of the advice and information seems to be distinctly old-fashioned. I cannot, therefore, recommend the book as a whole to social workers and others who seeks an easy guide to medical conditions in children placed for adoption. Perhaps such a book could be used for those who are likely to adopt children with medical needs, rather than for professionals; it might be a good source book for running groups of potential adopters. It suffers from breadth of coverage, and I think that most people who adopt children with particular needs would seek much more detailed information on a practical level about a particular condition from which the child they are adopting suffers. It is a constant complaint of parents with children with all kinds of medical and psychological disturbances that they are not well enough informed by the professionals. I think this book is only a very small step towards informing professionals about particular problems, and sadly I think therefore it is not the best source of information for the conditions covered.

LES SCARTH, *Consultant Psychiatrist, Royal Hospital for Sick Children, Edinburgh*

Post-Traumatic Stress Disorder in Children. Edited by SPENCER ETH and ROBERT S. PYNOOS. Cambridge: Cambridge University Press. 1986. Pp 186. £15.00.

The contributions in this book are based on 'first-generation' descriptive research relevant for the new DSM-III category of post-traumatic stress disorder as it applies to children. They outline children's responses to a variety of stresses which include witnessing of acts of violence (homicide, rape, suicidal acts), traumatic bereavements, childhood cancer, physical and sexual abuse, war and migration, group kidnaps, and natural disasters such as volcanic eruptions.

Some interesting observations are made which provide useful clinical insights. However, the diversity of situations addressed and the individuality of the approaches used to study them result in an unsatisfactory lack of cohesion, and the inclusion of children's responses to such varied traumas under the single category appears contrived. If a clear message emerges from this book, it is the need for further discriminating, clarifying work into the post-traumatic reactions of children.

ELENA GARRALDA, *Senior Lecturer in Child Psychiatry, University of Manchester*

The Psychoanalytic Study of the Child. Vol 41. Edited by ALBERT J. SOLNIT and PETER B. NEUBAUER. London: Yale University Press. 1987. Pp 667. £50.00.

This series has now reached its first volume in as many years, which would be testimony to its worth even if the list of distinguished contributors over the years (Anna Freud, Bowlby, Margaret Mahler and others) did not include many of the best-known names in the field of psychoanalysis and developmental psychology.

There are no names as familiar as these in volume 41, which continues the tradition of including a large number of contributions (28 chapters) by an even larger number of authors. The contributions are divided into five sections under the headings Psychoanalytic Theory, Trauma, Child Development (subdivided into childhood and adolescence), Clinical Contributions, and Applied Psychoanalysis. There is also a chapter on 'The executive functions of the ego', which is a wide-ranging discussion of psychodynamic approaches to the conceptual problems of free will and autonomy. Despite its title, much of the book is concerned with adult psychoanalysis, or with issues relevant to adult psychology.

A book such as this is one to be dipped into rather than read from cover to cover. However, the contents pages do not seem to offer much of immediate interest to the general reader, and indeed the language of the titles would deter many. Nathanson on 'The empathic wall and the ecology of affect' and a multi-authored chapter on 'Special solutions to phallic-aggressive conflicts in male twins' are cases in point. On further investigation the contributions range from thought-provoking (Edelson on 'Causal explanation in science and psychoanalysis') through workmanlike, but unexciting (Anthony's chapter on 'The contributions of child psychoanalysis to psychoanalysis', for example) to provocative (Abrams' chapter entitled 'Disposition and environment' rather mischievously shows how different psychoanalytic theories can explain the carefully researched observation that twins separated at birth turn out to be temperamentally very similar even when the nurturing families are very different).

What then might be the value of this collection to the general psychiatrist? Psychoanalysis both as a theoretical and practical discipline has contributed more to the day-to-day practice of psychiatry than is generally acknowledged, particularly to the general belief that any formulation of a psychiatric problem is incomplete without understanding of the patient's history, development and early relationships. This book is in the main concerned with the exploration and clarification of patterns as normal and abnormal development. There is also a great deal of interest to those with little interest in the practice of psychotherapy but who maintain an intelligent concern for art, philosophy, or science. It is a tribute to the editors that in the main the contributors to this volume follow the suggestion of one of the authors represented (Edelson): "Psychoanalysis is a body of knowledge that is important to a wider intellectual and scientific community than that comprised by psychoanalytic practitioners. In its documentation it should follow the scholarly practices, and accept the standards, of that community."

GRAEME MCGRATH, *Lecturer in Adult Psychiatry, University of Manchester*

Advances in Therapies for Children. By CHARLES E. SCHAEFER, HOWARD L. MILLMAN, STEVEN M. SICHEL and JANE R. ZWILLING. San Francisco: Jossey-Bass. 1986. Pp 450. £34.00.

If you want a quick, helpful and fairly current reference to specific treatment for a wide variety of child and adolescent disorders such as nightmares, shyness, temper tantrums, restlessness or overdependent relationships with adults, look no further. This is the latest volume in the series of reference books on treatment of emotional and behavioural problems in children and adolescents. Readers familiar with the earlier volumes will recognise the basic structure. There are six chapters devoted to classes of clinical problems, including neurotic behaviours, habit disorders, and disturbed relationships with children. Each has an introduction followed by sub-sections dealing with more specific clinical problems such as school phobia, temper tantrums, fire-setting, and social isolation, among many others. Every sub-section has an introduction followed by a brief but informative account of published reports describing different treatment techniques for the particular problem. These specially written accounts are the core of the book; they provide sufficient information to guide the implementation of treatment, and are accompanied by a commentary that draws out some of the important features of the treatment. The selections are unbounded by theoretical constraints – indeed, the eclecticism is intentional. Finally, each section ends with annotated references to yet other treatment approaches or to issues related to the same problem.

The authors give a number of reasons for eclecticism. One is in response to an increasingly widely recognised phenomenon in treatment: that treatment has to be specific to the problem; neither psychoanalysis on its own or behaviour therapy on its own will suffice to treat the diversity of child and adolescent problems. Instead, practitioners must be eclectic, chosing treatment to match problem and cicrumstances. Furthermore, recognising that treatment effects do not generalise, increased attention must be given to involving parents. Finally, they have been influenced by the trend towards community-based prevention and education. The selections reflect these views.

The danger of collections such as this is that they encourage technique-centered clinical practice. Although this has some uses, it also has many limitations. These dangers apart, this volume and its predecessors should be useful to trainees and others early in their careers. Experienced clinicians will also find it enormously helpful as a way of keeping up with current approaches to treatment. It is, after all, difficult to peruse the diversity of journals that publish treatment reports in order to keep up to date. With periodic updating this series will continue to provide an important service to clinicians. At this sort of price, however, it will be a candidate for departmental rather than individual budgets.

MICHAEL BERGER, *District Psychologist, St George's Hospital, London*

The Psychotic Core. By MICHAEL EIGEN. Northvale, NJ: Jason Aronson. 1986. Pp 387. $30.00.

Eigen's focus is not limited to those individuals who are psychiatrically diagnosed as psychotic or borderline. Rather, this book is a rich phenomenological and psychodynamic exploration of "the mad dimension of life", a discussion which has both breadth and depth. He argues that "overtly psychotic individuals make up a relatively small proportion of both the general and patient population, but psychotic attitudes and states can be components of a broad range of emotional states and mental disorders".

Drawing on a variety of analytic theorists' contributions, especially Bion, Eigen considers various dimensions of psychosis: hallucination, mindlessness, disturbances of boundaries, domination of mind by hate, and disturbances of epistemology, of knowing and the sense of knowing. Eigen demonstrates the links between these disturbances in overtly psychotic subjects and their more subtle manifestations in 'normal' life – that psychotic individuals are struggling with issues that concern us all, that are an inherent part of being alive, for example such as pain.

The psychotic hate which Eigen addresses is often directed not only against an external world perceived as threatening or ungiving, but also against the self, its needs, and its love because these are a source of pain. However, while hate is destructive, even more devastating in their anti-life aims are the self-erasing functions described by Bion, denoted by a minus sign: $-K$, $-L$, $-H$ refer to the psychotic individual's tendency to attack the linking activities of knowing, loving, and hating. Eigen gives descriptions of attempts to omnipotently annihilate emotional life in order to avoid emotional pain. For example, one patient spoke of a 'shredder' which operated to obliterate any emotional meaning that threatened to arise. She conveyed how therapy became the major source of pain in her life because if only she could obliterate the therapist she would not need to know how alone she felt: "she and shredder would live in peace".

Eigen argues that a sense of catastrophe may lie at the origin and core of mental life, in healthy development functioning as a signal to initiate urgent attempts to engage the caregiver. When catastrophe loses value as a signal and threatens to become the pervasive reality, the subject may adapt by various forms of blanking out, or mindlessness – much like some of the autistic phenomena described by Meltzer *et al* and Tustin.

Eigen argues that Freud's work was to "initiate the most detailed investigation in the history of Western thought into the way pleasure is substituted for pain". The study of psychosis further elaborates the link between madness and lies. The lies and mental trickery of psychosis are not limited to those who are psychiatrically diagnosed. For example, Eigen refers to a recent presidential election campaign in which the illusion of good feelings (represented by one candidate) was successfully presented as preferable to a focus on painful realities (represented by the other candidate).

Destructive though madness is for individuals and societies, Eigen doubts that it can ever be eliminated. He views madness and sanity as basic polarities which structure our existence, just as does the awareness of birth and death. Moreover, he concludes: "the horrific has its own beauty, its own ecstacy, and we ought not walk round it as if it were not there, no more than we should become one with it".

Along with James Grotstein, to whom the author refers, Eigen must certainly rank amongst the foremost of contemporary analytic therapists and theorists who are making significant steps in furthering our understanding of madness. Any clinician who has ever attempted to understand the thoughts and experiences of psychotic patients will find much that is illuminating and well described in this book.

PHIL MOLLON, *Regional Psychologist in Psychotherapy (Top Grade), Dryden Road Hospital, Newcastle-upon-Tyne*

The Language of Psychosis. By BENT ROSENBAUM and HARLY SONNE. New York: New York University Press. 1986. Pp 141. $32.00.

Any attempt to unscramble 'word-salads' and neologisms is to be welcomed. The authors, a psychiatrist and a linguist respectively, include many written and spoken examples of these fascinating phenomena. However, the very nature and content of the work, coupled with possible differences in Danish and North-American styles, may have resulted in quite large sections of textural incomprehensibility for the ordinary British psychiatric reader. Certain key words are indeed carefully defined, for example 'deixis', 'anaphoric', and 'transitivism', but their too frequent use represents a characteristic which, some may feel, obscures understanding of the authors' thesis, an extract of which follows:

"The binding arises by virtue of a systematic and structurally stable inscription of signifiers in a relatively ordered and logical form of text. The individual discursive register is where inscription takes place. It appears from the above that the unconscious is the most marked property and mode of operation of the discursive register. By this we do not mean that the discursive register has been fully described. This is not the case. Some of the other important functions and properties of the discursive register arise from the way in which the inscription of the discourse, for the most part, takes place."

Psychoanalytic terms and concepts are, as stated, certainly included and are alloyed with linguistics and semiotics.

HAROLD MAXWELL, *Consultant and Honorary Senior Lecturer in Psychotherapy, West Middlesex and Charing Cross Hospitals, London*

A Critical Dictionary of Jungian Analysis. By ANDREW SAMUELS, BANI SHORTER and FRED PLAUT. London: Routledge & Kegan Paul. 1986. Pp 170. £6.95.

In the introduction to the dictionary the joint authors' clearly stated task was an attempt to define, give the origins of, and trace the changing usage of terms used in analytical psychology. This is complemented by the inclusion of other psychological terminology, but particularly terms shared by psychoanalysts. There is an efficient system of cross-reference within the dictionary, and definitive comments by other authors are included. There is also an extensive bibliography. The authors acknowledge the influence of the two relatively recent publications of psychoanalytic dictionaries, written by Laplanche & Pontalis (1980) and Rycroft (1972), in making them aware of there being no such equivalent in analytical psychology.

Since Jung's death there has been an increasing interest in Jung's ideas and a broadening in the scope and terms of reference covered by post-Jungians. This "critical" dictionary was written to trace the evolution of definitions used in analytical psychology and as the basis for further discussion. Experienced practitioners will value the clarifications which can be used as the authors intended, namely as the focal point of discussions. For most other readers I think that this work is invaluable, not merely as a concise means of absorbing information, but as the stimulation to explore many of the concepts in much greater depth. It provides the nexus for both inter- and intra-disciplinary debate.

The authors describe Jung as an intuitive thinker who relied firmly on the expression of many of his ideas as images, thus creating the ongoing phenomenon of his word usage and ideas as constantly changing. Thus the early momentum for writing the dictionary stemmed from, "a shared empathy between those struggling to understand". I am grateful for this struggle reaching the readers in such a useful form.

HUGH GEE, *Jungian Analyst, London*

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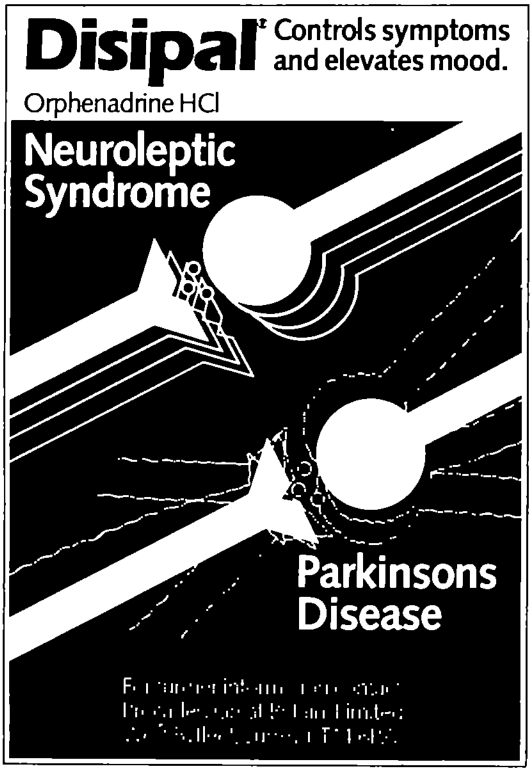

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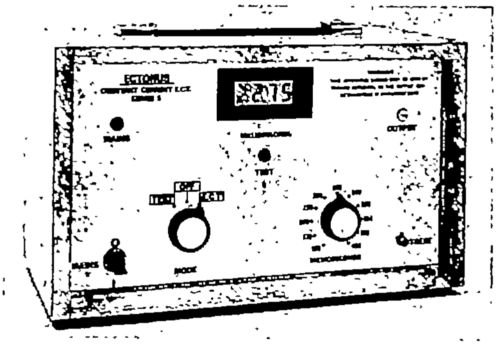

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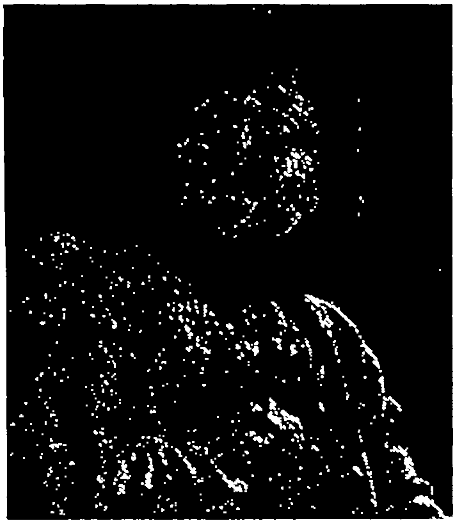

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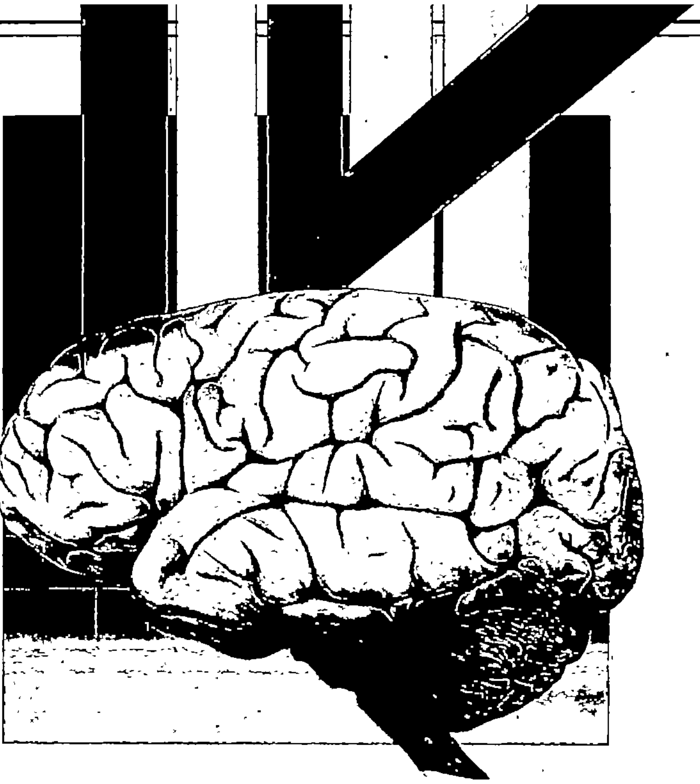

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British Journal of Psychiatry (1987), **151**, 435–439

Review Article

Mental Health in Africa:
I. The Extent of Mental Health Problems in Africa Today
An Update of Epidemiological Knowledge

G. ALLEN GERMAN

This paper is concerned with steadily developing knowledge of the epidemiology of some psychiatric disorders in sub-Saharan Africa. The diversity of 'Black Africa' is noted, and preconceptions about African psychiatry briefly discussed. Problems of sociological versus clinical philosophies are also mentioned. Early estimates of prevalence of psychiatric disorder in black Africa were universally low, being based on hospital data. More recent studies, sampling diverse populations, suggest a burden of psychiatric morbidity in black Africa which is not dissimilar to that found in more developed countries, and some studies suggest that rates in Africa may even be higher than in developed countries.

This two-part review, is concerned with mental health problems among black African people living in sub-Saharan Africa. In particular, research data obtained in anglophone countries will be emphasised, while bearing in mind that such data are not necessarily applicable to the entire continent. The intention is to update an earlier review (German, 1972).

The area under consideration is frequently regarded as homogeneous from traditional European viewpoints. In reality 'Black Africa' is exceedingly diverse: physically, culturally and socially it is very different from Europe and North America. From the point of view of the psychiatrist this is of great importance, since it permits mental disorder to be studied in a setting which maximises the difference between important background variables which have been thought to have aetiological significance in the production of disturbed human behaviour. If, despite differences in geography, climate, ethnicity, culture, and socio-economic structure, mental disorders are more or less similar to those found in other parts of the world and among other races of mankind, this argues a fundamental consistency in the expression of mental abnormality intrinsic to man himself. If, on the other hand, disturbances of behaviour show major differences with varying environments and cultures, then these disorders are more complex psychosocial reactions to varying life circumstances, highly diverse in their manifestations and courses, and unique to the individual and his personal life experience.

Before the 1960s, the decade of African independence, it was customary to regard mental ill health as being rare in Africa. Colonial psychiatrists such as Tooth (in Ghana) and Carothers (in Kenya) found evidence from their surveys of hospital populations to support the view that mental ill health was particularly uncommon among Africans, compared with Europeans. This idea sprang in part from the idyllic philosophies of Rousseau, and in part from the usually unstated belief that mental ill health was part of the price that Judaeo-Christian cultures had to pay for civilisation, for being responsible, and for opposing the Devil and all his works. In that same pre-independence era, generalisations about Africans included the views that their brains were inferior, that they functioned in a childish manner, and that they lacked a sense of responsibility. Their emotional distress was the short-lived and charming emotionality of the child, but not the bravely-borne pain of affective disorder. Responsibility was defined in terms of European priorities, while the responsible pursuit of African values was usually regarded as evidence of backwardness. Thus a series of cultural stereotypes was imposed on the African, and confirmation for these stereotypes came from epidemiological inquiries limited to patients in custodial mental hospitals – a highly selected and unusual population.

This sort of polemics tend to simplify the situation and ignore realities (such as the fact that a large number of Europeans are childish in their behaviour, lack a sense of responsibility, and spend nearly as much money on traditional European healers as they do on organised health services), and well illustrate the problems in determining the extent and nature of mental health and illness in Africa.

Great shortages of skilled manpower, particularly with adequate research training, persist in Africa. This is not entirely due to a lack of scientists who wish to work in Africa. Researchers with extensive experience of African clinical psychiatry, be they African or expatriate Europeans, are few in number, and since in most cases they have to cope with heavy service demands their experience of African clinical settings cannot be taken advantage of in research work. The visiting researcher is handicapped by lack of knowledge of language, custom, and culture, and by lack of day-to-day familiarity with the common modes of presentation of psychiatric disorder. This is a serious handicap which is not always apparent to the trained clinician undertaking research in his own culture.

The data base with regard to demographic structures and patterns is sadly inadequate. Also lacking are adequate data-processing facilities and laboratory facilities for biological investigation. These problems are further compounded by communication and transportation problems: by the sheer size of the areas to be studied, by inadequate libraries and literature resources, and by linguistic diversity which is a problem for even the well-trained African professional.

In addition to these practical problems are those which result from philosophical issues. European researchers may produce immaculate work, but be vulnerable to the charge of ethnocentricity blinding them to more subtle aspects of African psychopathology and culture. This has become less of a problem, since the majority of psychiatrists now working in anglophone Africa are native Africans with extensive experience in their own societies. A substantial amount of research now emanating from black Africa has been undertaken by black African clinicians; this is a development of considerable significance.

Ethnocentricity is not the only factor, however, which can distort conclusions. It is perhaps significant that non-psychiatric researchers are those most likely to conclude that African mental health and mental disorder is culture-bound and not capable of being comprehended within Western psychiatric categories. To be fair, it is also of significance that most of those who insist that mental disorder in Africa is comparable with that found in Europe and North America, and is susceptible to similar classification, are clinical psychiatrists (albeit now mostly African ones). Among psychiatrists who repudiate nosological and classificatory systems of a Kraepelinian or DSM-III type are to be found large numbers whose interests are primarily in psychoanalysis, social psychiatry, or psychiatric anthropology. The viewpoint clearly influences the conclusions reached.

The extent of mental disorder

Early studies of the extent of mental disorder in Africa relied almost exclusively on hospital statistics. Most colonial African countries had one central mental hospital to which patients were brought, usually because they had become violent and disruptive within their communities. This may be why hospital statistics repeatedly indicate an excess of male admissions over female (usually in a ratio of two to one). These hospital studies led to the conclusion that in most parts of British Africa the point prevalence of psychiatric disorder varied between 0.1 per 1000 at risk (Carothers, 1953) to 0.96 per 1000 (Tooth, 1950). Carothers also obtained information from district commissioners and chiefs, and suggested a figure of 0.37 per 1000 at risk as representing the prevalence of psychiatric morbidity in Kenya.

All these statistics, if taken at face value, would suggest an extremely low prevalence for mental disorder in Africa. They also indicate a relatively low admission rate to psychiatric hospitals, compared with more developed parts of the world. The great range in the figures (0.1–0.96) indicates their probable unreliability.

In view of the tendency to select violent patients for hospital admission, the authors of these reports were not in a position to describe adequately the occurrence and nature of psychoneurotic disorders, or of the 'quieter' psychoses such as psychotic states of depression. The absence of such forms of pathology among their case material led to the supposition that these disorders were exceedingly rare in Africa.

In a previous review (German, 1972) some note was made of the difficulties which still plague studies of prevalence in Africa. Problems relating to the definition of psychiatric disorders – particularly conditions such as the neuroses and personality disorders, where it is important to establish cut-off points to distinguish what will be considered a case and what will be excluded – have only recently been tackled in the African context. Consequently, earlier studies of prevalence, based on field research, have either sought to avoid diagnostic definitions or have suffered from uncertainties as to what was being counted.

Epidemiological questions

In the face of these problems several strategies have been employed, each seeking to answer different sorts of question about prevalence:

(a) What is the prevalence of psychiatric morbidity among accessible and easily studied populations?

(b) What proportion of general hospital clinic attenders are primarily psychiatrically disturbed?

(c) How many Africans from a given community consult someone because of psychiatric disorder in the course of a year?

(d) If one samples a total population, what is the point prevalence of significant psychiatric morbidity in that population in the African setting?

Prevalence in accessible populations

One of the easiest groups to study with epidemiological techniques is one of university students. African universities tend to be self-contained, and provide comprehensive internal health services. All students are registered with and examined by the health service on admission, and continue to rely on its services throughout their student career.

German & Arya (1969) published results of a study of psychiatric morbidity among the students of Makerere University College. They used the same techniques as Kidd & Caldbeck-Meenan (1966) used for a similar study at the Universities of Edinburgh and Belfast. The overall annual prevalence for significant psychiatric morbidity among the student body at Makerere was 10.8%, compared with 11.6% in Edinburgh and 10.2% in Belfast. These figures were broken down into broad categories of psychiatric diagnosis; 6.7% of the Makerere patients were psychotic, 85.9% were psychoneurotic, and 7.4% were thought to show significant disorders of personality. Comparable figures for Edinburgh were 4.6%, 90.2%, and 5.2% respectively; for Belfast they were 2.0%, 91.0%, and 7.0%.

Other studies of African student populations have since been carried out. Jegede (1980) surveyed 145 female and 725 male first-year students at the University of Ibadan in Nigeria. The Health Opinion Survey, the Eysenck Personality Questionnaire and the 65-item General Health Questionnaire were used, as well as self-ratings of mental health. Overall, 6.3% of the group rated their mental health currently and during the preceding 4 weeks as being poor or only fair. The methodology of this study was different from that of German & Arya, and the prevalence rates obtained cannot be directly compared. However, Jegede pointed out that his rates were remarkably close to the prevalence rates of 7–16% reported by Segal (1966) in a review of five such university studies carried out since 1960.

While these college studies cannot be directly compared, they suggest a weight of psychiatric morbidity in African students which is comparable with that in the developed world; this seems to hold true even for the distribution of diagnoses across the broad categories of psychoses, psychoneuroses and personality disorders. African students might be regarded as closely comparable to students anywhere else, but it should also be remembered that these students, although an élite, have been brought up within their distinctive cultures, with personality traits and attitudes, belief structures and behaviours, and concepts of illness which closely approximate those to be found within the greater culture around them. In general, there is little evidence to suggest that being intelligent, being African, or having gone to university particularly protects young Africans from mental ill health.

Research interest has also been directed at the psychiatric status of pregnant and puerperal women in Africa. In a detailed and extensive study, Cox (1979) compared 263 pregnant Ugandan women with 89 non-pregnant women, using a semi-structured psychiatric questionnaire. He reported that 16.7% of the pregnant women had definite psychiatric disorders, and that a further 14.1% showed possible psychiatric morbidity; the comparable figures for the non-pregnant women were 7.9% and 4.5%. Of the pregnant women, 8% suffered from a depressive neurosis, 2% from a depressive psychosis, 3.4% from an anxiety neurosis, and 1.9% from a phobic neurosis. Cox noted that the "belief that Africans have a trouble-free natural pregnancy is not substantiated". His results confirm a report by Assael *et al* (1972) that 24% of pregnant women in the same population showed evidence of conspicuous psychiatric morbidity, the bulk of which was of a depressive sort. It is of interest that both these Ugandan studies report prevalence rates in excess of rates reported from Europe: 17.1% in Sweden (Nilsson & Almgren, 1970) and 16% in Britain (Kumar & Robson, 1978). Cox noted an association between psychiatric morbidity during pregnancy and marital separation.

Another study of puerperal psychiatric disturbances has been reported from Tanzania (Harris, 1981). Harris found that 38% of puerperal Tanzanian women showed clear evidence of 'maternity blues' and commented that the prevalence of such disturbances in these East African mothers is similar to that in other cultures. He concluded that 'maternity blues' are supra-cultural in their manifestations.

Psychiatric disorder among general hospital out-patients

A considerable number of African studies have addressed this question. It is important that hospital administrators and health planners should be aware of the frequency with which people with primary psychiatric disorder are being dealt with in general clinics by health personnel who may have little familiarity with psychiatric morbidity.

One of the older, but most detailed, studies of this sort was carried out in Ethiopia by Giel & Van Luijk (1969). They reported that 18.5% of general out-patients attending a teaching hospital in Addis Ababa were primarily suffering from psychiatric conditions, compared with only 9.5% diagnosed as suffering from infectious diseases. In a more rural population served by a provincial general hospital only 7% of general out-patients were found to be psychiatrically ill, while 28.7% demonstrated evidence of infectious disease. Finally, at a rural health centre serving a totally unselected and largely self-referred population (akin to a general practice in the western world) 19.5% of out-patients were deemed to have psychiatric disorders, and 17.1% to have infectious diseases. This study is of particular interest in that it provides statistical comparisons with infectious diseases, which occupy the forefront of medical attention in Africa and other developing regions.

A Nigerian general practitioner with a special interest in psychiatry reported that 1460 new patients attended his general clinic over a period of three months (Mbanefo, 1971). The 1732 diagnoses made included 265 cases of malaria, 264 of psychiatric disorder, 181 of respiratory infections, 262 of other infectious diseases, and 55 of gynaecological problems. Overall, 15% of his patients, drawn from mixed urban and rural populations, were diagnosed as having a psychiatric disorder. A more recent study of this sort was reported from rural Kenya by Ndetei & Muhangi (1979). Patients seen at a walk-in/walk-out clinic over a period of 30 days were surveyed. Of 140 patients, 20% were found to be psychiatrically disordered and 59.3% physically ill; the diagnosis of the remaining 20.7% was uncertain. The majority of the psychiatric cases showed anxiety and depressive symptoms.

Thus it would appear that in relatively unselected attenders at general out-patient clinics in Africa approximately 20% are primarily psychiatrically disordered. Most of these reports stress the frequency with which psychological distress is presented as somatic disturbance. Such modes of presentation are particularly confusing to the general health worker without experience of psychiatry.

Consultation rates in a given community

Studies designed to answer this sort of question are difficult to undertake in Africa: they require a field approach to either a total population or a representative sample, or a thorough search of all possible consultation agencies in an attempt to locate attendees.

Two studies have provided information in this area. Giel & Van Luijk (1969), in addition to the hospital and health centre investigations discussed above, surveyed a defined catchment area in an attempt to discover how many people in that area consulted someone for psychiatric illness in the course of one year. The authors used the rural area in which the rural health centre was established, and in addition to consultations with official health agencies sought data on persons consulting local healers and witch-doctors. They believed that few consultations were missed using this technique. They found an annual consulting rate for psychiatric disorder of 148 per 1000 of the population at risk, and compared this with the rate of 139.4 per 1000 for England reported by Shepherd *et al* (1966).

In contrast to this, Binitie (1981) found a rate of 35.7 per 1000 at risk in a rural area of Bendel State in Nigeria. The population studied comprised 2325 workers on an oil palm plantation, relatively isolated from surrounding populations and provided with an internal health service. Binitie does not appear to have taken account of other consultation agencies, although he points out that the families and dependents of the workers were in the habit of consulting away from the unit health centre. Nor is mention made of whether or not these subjects consulted with traditional and native healers, and in view of the well-established organisation of such healers in Nigeria it seems likely that substantial numbers of the catchment population might have been missed as a result.

Point prevalence in a total population

Two epidemiological studies are worthy of mention in this context. They were conducted over a decade apart and utilised different methodologies. The first was carried out in Western Nigeria, in urban and rural populations around the city of Abeokuta (Leighton *et al*, 1963). The methodology was designed to produce data comparable to those obtained in similar researches in Canada. African subjects, male and female, living in the urban area and in rural villages were assessed in terms of the presence of specified psychiatric symptoms rather than in terms of diagnostic groups or syndromes. It was found that 21% of the respondents in the villages and 31% of those in the city showed significant symptoms of psychiatric morbidity at the time of the survey. Probable psychiatric symptoms were present in a further 19% of villagers and 14% of city dwellers. The authors provided a wealth of data, but perhaps their most significant comment was that in comparison with Canadian samples "the similarity of the two samples is much more impressive than the differences. In view of the contrast between the cultures and life situations, this is truly remarkable. The similarity applies both to pattern quality (anxiety, depression, etc.) and to prevalence in most of the categories tabulated".

The second study approximates more closely to the ideal epidemiological study than any other carried out in black Africa to date (Orley & Wing, 1979). These workers sought to establish data on psychiatric morbidity for a total population sample. Orley conducted the fieldwork in rural Uganda, using techniques developed by Wing for the assessment of psychiatric symptoms and their categorisation into psychiatric syndrome groups. Data obtained from south-east London in a similar manner were available for comparison. Total populations in two small Ugandan villages were interviewed using the Present State Examination (PSE) and standard methods of case identification. The data obtained were then analysed using an index of definition to establish cut-off points and the CATEGO program for syndrome identification. Orley is an experienced psychiatrist, and is also qualified in anthropology. At the time of the survey he had spent several years working in rural Uganda, conducting anthropological researches through participant observation. He had learnt the local language, Luganda, to a high level of fluency. He also used the PSE as translated by himself into Luganda, following extensive work with local Luganda speakers and scholars to establish the validity of the translation. The methodology is meticulously described, and potentially repeatable given a similar investment of labour. A number of aspects of this study make it a highly significant piece of work, the results of which require careful attention. They include the careful methodology, and the facts that a total population was sampled, that standardised and reasonably objective methods were used to obtain and analyse data, and that the population in question was rural and remote from Western influence, subsisting in a traditional manner.

Orley & Wing reported the prevalence of psychiatric morbidity at varying levels of certainty or definition. Eight levels of confidence are used, level 5 being the threshold and levels 6–8 indicating increasing degrees of confidence that the symptoms present could be classified into one of the categories of functional psychoses or neuroses used in the International Classification of Diseases (World Health Organization, 1978). In brief, 4.9% of the total population showed definite psychiatric syndromes, with an additional 20.4% of the population showing evidence of psychiatric distress at threshold level. There was no difference between the proportions of each sex showing definite psychiatric syndromes, and little difference at the threshold level. Combining all cases at threshold level and above, 27% of the women and 24% of the men showed evidence of psychiatric disorder. In comparison, the authors reported that in a suburb of South-East London 11% of women showed evidence of psychiatric disorder at above-threshold level – i.e. the point prevalence of psychiatric morbidity in rural Urganda is at least twice that found in South-East London.

With regard to syndrome categories, Orley & Wing reported that 2% of the men and 2.2% of the women in their population showed evidence of hypomania (compared with 0.4% among women in South-East London); evidence of depressive disorders was found in 14.3% of the Ugandan males, 22.6% of the Ugandan females, and 9.3% of the South-East London females. Anxiety states were detected in 3.1% of the Ugandan males, 4.3% of the Ugandan females and 1.3% of the South-East London females. Particularly striking is the high frequency of depressive disorder; this will be discussed in Part II of this paper.

As this study used the most sophisticated and exacting methodology employed to date in tackling the problem of the prevalence of psychiatric morbidity in Africa, its results must be refuted or replicated as a matter of some considerable urgency. Until that is done it must be taken as the best evidence available relating to the extent of mental illness in African populations.

This brief review of studies of prevalence in Africa has focused on the more significant studies. There is a wealth of data available based on clinic and hospital populations, which has not been commented on. However, the general trend is clear: the extent of mental illness in black Africa is at least as substantial as in other parts of the world where data have been obtained, and there is some disturbing evidence which suggests that morbidity rates may be even greater. Furthermore, the frequency of psychotic and psychoneurotic illnesses also appears to be approximately the same as elsewhere. Finally, despite previous assertions to the contrary, among these common psychoneurotic presentations the familiar symptoms of depression and anxiety seem to be as ubiquitous as everywhere else.

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Mental Health in Africa:
II. The Nature of Mental Disorder in Africa Today
Some Clinical Observations

G. ALLEN GERMAN

Aspects of clinical psychiatric syndromes described in Africa which are discussed include the issue of schizophrenic disorders having a better prognosis in developing countries; controversy over this is by no means at an end. There is an increasing realisation as to the frequency of affective disorders in Africa; while somatisation is common, cherished beliefs, such as the absence of guilt, have not been confirmed by more recent research. Nor is suicide as infrequent as has been suggested. The relationship of background physiological abnormalities of cerebral functioning may be relevant to some of the clinical issues that are currently under discussion in African psychiatry.

An important question at the operational level is whether or not defined criteria for a given mental disorder can be shown to apply in Africa with reliability comparable to that obtained in more developed parts of the world. It is only in the past 20 years that attempts have been made to produce detailed operational definitions of psychiatric symptoms and to train psychiatrists and other research workers in the reliable use of such definitions. The Present State Examination (PSE) (Wing & Sturt, 1978) is an example of development in this area. It was one of the first such instruments to be developed and applied widely in different cultures; its use by Orley in Uganda was described in Part I of this review. Other standardised systems of psychiatric symptom elicitation have been developed, together with standardised definitions of syndrome complexes built on these symptoms. The availability of such tools enables psychiatrists in different settings, provided they have had comparable training, to study the distribution of symptoms and syndromes in various cultures and to draw conclusions as to the nature of such phenomena in various parts of the world.

Schizophrenia

Recognising the potential of these developments, the World Health Organization has pioneered research approaches to various psychiatric disorders, with the intention of obtaining information on the distribution of psychiatric symptoms and syndromes in those parts of the world where culture and ethnicity differ widely from the Western society in which current concepts of psychiatric disorder were developed. Foremost among these studies is the International Pilot Study of Schizophrenia (IPSS), a multi-centre examination of the symptom profiles of psychotic patients in eight different locations throughout the world. One of these is at Ibadan (in Oyo State, Nigeria); the others are in the UK, the USA, the USSR, India, Denmark, Czechoslovakia, and Colombia. The IPSS, in its initial phase, was not concerned with establishing the prevalence of schizophrenia but with determining whether or not (a) psychiatrists in different parts of the world could use tools such as the PSE reliably, and (b) schizophrenia, as defined by standardised criteria, could be identified in disparate cultures. The answer to both these questions has been found, after a decade of intensive work, to be in the affirmative (Luria & McHugh, 1974; WHO, 1973; WHO, 1975; Wing & Sturt, 1978). Schizophrenia occurs in black Africans with the same presenting symptoms and the same patterns of symptoms as were originally described in Europe during the first decade of this century, and thus many of the controversies which raged in the African psychiatric literature some fifteen years ago have now been settled.

A previous review (German, 1972) discussed problems relating to the distinction between 'process schizophrenia' and schizophrenia-like syndromes occurring in such settings as organic disease, epilepsy, or psychogenic stress. Many psychiatrists in Africa made no distinction between these two groups; others insisted that a diagnosis of schizophrenia should only be made in the absence of organic disease, or where affective, confusional, and comprehensible psychological symptoms were not dominant in the clinical picture. As a result of these differences, there was considerable controvery as to the profile of schizophrenic symptoms in Africa and dispute as to prognosis. Those with the broader concept of the disorder reported greater heterogeneity of outcome, while those with more restricted notions argued forcefully that schizophrenia in Africans was identical to that seen in Europeans (Boroffka, 1964; Fortes & Mayer, 1969). Such differing concepts of schizophrenia are bound to lead to controversy. However, with the selection of accepted operational definitions for the symptoms of the disorder, and diagnostic criteria in terms of the profile of symptoms, it becomes possible to seek answers to fundamental questions about schizophrenia.

A different view of schizophrenia could lead to a different and better prognosis for patients in developing Africa than in the developed world: if workers in Africa were using a concept of schizophrenia which included many short-lived transient reactions to organic, affective, and psychogenic precipitants, then schizophrenia in Africa would have a better prognosis than elsewhere. Undoubtedly, some of the impression of good outcome is attributable to such factors. However, with the development of the IPSS methodology it has now become possible to follow up defined cohorts of schizophrenia in different parts of the world. The IPSS goes to some lengths to exclude organic, affective, and psychogenic psychotic states and, given that the described method of assessment has been properly followed in each centre, there is evidence to suggest that schizophrenia, even thus defined, has a better prognosis in certain developing societies than in more developed ones. The matter is still not entirely settled, since there remain some inadequacies yet to be dealt with in this major study (Day, 1980).

Murphy (1976) studied local and traditional definitions of insanity in Eskimo and Yoruba (Nigerian) cultures. This study is of particular interest because the author, an anthropologist, confesses to conclusions contrary to her expectations. She produced evidence that insanity, comparable with European concepts of psychosis, is recognised and named on the basis of the same patterns of disorder in culturally disparate groups. In addition, she advances evidence for the universality of the psychoneurotic experience, and also for similar prevalence rates of all these categories of disorder throughout the world. She stated, "Rather than being simply violations of the social norms of particular groups, as labelling theory suggests, symptoms of mental illness are manifestations of a type of affliction shared by virtually all mankind." Her explorations of this matter are particularly concerned with schizophrenia, and her eloquent descriptions of Eskimo and Yoruba concepts of this disorder are strikingly similar to those of Kraepelin.

Depression

Another area of considerable discussion concerns the nature of depression and other affective psychoses in the African setting. During the colonial period it was generally stated that depressive illness was rare in Africans (Prince, 1968). Prince pointed out that up to 1957 13 out of 14 authors reported that true psychotic depressions were rare, and that if they did occur they were short-lived and of little severity (the exception was Field (1960)); in the period 1957–1968, 21 papers were published which expressed the contrary view. He suggested that while there may have been a real increase in the prevalence of depressive illnesses, it seemed more likely that when studies were based mainly on hospital populations, cases of depression were rarely detected, since they were rarely admitted to institutions. He also suggested that in the pre-independence era depression was implicitly regarded as the 'property' of people with certain attitudes to life: responsible Judaeo-Christian attitudes, saturated with the work ethos. Another possibility is that more modern views of depression, which include the concept of depressive illness without marked emotional shift (masked depression) have enabled recent researchers to detect a far greater number of depressive syndromes in African populations. Finally, depression achieves its greatest frequency in persons over the age of forty; since in most African populations statistics indicated that until recently few Africans lived beyond this age, it may be that with increasing life expectancy more cases of depression are becoming apparent. Clearly, age-specific rates must be calculated for any valid study of this problem.

Current workers are unanimous in agreeing that depressive illness, psychotic or otherwise, is common in Africa. Dispute persists, however, about the nature of symptoms. Asuni (1961) concluded that feelings of guilt were rare and that paranoid projection was a more common feature of depressive illness in Africa. Binitie (1975), in a sophisticated statistical study, concluded that, while depression seems remarkably similar in Nigeria and London, somatic symptoms are commoner in Africa, and guilt with suicidal ideas commoner in England. Most workers would have agreed with Binitie (including the present author, who has presented evidence (German, 1972) to support the view that ideas of guilt were relatively rare in African subjects), were it not for the more recent work of Orley & Wing (1979), who found no support for these earlier opinions.

In view of the methodological precision of the Orley & Wing study, and particularly in view of Orley's substantial familiarity with the language of his study population, their findings must be given due attention. They report a frequency of pathological guilt among the Ugandan subjects which is approximately four times greater than that found in London. This is entirely opposed to previous viewpoints, and may either represent a constellation of symptoms peculiar to a limited area, or demonstrate that the history of exploded myths in African medicine continues to be written. In view of the significance of this study, it can no longer be stated with certainty that feelings of guilt are only found in highly individuated people brought up in nuclear families which tend to emphasise personal responsibility.

The issue here may not be so much whether individual symptoms are present or absent, but the extent to which given population groups emphasise these symptoms in their concept of priorities – or in their concept of what is appropriate in a state of illness. It would appear that most African patients present somatic symptoms in depression frequently and with considerable force, in keeping with their view of the nature of states of disease and with their relative psychological unsophistication. It is not inconceivable that in all depressives there are equal feelings of somatic unease and of guilt; the extent to which either of these is presented may be merely a reflection of culturally determined assessments of priority or relevance.

Suicide

The prevalence of suicide in Africa is another area of dispute. Some studies have found suicide rates to be low; Asuni (1961) and Laubscher (1937) quoted annual rates of approximately one successful suicide per 100 000 persons in Nigeria and South Africa respectively. However, Fallers & Fallers (1960) found 8.5 suicides per 100 000 in the Busoga District of Uganda, while Rwegellera (1978), compiling rates from the city of Lusaka in Zambia, reported an overall annual suicide rate for Africans of 12.8 per 100 000. The comparable rate in Lusaka for Europeans was 20.9 per 100 000.

Rates therefore range from those which are exceedingly low by European standards to those which are comparable with figures reported from European countries such as Britain. The reasons for these differences are not clear. Certainly suicide is not an easy behaviour to diagnose accurately, particularly in countries where police and judicial services may be meagre. In addition, as Orley (1970) has pointed out, suicide in most African cultures is regarded as a terrible act, which attracts cultural sanctions (including secrecy) which may well distort reports of its occurrence. On the other hand, it could be argued that more recent studies such as that by Rwegellera have concerned themselves with urban populations in large cities which are undergoing rapid and traumatic social change, a situation ripe for the enhancement of suicide. The matter is not yet certain, but it appears that in traditional and stable African communities suicide rates are somewhat lower than in those other African communities, both rural and urban, which have been exposed to social changes during the past 20 years.

Transient psychoses

In the earlier discussion of schizophrenia, it was noted that short-lived transient psychotic reactions to various precipitants are particularly common in Africa. Most authors who have written about African psychiatry have commented on this, with occasional exceptions such as Rwegellera (1977). These states have been previously discussed in some detail by the present author (German, 1972; Assael & German, 1970). Various names are applied to the states, the commonest used in anglophone Africa probably being 'acute psychotic reaction'. They are variously described, and appear to form an amorphous category characterised by features such as confusion, depression, mania, homicidal excitement, and hysterical behaviour (Carothers, 1953; Shelley & Watson, 1936; Tewfik, 1958; Field, 1960). In some cases paranoid delusions, sometimes complicated by clouding of consciousness, lead to these states being described as schizophreniform. Lambo (1955, 1960) suggested that they resemble hysterical, epileptic and other organic states, but he also emphasised the importance of cultural factors. Haworth (1968) regards the conditions as being essentially reactive, and comes close to the Scandinavian position on reactive psychoses, which regards the occurrence of schizophreniform illnesses as being a common consequence of psychological stressors.

Rwegellera (1977), in his review of first admissions to a mental hospital in Lusaka, argued that the vast majority of these conditions were atypical presentations of affective disorder, mainly depression. While his observations must be given due weight in view of his careful approach to diagnosis based on form rather than on the content of symptoms, his observations still beg the question as to the frequency of so many atypical presentations. It may be that depression is more likely to present when it is atypical and is characterised by more bizarre and disruptive symptoms, such as hysterical or schizophreniform features.

Other factors may contribute. Assael & German (1970) argued for the conditioning effect of background cerebral dysfunction, this being a likely factor in societies where cerebral development during infancy is frequently prejudiced by protein or calorie malnutrition, or cerebral malaria. Under such circumstances there may well be tenuous cerebral control over florid affective states, and there is also a greater likelihood of hysterical phenomena manifesting themselves in states of psychic distress where cerebral integration is impoverished. Apart from that, cultural attitudes in Africa do not discourage dramatic and vigorous emotional reactions to states of psychic pain or distress, and this is undoubtedly an important factor. Confusional contaminants may reflect the frequency with which these conditions emerge against a background of low-grade physiological disturbance such as that provoked by intercurrent infections, parasitism, vitamin deficiencies, and other states commonly found in poorer societies. Indeed, Jilek & Jilek-Aall (1970), in discussing the frequency of these states in Africa, pointed to the fact that similar acute transient disturbances were well-known in Europe at the end of the nineteenth century, and were regarded by psychiatrists of that era, particularly in Germany, as being related to poor standards of medical care and general health.

Organic psychoses

As might be expected, acute brain syndromes are common in Africa, their frequency reflecting the frequency with which human beings are exposed to severe and acute disorders of physiology capable of disrupting normal brain functioning. The commoner causes of these disorders in Africa have been listed by Jilek & Jilek-Aall (1970), and discussed by German (1972). Infections, parasitic and deficiency diseases, and a wide-ranging abuse of drugs are ubiquitous to a degree long forgotten in the developed world.

The clinical features of the resulting acute brain syndromes are as described elsewhere, although hallucinations and delusions (as in any other psychosis) are coloured by local preoccupations. There does however appear to be an unusually high frequency of such acute organic psychoses in apparently healthy young adults where the provocative toxic or infectious state seems less than would be necessary to lead to such a consequence. The situation may be analogous to the ease with which acute brain syndromes can be precipitated in elderly people living in developed countries. German (1979) argued that this would be consistent with pre-existing diminution of cerebral reserve due to previous deprivation. It may be useful to consider the profile of psychotic states and psychotic reactions that occur in elderly people in developed societies as being similar to the profile of both acute psychotic reactions and acute brain syndromes occurring in younger people in developing countries. In the psychogeriatric situation stress, background reduction of efficient cerebral functions, inefficient renal and hepatic mechanisms, and social expectations are factors which influence the amorphous patterns of psychotic states. These particular African problems therefore might best be understood within some such paradigm.

Neuroses

Comment has already been made on the frequency of psychoneurotic states. Evidence suggests that they are particularly common in Africa, including rural Africa (Orley & Wing, 1979). Murphy (1976), while noting that among Western Nigerians there are no specific word labels for the various neurotic states or for the overall concept of neurosis, also pointed out that "words for emotional responses that we might classify as manifestations of anxiety or depression constitute a very large vocabulary". She also noted that most of these emotional phenomena are thought of as illnesses for which the traditional doctor has effective cures. She went on to observe that among the Yoruba of Nigeria these conditions are much more frequent than psychotic states, the ratio being approximately 12 to 1.

As seems to be usual in African society, it was those neurotic conditions with the more dramatic presentations which were first noted and put forward as peculiar to the continent. Thus hysteria (Dembovitz, 1945) and vividly described anxiety states (Vyncke, 1957) have been regarded as characteristic. The more recent studies of Orley & Wing (1979) have revealed the frequent presence of quieter neurotic states characterised by depression and anxiety, and have done so by looking at total populations rather than mental hospital populations.

The Orley & Wing study also drew attention to the frequent occurrence of obsessions in African men and women. This is a matter of great interest, since most previous authors repeatedly emphasised the great rarity of both obsessional behaviour patterns and obsessional illness in black Africans. Orley & Wing did not comment on the possible reasons for this finding, but it is consistent with the possibility that when epidemiological studies of neurotic disorder in Africa are conducted with great care and precision there will emerge evidence to suggest that emotional and psychological response patterns have a fundamental consistency irrespective of human race and culture.

There seems to be little evidence available to suggest that psychoneurotic conditions are fundamentally different in Africa, either in prevalence or form. However, and this appears to be true of any form of psychiatric or emotional disturbance, symptom content is very much a reflection of local preoccupations. These preoccupations may be unique to a family, to a village, or to a wider culture. In general, Kraepelinian systems of classification would regard content aspects of clinical pictures as having little or no relevance to diagnosis. On the other hand, psychodynamic formulations of psychiatric distress pay considerable attention to content, as do more sociological systems. It is probable that these different viewpoints are largely responsible for the confusion which sometimes seems to exist about mental illness in Africa.

Final comments

Little has been said in this review about vital and wide-ranging issues in black Africa which have an important bearing on mental illness and mental health. Factors such as drug abuse, the break-up of traditional family structures, population mobility due to political upheaval and socioeconomic shifts, and recurrent episodes of famine and other deprivations are a feature of the area. Africa has one of the largest mobile refugee populations in the world. Nor is Africa immune to those social changes which have cast a heavy shadow over the rest of the world in recent decades. Several studies testify to the alarming prevalence of alcoholism in secondary schools. Oshodin (1980, 1981) reported that 18–40% of high school students in Nigeria showed a pattern of drinking behaviour consistent with alcohol abuse. Smith (1982) has drawn attention to the rapid increase of alcoholism and alcohol-related social, health and economic problems in Third World countries, and has pointed out that there is substantial evidence to suggest that many alcohol problems can be prevented by administrative and political measures. He believes that if Third World governments were to wake up to the threat of alcohol problems, they might be able to avoid them without a huge expenditure of time, money and effort.

However, there are major problems to be addressed if mental disorders and their associated evils are to be adequately prevented, managed, or controlled in Africa. One is the difficulty of convincing governments and leaders as to the importance of these disorders and the major impact that they have on community stability, security, and development. In addition, models developed in Europe and North America for the management and control of mental disorder are transported without thought to the African setting, and this has led to the diversion of an undue amount of health resources into the establishment of medical schools and technologically advanced hospitals and the training of medical specialists. The provision of medical schools and hospitals may appeal to politicians as being tangible evidence, to an unsophisticated population, that they should be re-elected, but it is difficult to escape the conclusion that African mental health problems require something vastly different.

As a result, international policies with regard to mental health have increasingly urged the development of health services based on village health workers and other primary health care workers (World Health Organization, 1978). The logic of such a shift is unassailable. Africa lacks the wide-ranging general practice services which are a feature of European medicine, and which probably cater for 90–95% of the needs of mentally and emotionally distressed persons in Europe. Without such a buffer, specialists in mental health in Africa are faced with an impossible task, and have difficulties functioning as they have been trained to function in the new hospitals which have developed (German *et al*, 1980). The World Health Organization programme also envisages that the expansion of primary health services will be an integral part of a development directed at redressing social evils, or at supporting, through education and community resources, their many victims. Social breakdown, poverty, population mobility, famine, and drug abuse are only a few of these evils which are not only of great significance in the precipitation of mental ill health, but of even greater significance in the erosion of community and national health to a point where it becomes ludicrous to talk of health at any level. It is therefore clear that a body charged with concern about the world's health must address itself to related social problems which must be solved, or mitigated, before sophisticated health techniques can be deployed in any credible way.

Psychiatric manpower is probably relatively scarcer in developing Africa than it was twenty years ago. At the same time, social and political turmoil has increased and the number of persons seeking assistance for mental ill health has continued to rise rapidly. If the WHO goal of "health for all by the year 2000" is to be realised to any extent, then it is imperative that in most countries of the world there be a major campaign launched at a political level to encourage a much wider view of the concept of health, and those circumstances, attitudes, and structures necessary for its promotion and maintenance. Such a campaign will have many requirements; one is that decision-makers and thinkers in the Third World should be familiar with current evidence which suggests that formal mental illness is as significant a problem in the Third World as anywhere else, and indeed may be an even greater problem in terms of impact and sheer numbers.

In conclusion, the extent and nature of mental ill health throughout black Africa does not appear to be such as to suggest that psychiatric disorders are culture-bound and the prerogative of so-called advanced societies. Problems relating to mental health appear to be intrinsic to the nature of man and to reflect the impossibility of separating biological, psychological, and cultural influences. Although the 'medical model' - or perhaps more precisely the 'Western nosological model' - seems capable of adequately accounting for the nature of the majority of these African mental ill health problems, purely medical approaches are not in themselves sufficient to contain or reduce these problems. Within developing countries there is an urgent need to rethink methods of health care to take account of the realities of local demographics, socioeconomic conditions, and cultural values.

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Anthropology and Psychiatry

The Role of Culture in Cross-Cultural Research on Illness*

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To illustrate the contribution anthropology can make to cross-cultural and international research in psychiatry, four questions have been put to the cross-cultural research literature and discussed from an anthropological point of view: 'To what extent do psychiatric disorders differ in different societies?' 'Does the tacit model of pathogenicity/pathoplasticity exaggerate the biological aspects of cross-cultural findings and blur their cultural dimensions?' 'What is the place of translation in cross-cultural studies?' and 'Does the standard format for conducting cross-cultural studies in psychiatry create a category fallacy?' Anthropology contributes to each of these concerns an insistence that the problem of cross-cultural validity be given the same attention as the question of reliability, that the concept of culture be operationalised as a research variable, and that cultural analysis be applied to psychiatry's own taxonomies and methods rather than just to indigenous illness beliefs of native populations.

Anthropology and medicine enjoy a long relationship. Among the late nineteenth and early twentieth century founders of anthropology were physicians who possessed social psychological and psychosomatic orientations (e.g., W.H.R. Rivers in England, Paul Broca in France, Rudolph Virchow in Germany). Key figures in the development of psychiatry maintained a cross-cultural interest and read the relevant anthropological literature (e.g., Emil Kraepelin, Sigmund Freud, Adolph Meyer, Aubrey Lewis, to mention only a few). Similarly, leading figures in twentieth century anthropology (most notably Alfred Kroeber and Edward Sapir in the US, Bronislaw Malinowski and Meyer Fortes in England, and Claude Lévi-Strauss in France) kept up with relevant developments in psychiatry and particularly psychoanalysis. In the 1940s and 1950s, the 'Culture and Personality' school of North American cultural anthropology brought together anthropologists and psychoanalysts in research seminars and collaborative projects. Even at present, although the two fields are moving in very different directions, many anthropologists maintain strong interest in psychiatric questions and, at least in North America, a small number are members of psychiatric research units. On the other side, psychiatrists conducting international, cross-cultural and cross-ethnic studies frequently cite the writings of anthropologists, and there is even a small cadre of psychiatrists who, like myself, are trained in anthropology.

Yet, for all this cross-disciplinary interest (at least in North America), there is little evidence that anthropology exerts any significant influence in psychiatry, or vice versa. Even in cross-cultural and social psychiatry – the two subdisciplines that come the closest in problem framework to the concerns of the social sciences – anthropology is marginal: more of informal, heuristic interest than of central, substantive importance. Rarely are psychiatric trainees systematically taught about relevant anthropological concepts and findings, even where those trainees spend significant time practising in cross-cultural or cross-ethnic settings. Just as rare is the psychiatrist engaging in cross-cultural research who raises anthropological questions and findings pertinent to his enquiry. This is a harsh judgement, but I believe the facts support it: psychiatry proceeds, even or especially in centres that are non-Western or ethnic, as if anthropology did not exist, or was irrelevant to illness and care (Kleinman, 1985).

Conversely, anthropology graduate students most often go to the field to study the relationship of culture to illness or healing with hardly any systematic preparation in relevant diagnostic, epidemiological or treatment issues. Indeed, there are not a few anthropologists studying illness who seem remarkably unaware of key changes in psychiatry's data base and conceptual orientations. It is embarrassing to read anthropological accounts that provide sensitive discriminations of the heterogeneity of folk healing systems but caricature biomedicine and psychiatry.

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This situation is, to my mind, deplorable. There are, however, heartening indications that it may improve. The argument I will advance is that much is to be gained by a robust, two-way relationship between psychiatry and anthropology; ambitious and fundamental exchange across the two disciplines could contribute in quite practical ways to the strengthening of both. In this paper I will illustrate what an anthropological orientation can contribute to psychiatry by examining four key questions that anthropology raises concerning cross-cultural and international research in psychiatry. While almost all psychiatrists are impressed by the revolutions in biochemistry and neuroscience and their transforming effects on the profession, few realise that medical and psychological anthropology have also undergone a transformation which has made anthropological questions more pertinent to epidemiological, psychopathological, and treatment concerns.

Four Anthropological Questions for Psychiatry

To what extent do psychiatric disorders differ in different societies?

An anthropological reading of the literature in cross-cultural and international psychiatry reveals a strong bias of psychiatrists toward 'discovering' cross-cultural similarities and 'universals' in mental disorder. First, this bias should not come as any surprise. Much of cross-cultural psychiatric research has been initiated from a wish to demonstrate that psychiatric disorder is like other disorders: it occurs in all societies, and it can be detected if standardised diagnostic techniques are applied. Clearly this was an interest of the WHO's International Pilot Study of Schizophrenia (1973), which expected that core symptoms of schizophrenia would cluster together in more or less the same way in Western and non-Western, industrialised and non-industrialised, societies. It should come as no surprise that this is what the IPSS found, in a non-epidemiological, clinic-based comparison that applied a template of symptoms to psychotic patients in a range of societies to identify groups of patients who seemed similar. The problem with this study is that it leaves out those patients who fail to fit the template, the very patients of greatest interest from a cultural perspective, because they could be expected to reveal the greatest amount of cultural diversity. The IPSS also found that outcome varies inversely with the social development of the society – a striking finding attesting to cultural difference, but one that has taken back seat to the emphasis on cross-cultural similarity (World Health Organization, 1979).

Following IPSS came the Determinants of Outcome Study, a similar study but one which began in an epidemiologically more rigorous manner with first-contact incidence sample of patients with psychotic disorder. Let us look in detail at a report of the findings of this study of more than 1300 cases in twelve centres in ten countries (Sartorius *et al*, 1986). It is a very important project that has been conducted with more rigour than most in the cross-cultural field, and it illustrates the divergent perspectives of psychiatry and anthropology. The authors note:

> The frequency of the use of individual ICD subtype rubrics varied from 0 to 65% of the cases in the different centers. Overall, paranoid schizophrenia was the most commonly diagnosed subtype followed by that of 'other' (undifferentiated) and acute schizophrenic episodes. However, in the developing countries the acute subtype diagnosis was used almost twice as often (in 40% of the cases) as the diagnosis of the paranoid subtype (in 23% of the cases). Catatonic schizophrenia was diagnosed in 10% of the cases in developing countries but in only a handful of cases in the developed countries. In contrast, the hebephrenic subtype was diagnosed in 13% of the patients in the developed countries and in only 4% of the patients in developing countries.

Despite this impressive evidence of cross-cultural differences, the authors conclude:

> Patients with diagnosis of schizophrenia in the different populations and cultures share many features at the level of symptomatology . . .

and

> Once the existence of broad similarities or manifestations of schizophrenia across the centers was established . . .

The authors are of course correct that they do have evidence of 'broad similarities', evidence they choose to highlight. But they also have evidence of substantial differences that they choose to de-emphasise.

Or take, as another example, the data on annual incidence of schizophrenia. Two calculations are made – one for a 'broad' diagnostic definition of schizophrenia that includes virtually all cases sampled, and another for a restrictive definition based on the CATEGO computer program class S+. For the former, the rates per 10 000 population range from 1.5 in Aarhus to 4.2 in Chandigarh's rural center. For the latter, the range narrows impressively from 0.7 in Aarhus to 1.4 in Nottingham. Sartorius *et al* discuss this change by arguing that the application of the restrictive definition does not result in loss of statistical significance owing to decreased sample size. They conclude later in the paper that there is a relatively uniform rate of incidence for schizophrenia across the ten societies. From the perspective of psychiatric epidemiology and biostatistics this may be a valid conclusion, but from an anthropological perspective it is tautological. The 'broad' sample, from the anthropological perspective, is the valid one, since it includes all first-contact cases of psychoses meeting the inclusion and exclusion criteria. The 'restricted' sample is a 'constructed' sample, since it places a template on the heterogeneous population sample and stamps out a homogeneous group of clinic cases. The restricted sample demonstrates to be sure that a core schizophrenic syndrome can be discovered among first-contact cases in widely different cultures. This is an important finding, but it is not evidence of a uniform pattern of incidence. Indeed, the restrictive sample leaves out precisely those cases that show the greatest cross-centre and cross cultural differences and therefore the ones that disclose that the pattern of incidence is not uniform. This sample is of most interest to anthropologists since it demonstrates a wide range of cultural differences. Ironically, the finding of a wider range of incidence, which is in keeping with the finding in other studies of significant variation in the epidemiology of schizophrenia world-wide, should be of most interest to those concerned with the 'biological base', because this is what would be expected of a genetically based disorder. Several other key instances could be cited in which the evidence reviewed by the WHO group discloses both important similarities and important differences, but the authors elect to focus principally on the former, namely the 'universals'.

When this outstanding group of investigators review the data on the differential course of schizophrenia across centres (with better outcome in the developing societies), they report that this finding holds up even when mode of onset (acute versus chronic) is controlled. For over 10 years this finding has been the most provocative to emerge in cross-cultural psychiatry. Readers will be disappointed, however, if they hope to learn more about its sources or implications. The authors are silent on these points, which apparently have not received detailed investigation. That is to say, the most important finding of cultural difference – arguably the single most important finding in the study – receives scant attention compared to that devoted to the findings of cross-cultural similarity. In the summary of the paper, the other findings of cultural difference in mode of onset, symptomatology, and help-seeking are de-emphasised as well.

If this were a single instance of interpretive bias in cross-cultural psychiatric research, it would be one thing: in fact, it is representative of the dominant interpretive paradigm in cross-cultural psychiatry. There is a tacit professional ideology which functions to exaggerate what is universal in psychiatric disorder and de-emphasises what is culturally particular. This view fails to acknowledge, moreover, what the evolutionary biologist knows: biology is the great source of diversity, not just of uniformity (Mayr, 1982). Had I reviewed the anthropological literature, I could have demonstrated the obverse.

A more valid interpretation of the data base emerges when the two viewpoints, both biased but in opposite directions, are reviewed together. The findings for schizophrenia, major depressive disorder, anxiety disorders, and alcoholism disclose both important similarities *and* equally important differences. Hence the first anthropological question (How do psychiatric disorders differ across cultures?) is a necessary complement to the regnant psychiatric question (How are psychiatric disorders similar across cultures?).

Indeed, for all the talk about ethnological comparisons, anthropology is essentially a field of detailed, intensive, single-culture studies in which comparisons are made by a scholarly review of the literature on other cultures (Kleinman & Good, 1985) rather than by 'controlled' cross-cultural comparisons. Hence anthropological research points to the importance for psychiatry of scholarly analysis of studies of mental illness in one culture to deepen our understanding of cultural differences: in turn it benefits from psychiatry's example of undertaking multicultural projects in which mental illness is studied simultaneously in the same methodological framework in different societies – something anthropology has not done.

I have taken the liberty to be critical of the WHO research program because of its importance and the fact that the WHO group's studies are among the most rigorous and sophisticated in the cross-cultural field. I have every reason to believe that following this initial stage of demonstrating cross-cultural universals in mental illness the WHO group will begin to correct the balance by turning to the differences, which is already beginning to happen (c.f. Marsella *et al*, 1985).

Is there a tacit model in cross-cultural psychiatric research which exaggerates the biological dimensions of disease and de-emphasises the cultural dimensions of illness?

This question is a corollary of the first question. An anthropological analysis of cross-cultural research in psychiatry shows that the model of pathogenicity/pathoplasticity comes fairly close to being a professional orthodoxy. In this revealed version of the psychiatric truth, biology is presumed to 'determine' the cause and structure of disorder, while cultural and social factors, at most, 'shape' or 'influence' the content of disorder. The classical example is paranoid delusions in schizophrenia or depressive psychosis: the biologically based disease causes the delusional thought process but the system of cultural beliefs organises the content of paranoid thinking – here as fear that the CIA is out to harm one, there as belief that the KGB is the culprit.

The widespread finding that somatic symptoms in depression and anxiety disorders play a more central role in the experience and expression of disorder in non-Western societies and among ethnic groups in the West is another informative case. Somatic amplification and hypochrondriacal focusing are seen as distinctive manifestations of a similar underlying disorder. The latter is said to be 'masked' by the former. In this stratigraphic vision, biology is bedrock, whereas psychological, and in particular social and cultural, layers of reality are held to be epiphenomenal. They need to be stripped away to disclose the 'real' disease. As for medical anthropology's distinction between illness and disease – in which illness is the patient's perception, experience and communication of symptoms, while disease is the clinician's reformulation of the problem in terms of psychiatric models – disease is taken to be 'real' and hidden by the illness, which is a cultural artifact: catatonic or somatic or hysterical manifestations of the underlying causal process. Diagnosis then becomes reductionist, the semiotic interpretation of 'signs' of disease as an entity or object out of "the blooming, buzzing confusion" of illness symptoms.

The anthropological perspective suggests an alternative model. Depression experienced entirely as low back pain and depression experienced entirely as guilt-ridden existential despair are such substantially different forms of illness behaviour with different symptoms, patterns of help-seeking, course and treatment responses that though the disease in each instance may be the same, the illness rather than the disease is the determinant factor. There is overwhelming evidence in North American society that the social and psychological components of the illness experience of chronic pain are more powerful determinants of disability and return to work than the biological abnormalities, which are usually 'real' enough (Yelin *et al*, 1980; Stone, 1984). Hence in the newer medical anthropological model, biological and cultural factors dialectically interact. At times one may become a more powerful determinant of outcome, at other times the other, but most of the time it is the interaction (the relationship) between the two which is more important than either alone as a source of amplification or dampening of disability in chronic disorder. Furthermore, that dialectic may transform the biology just as it alters social relationships (Lewontin *et al*, 1984).

The pathogenetic/pathoplastic model also does an injustice to the culture-bound syndromes. The tendency is to interpret these as illness manifestations of particular 'underlying' disease: e.g., susto is depressive disorder; semen loss syndromes are anxiety disorders; amok is brief reactive psychosis; etc. The picture is much more complex. Carr (1978, 1985) shows that amok is a final common pathway of behaviour along which are channelled various kinds of problem: acute and chronic psychoses to be sure, but also criminal behaviour without psychopathology, alcohol- and drug-induced states, and so forth. Guarnaccia *et al* (1987) and Low (1985), among others, demonstrate that *ataque de nervios* among Hispanics is not simply conversion disorder, but may be anything from a medical disease, to schizophrenia, to a culturally appropriate bereavement reaction. The anthropological model of an idiom of distress offers a more accurate mapping of the experience of culture-bound disorder, and its sources and consequences, than does the medical model. Alternatively, the demonstration of Rubel *et al* (1984) that susto is associated with higher rates of mortality indicates the necessity of combining biomedical and anthropological assessments.

What place does translation have in cross-cultural research?

Medical, including psychiatric, research often procedes as if translation were a nuisance to be quickly handled in much the same way as one controls the demographics in matched samples. For psychologists, translation is rendered a technical problem, one that can be handled through a rigorous process of translation by one set of bilingual key informants, back-translation into the original language of the psychometric instrument by another set of bilingual informants, negotiation of the differences, and testing of reliability of the instrument, when compared with other measures of the same phenomena that have already been used in the recipient society, and when used by different groups of investigators. But, for the anthropologist, translation is neither of nuisance value nor a strictly technical problem; it is the very essence of ethnographic research. For the anthropologist, translation of findings into terms and categories that allow for cross-cultural comparison is the final, rather than as in psychiatric research, the first step. Therefore, the anthropological contribution to psychiatry in this area is to model a much more rigorous, systematic and contextual approach to translation. Let us examine a few examples.

Most assessment instruments are developed in a vernacular that is quite difficult to translate into other languages. North American diagnostic instruments, for example, employ the terms 'feeling blue' or 'feeling down' to assess dysphoria. A strictly lexical translation of these terms is meaningless in most non-Western languages. Manson *et al* (1985) translated the NIMH-sponsored Diagnostic Interview Schedule (DIS) into Hopi, an American Indian language. One DIS item combines the concepts of guilt, shame, and sinfulness. Twenty-three bilingual Hopi health care professionals clearly distinguished each of these concepts from the others and indicated that three separate questions were required to avoid confounding potentially different responses. Kinzie *et al* (1982) discovered much the same thing in developing a Vietnamese language depression scale. They found that 'shameful and dishonored' but not 'guilt' discriminated depressed from non-depressed Vietnamese Americans.

Gaviria *et al* (1984), working with a translation of the DIS in Peru, detected five kinds of problem in its validity that offer a broader sense of problems in translation. First, by content validity they meant that the content of instruments must be relevant in the culture into which the instrument is translated. Thus, many of the substances in the substance abuse section of DIS were unavailable in Peru; yet coca paste, a major drug in Peru, did not appear in DIS. Secondly, semantic validity requires that the words used in the original DIS and the new instrument have the same meaning, a problem we have already reviewed. Thirdly, technical validity refers to societies where languages are not written (or at most by an elite), schooling is limited and rates of illiteracy are high. In this context, the process of answering a questionnaire is foreign and one may elicit answers that represent a misunderstanding of intentions more than an accurate reflection of affect or thought. Fourthly, criterion validity measures whether responses to similar items relate to the same normative concept in two cultures. The presence of culturally consonant hallucinations among American Indians undergoing uncomplicated bereavement is normative in these tribal societies but not so in the broader North American culture. Assessment of hallucinations among American Indians must take account of this alternative norm. Fifthly, conceptual validity requires that responses to an interview relate to a theoretical construct within the culture. Neurasthenia is a key popular concept of disorder in China but no longer forms a coherent category for most North Americans (Kleinman, 1986). For a research interview to be conceptually valid in China, it would need to operationalise this category, not simply list its symptoms. In their entirety, several symptom check-lists include items that appear in locally salient syndromes like neurasthenia. By not organising these into operationalised syndromal clusters, the symptom check-lists fail to elicit subjects' responses.

Intra-cultural diversity makes the process of culturally meaningful translation even more complex. Canino *et al* (1987) found that in using the Spanish language version of the DIS developed at the University of California for use with Mexican Americans in a study of Puerto Ricans, they had to alter 67% of the items to adapt the instrument to the colloquial Spanish spoken by Puerto Ricans.

To adequately assess cultural differences, it is essential to translate local idioms of distress and add them to standard questionnaires. This may seem obvious, but it is not routinely done. For example, Ebigbo (1982) has demonstrated that Nigerian psychiatric patients have a unique set of somatic complaints which are not represented in standard symptom-screening scales. In a number of African cultures, anxiety is expressed as fears of failure in procreation, in dreams and complaints about witchcraft. To adequately evaluate psychopathology in these settings, those common idioms must be assessed. The most impressive attempt to take translation into account in a psychiatric epidemiological study is the study by Manson *et al* (1985) of depressive disorder among Hopi. They built an instrument with two components: a very carefully translated version of the DIS that met each of the issues reviewed above, and an operationalised list of symptoms and categories from Hopi indigenous nosology that had a prima facie resemblance to depression.

From an anthropological perspective, the problem of translation is that there may exist objective and universal referents for a term like headache which enables a process of searching for semantic equivalence. But, as Good *et al* (1986) note,

> In general, however, the referents of symbols – i.e., their meaning – are aspects of a culture or a life world, not objects outside of language through which language obtains meaning. 'Heart discomfort' for Iranians is not the equivalent of 'heart palpitations' for Americans; it does not *mean* the same thing (c.f. Good, 1977). It is a symbol which condenses a distinctive set of meanings, a culture-specific semantic network . . . Complaints of feeling impure in India refer to a semantic domain of profound cultural significance, one which expresses distinctions in caste, sexuality and social hierarchy: there is no equivalent among North Americans.

The Chinese term *huo qui da* refers to a feeling of heat rising from abdomen into the chest. It is based on the traditional Chinese medical concept that *qi* (vital energy) can be excessively hot and thereby cause symptoms. But it also metaphorically conveys the idea of an irascible personality. This metaphoric meaning would not be tapped by standard research instruments or translating practices: yet it is important for psychiatric assessment. Fear of obesity and of houseboundness in the USA does not carry the same meaning in South Pacific, African and Asian societies. Ideally, clinical care should provide a kind of culturally sensitive mini-ethnography of a patient that encompasses these cultural and personal metaphors. How to achieve this in a research project is a major problem. But the presence of such information can make a great difference in the validity of cross-cultural research findings.

Does the standard approach to cross-cultural research in psychiatry commit a 'category fallacy'?

A category fallacy (Kleinman, 1977) is the reification of a nosological category developed for a particular cultural group that is then applied to members of another culture for whom it lacks coherence and its validity has not been established. Shweder (1985), for example, suggests that among traditionally oriented rural populations, in many non-Western societies, the phenomenology of depressive disorder is better captured by local syndromes of 'soul loss' than by Western existential categories. A psychiatrist from such a society could operationalise the concept and symptoms of soul loss in his society, then organise them into a questionnaire, establish its reliability for use in his society, then translate its items into English, have them back-translated into the original language by another team of bilingual mental health workers, adjust the questionnaire for semantic equivalence, measure its reliability in the hands of native English-speaking clinicians, and apply the questionnaire to a stratified sample of an urban, middle class North American population in an epidemiological survey. He would come up with prevalence data. But would such data be valid, inasmuch as the disorder soul loss has no coherence for middle class North Americans? Obeyesekere (1985) has illustrated the same problem by conjuring up a South Asian psychiatrist studying semen loss syndrome among North Americans. This is a category fallacy, and it occurs routinely in cross-cultural psychiatric research, only the other way around, by the imposition of Western categories in societies for which they lack coherence and validity.

Dysthymic Disorder in DSM–III (or neurotic depression in ICD–9) is a possible example. It may hold coherence in the more affluent West, but it represents the medicalisation of social problems in much of the rest of the world (and perhaps the West as well), where severe economic, political and health constraints create endemic feelings of hopelessness and helplessness, where demoralisation and despair are responses to real conditions of chronic deprivation and persistent loss, where powerlessness is not a cognitive distortion but an accurate mapping of one's place in an oppressive social system, and where moral, religious and political configurations of such problems have coherence for the local population, but psychiatric categories do not. This state of chronic demoralisation, furthermore, is not infrequently associated with anaemia and other physiological effects of malnutrition and chronic tropical disorders that mirror the DSM–III symptoms of Dysthymic Disorder. In such a setting, is the psychiatrist who is armed with a local translation of DIS or other clinical instruments devised for Western populations in order to study the prevalence of Dysthymic Disorders any different from the Nuer psychiatrist studying soul loss in midtown Manhattan? Great care must be exercised in the application of this diagnostic category to assure that its use is valid.

For the psychiatric epidemiologist, determining a 'case' of a disorder from a person with distress but no disorder is the crucial dilemma. In making that distinction, taxonomy becomes entangled in its own decision rules. For patients with loss of energy due to malaria, appetite disturbance owing to the anaemia of hookworm, sleeplessness associated with chronic diarrhoeal disease, and dysphoria owing to poverty and powerlessness, the difference between 3 and 4 'vegetative' complaints is the difference between becoming a case of depression as a disease or being an instance where depression is a socially caused type of human misery. DSM–III was not created with such problems in mind, but it is applied in such settings. The result is as distorted a view of pathology as it is an inappropriate use of diagnostic categories.

Conclusion

Again, we confront the problem of validity. In a recent major statement on psychiatry, McHugh & Slavney (1986) define reliability as the "verification of observations" and validity as the "verification of presumptions". These influential American psychiatrists further define reliability as "the consistency with which one can make an observation". They claim that

> the reliability of some psychiatric observations is high. This indicates that psychiatrists can determine whether a patient does or does not have abnormal mental states such as delusions and hallucinations.

Reliability, following their own useful definition, indicates no such thing. It indicates only that the measurement of the observations is consistent. It does not tell us if the observations are valid: that is, whether a patient does or does not have an abnormal mental state. Here is an example. Ten psychiatrists trained in the same assessment technique and diagnostic criteria who are asked to examine 100 American Indians shortly after the latter have experienced the death of a spouse, a parent or a child may determine with close to 100% consistency that those individuals report hearing, in the first month of grieving, the voice of the dead person calling to them as the spirit ascends to the afterworld. That is reliability of observation. But the determination of whether such reports are a sign of an abnormal mental state is an interpretation based on knowledge of this group's behavioural norms and range of normal experiences of bereavement. Hearing the voice of the dead is an expected experience in bereavement among a number of American Indian tribal groups; this experience does not portend psychosis or other abnormal complications of bereavement. Thus, to interpret these normal experiences as 'hallucinations' with all the significance of pathology that term connotes is reliable but *not valid*.

For McHugh & Slavney, and many psychiatrists who are less theoretically sophisticated, observations are direct representations of reality. A word, hallucination, points to an empirical reality – i.e., abnormal mental state – in the real world. For the anthropologist, the word is a sign that signifies a meaningful phenomenon in a practical, day-to-day world mediated by a cultural apparatus of language, categories, taxonomies, and different hierarchies of relevance. In such a local world of culture, interpretations are always judgements whose reliability may be determined by consistency of measurements but whose validity needs to be established through *understanding* that particular cultural context. Validation is not simply verification of concepts used to explain observations. Rather it is as well verification of the meaning of the observations in a particular social system (a particular village, town, research laboratory, or clinic). Perception is a process of observing *and* interpreting in a practical situation guided by concerns of relevance influenced by human interests. Anthropologists would not say that clinical assessment cannot be validated, but that such validation is at heart an ethnographic enterprise. Thus, to avoid a category fallacy, cross-cultural research must be grounded in the local ethnographic context. This simply does not take place in most psychiatric studies.

I have tried to illustrate through these four questions some of the practical contributions of an anthropologically informed viewpoint to cross-cultural psychiatric research. Perhaps the most availing role for anthropology in relation to psychiatry is to continually remind us of these dilemmas, to challenge the hubris in our attempts to medicalise the human condition, to encourage humility in the face of alternative cultural formulations of human problems, which for anthropologists are different visions of the world rather than reflections of 'ignorance', and to make us uncomfortable with our taken-for-granted professional categories and the tacit 'interests' they represent.

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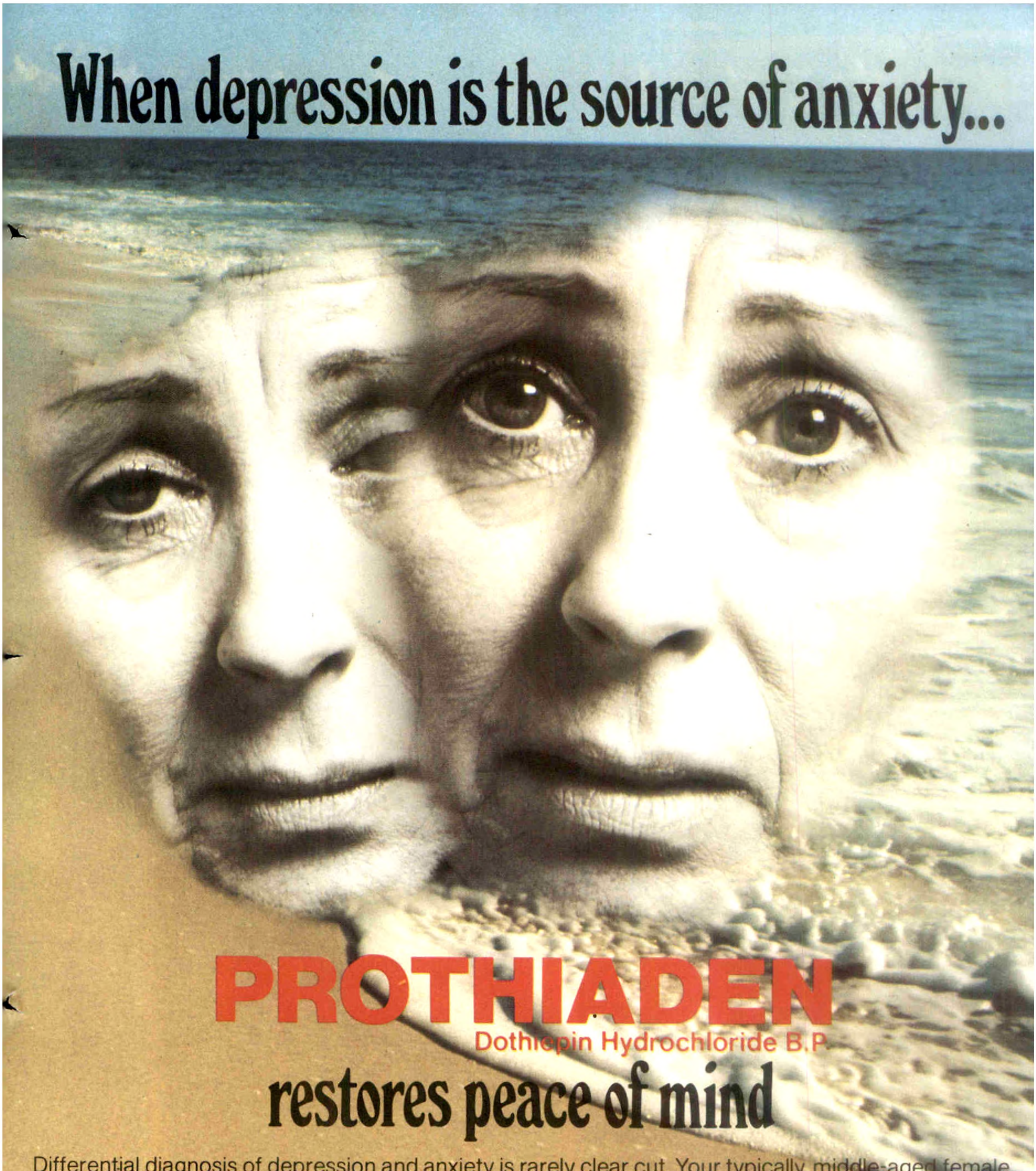
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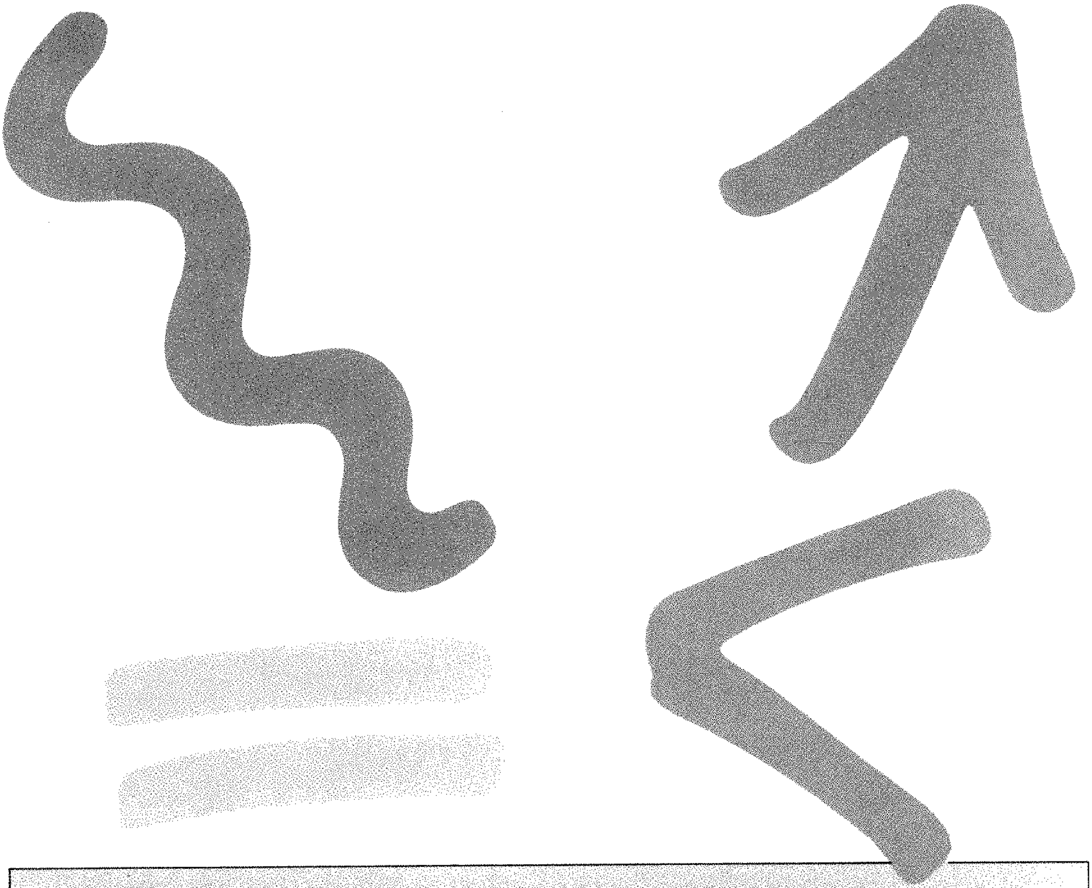

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Calcium-Activated Potassium Conductance

An Alternative to the Dopamine Hypothesis of Neuroleptic Action?

TIMOTHY G. DINAN

Neuroleptics are structurally a heterogenous group of compounds which possess antipsychotic activity. They increase dopamine metabolites by blocking dopamine receptors and enhancing presynaptic turnover. This forms the cornerstone of the dopamine hypothesis of neuroleptic action, which is supported by wide-ranging behavioural, physiological and biochemical studies. It is, however, clear that neuroleptics are far less specific for the dopamine receptor than was previously considered. They influence a range of neuronal activities, including calcium-activated potassium conductance, which governs the rate of action potential generation by many neurones. Recent physiological studies indicate that all commonly used neuroleptics alter calcium-activated potassium conductance in central neurones, in concentrations similar to those achieved clinically. An adaptive increase in calcium-activated potassium conductance mechanisms in key sensory processing neurones would render the psychotic patient less susceptible to bombardment by environmental stimuli. This action may explain in part the therapeutic effect of neuroleptics.

Neuroleptics are structurally a heterogeneous group of compounds which possess antipsychotic activity (Snyder *et al*, 1974). More than 20 years have passed since their capacity to increase dopamine (DA) metabolites, by enhancing the presynaptic turnover of DA, was demonstrated. Carlsson & Lindqvist (1963) observed that both chlorpromazine and haloperidol stimulate the accumulation of o-methylated metabolites of DA and also noradrenaline (NA). The authors postulated that these antipsychotic drugs act by blocking DA and NA receptors. A feedback activation was suggested as the mechanism underlying the acceleration of agonist synthesis. The work had a major intellectual impact and since then a vast literature has developed examining further the DA hypothesis of neuroleptic action.

Amphetamine is known to produce a paranoid psychosis in many respects indistinguishable from schizophrenia (Connell, 1958), and the stereotypic behaviour induced in rats by amphetamine has been advocated as an animal model of schizophrenia (Randrup & Munkvad, 1974). This latter behaviour pattern is claimed by Iversen (1978) to be due to overactivity of DA neurones and to be abolished when such neurones are selectively destroyed by the neurotoxin 6-hydroxydopamine or when postsynaptic receptors are blocked with neuroleptics. The potency of this response correlates highly with the rank order of neuroleptics in terms of clinical efficacy.

A high correlation ($r = 0.95$) has been found between the stimulating action of neuroleptics on striatal DA synthesis and their apomorphine antagonist action (Carlsson, 1978). Apomorphine is a powerful DA agonist and the fact that the ED_{50} values (amount of drug required to produce a 50% maximal response) in both cases are approximately equal, suggests similar affinities for receptors. Together with this, X-ray crystallographic studies indicate that chlorpromazine in its preferred conformation can be superimposed on DA and NA, while those phenothiazines lacking antipsychotic action are less liable to assume the conformation. This finding by Snyder *et al* (1974) provides a molecular mechanism by which phenothiazines can block DA receptors.

Further biochemical support

When DA receptors are labelled with ^{3}H-DA, the relative potency of phenothiazines against this receptor parallels their antipsychotic potency (Seeman *et al*, 1976). However, this high correlation is not maintained when the butyrophenones are tested. Snyder *et al* (1974) investigated the affinities of a wide range of phenothiazines and butyrophenones in competing for ^{3}H-haloperidol binding to the DA receptor. Spiroperidol, which is the most potent antagonist of DA-mediated behaviour in animals, was found to be the most potent inhibitor of ^{3}H-haloperidol binding.

It has five times greater affinity for this receptor than fluphenazine and a 40-fold greater affinity than chlorpromazine. Overall, a high correlation can be established between the molar pharmacological potencies of various neuroleptics and their affinities for the ^{3}H-haloperidol binding sites. The Johns Hopkins' group postulate that the DA receptors exist in two states, an agonist and an antagonist (Creese *et al*, 1975). This helps explain why DA agonists are over 50 times more potent in competing with ^{3}H-DA than ^{3}H-haloperidol for binding, while the reverse is true for antagonists.

Kebabian *et al* (1972) found that when various phenothiazines are compared, their potency as inhibitors of DA-stimulated cAMP formation is similar to their *in vivo* potencies as antipsychotics. The authors postulated that the postsynaptic actions of DA were mediated by a DA-sensitive adenyl cyclase, and that this enzyme activity correlates with the post-synaptic effects of DA. However, butyrophenones and diphenylbutylpiperidines have been shown to be less potent than chlorpromazine as inhibitors of adenyl cyclase (Iversen, 1975). The most potent butyrophenone spiroperidol is weaker than haloperidol or chlorpromazine in inhibiting DA-sensitive adenyl cyclase, while sulpiride, a benzamide derivative, is devoid of such activity. It now seems clear that there are at least two types of DA receptor: the D-1 type, which enhance adenyl cyclase activity, and the D-2 type, which either decrease or have little effect on cAMP (Cross & Owen, 1980). High affinity binding sites for ^{3}H-haloperidol correspond to the D-2 receptor, while high affinity binding sites for ^{3}H-flupenthixol identify the D-1 adenyl cyclase linked receptor. The DA hypothesis can easily adapt to the fact that there is more than one type of DA receptor and that neuroleptics vary in their potency at a single subtype of receptor. Thus Iversen's (1975) finding that butyrophenones are less potent than chlorpromazine as inhibitors of adenyl cyclase is accommodated.

Physiological support

Direct physiological evidence to support the DA hypothesis was provided by Bunney *et al* (1973). They found that iontophoresed DA on dopaminergic zona compacta neurones of substantia nigra produces a powerful inhibition which is abolished by neuroleptics administered either systemically or iontophoretically. When neuroleptics are given alone they significantly increase the rate of neuronal firing by blockade of the DA autoreceptor. Similar results have been reported for postsynaptic DA receptors. Recordings from septal neurones, for instance, have demonstrated that neuroleptics block DA-induced but not NA-induced inhibition (Bunney & Aghajanian, 1978). Further physiological evidence is provided by the fact that intravenous amphetamine decreases the firing of substantia nigra neurones but firing is returned to normal by chlorpromazine (Graham & Aghajanian, 1971).

Arguments against the DA hypothesis

It is now clear that neuroleptics are far less pharmacologically specific for the DA receptor than was previously thought. There is abundant evidence to indicate that they alter a wide spectrum of neurochemical activities. Chronic haloperidol, for instance, increases ligand binding not only to DA receptors but also serotonin and to a lesser degree alpha-noradrenergic receptors in the cerebral cortex (Mueller & Seeman, 1977). These latter changes in noradrenergic receptor binding are associated with profound alterations in the physiology of the noradrenergic nucleus locus coeruleus (Dinan & Aston-Jones, 1984, 1985). In fact, the affinities of many neuroleptics in competing for WB-4101 binding are similar to those of the classic alpha-antagonists phenotolamine and phenoxybenzamine (Peroutka *et al*, 1977). Not only can neuroleptics alter monoaminergic systems other than those which use DA as a transmitter, but there is also evidence to demonstrate their effects on cholinergic (Sherman *et al*, 1978), peptidergic (Somoze, 1978) and amino-acidergic (Perry *et al*, 1979) transmission.

Further arguments against the DA hypothesis have also been put forward. It must be remembered, for instance, that the number of dopaminergic neurones in the mammalian brain is relatively small and largely confined to the brainstem (Ungerstedt, 1974). Although these neurones clearly influence those at a cortical level, it has been argued that a disease of higher cognitive functioning such as schizophrenia must involve neurones at the highest level and not simply reflect a dysfunction of brainstem neurones, irrespective of how highly arborised they might be (Dinan, 1984). If such is the case, it seems unlikely that the modification of dopaminergic function would significantly influence the psychic processes involved in schizophrenia and thus adequately account for the antipsychotic action of neuroleptics. The fact that amphetamine psychosis closely resembles paranoid schizophrenia is regarded as strong evidence for implicating DA with the psychopathology of schizophrenia (Iversen, 1978); however, there has been little in-depth investigation of amphetamine action on extra-monoaminergic systems, which might also conceivably mediate the psychic effects of amphetamine. The bluderbuss lesioning of DA neurones abolishes the stereotypic behaviour produced by amphetamine in rats (Iversen, 1978), clearly implicating DA in stereotypic behaviour, but obviously not negating possible non-dopamine actions of amphetamine.

Neuroleptics and calmodulin

The potent action of butyrophenones and phenothiazines as calmodulin antagonists has been described (Weiss & Levin, 1978). Calmodulin plays a pivotal role in mediating the second messenger actions of calcium within the neurone, including the activation of a variety of enzyme systems (Stoclet, 1981). The piperazine phenothiazine, trifluoperazine, has been shown not only to be a potent calmodulin antagonist but also to reduce inward calcium currents in neurones (Chapman & Neher, 1984). Together with other neuroleptics, it depresses platelet aggregation induced with platelet-activating factor, which Levy (1983) indicates is mediated by a calcium/calmodulin mechanism. Studies on the diphenylbutylpiperidines such as pimozide indicate their capacity to inhibit nitrendipine binding to the verapamil/prenylamine class of calcium channel (Gould *et al*, 1983). They also inhibit potassium-induced calcium-dependent contractions of rat vas deferens (Gould *et al*, 1983).

Calcium-activated potassium conductance

Many neurophysiologists are actively engaged in a study of Ca^{+2}-activated K^+ conductance mechanisms in a wide variety of neurones. This conductance is important in governing the rate of action potential generation by a neurone and thus the rate of transmitter release (for review see Meech, 1978). The process begins with the opening of voltage-gated Ca^{+2} channels, resulting in the movement of Ca^{+2} inward. This Ca^{+2} together with that mobilised within the cell brings about the opening of K^+ channels, probably acting via an intermediate calmodulin-like protein. The K^+ moves along its electrochemical gradient, causing a hyperpolarisation of the membrane, which can be sustained, in many cases, for several seconds. The mechanism thus plays an important role in determining the rate of information processing by an individual neurone and in modulating the signal transmitted by the neurone.

Recent work both on the noradrenergic nucleus locus coeruleus (Dinan & Aston-Jones, 1984, 1985; Dinan, 1986) and the hippocampal pyramidal CA1 neurones (Dinan *et al*, 1987) indicates that neuroleptics alter Ca^{2+}-activated K^+ conductance. When acutely administered, those neuroleptics effective as antipsychotics depress the slow afterhyperpolarisation seen in hippocampal CA1 neurones. This afterhyperpolarisation is mediated by a K^+ conductance which is Ca^{+2}-dependent. In the locus coeruleus, haloperidol depresses both orthodromic- and antidromic-induced poststimulus inhibition, which is again mediated by a similar conductance. Dinan & Aston-Jones (1985) have shown that chronic haloperidol treatment results in the inactivation of many locus coeruleus neurones, rendering them less sensitive to incoming stimuli. White & Wang (1983), working on zona compacta neurones of the substantia nigra, report similar findings.

Defective filtration in schizophrenia

Broadbent (1958, 1971, 1977) has postulated a defect in the perceptual filtration mechanism in schizophrenia, which results in the individual being bombarded by environmental stimuli, overloading his cognitive processing capacity. Several studies support this delineation of specific defects in the filtering phase of information processing. Broen & Nakamura (1972) found that chronic non-paranoid schizophrenics demonstrate a more restricted range of sensitivity toward peripheral sensory channels than do acute paranoid schizophrenics or normal subjects. They also found that schizophrenics perform significantly less well than controls on span-of-apprehension tests when irrelevant noise is present, but no differences are noted when there are no irrelevancies. Schizophrenics also perform poorly when tasks which involve switching attention between stimulus modalities are employed, or in tests of discrimination shifts (Kristofferson, 1967).

The acute antipsychotic actions of neuroleptics, together with their long-term prophylactic effects, may partly be explained in terms of rectification of a defective perceptual filter. As Ca^{+2}-activated K^+ conductance is an important processing mechanism for many sensory input neurones, it provides a key neuronal site for altering the filtration of environmental and internal sensory information. For example, some visual system neurones rely heavily on such a conductance for processing information (Llinas & Lopez-Barneo, 1983). An adaptive increase in Ca^{+2}-activated K^+ conductance in such neurones, in the weeks following commencement of neuroleptic treatment, would render the individual less susceptible to bombardment by environmental stimuli. We know for instance that neuroleptics enable schizophrenics to withstand greater levels of expressed emotion from the environment without breakdown (Brown & Birley, 1968; Leff & Wing, 1971).

Crow *et al* (1976) have provided a concise summary of the DA hypothesis:

> Since many behavioural effects of the amphetamines are due to increased central DA release and since most neuroleptic compounds are blockers of central DA receptors, the symptoms of schizophrenia may be due to abnormal increase in central DA release, and the effects of this increase are diminished by partial blockade of the receptor site.

The hypothesis so stated is clearly heavily influenced by our present understanding of the aetiology of Parkinson's disease and does not adequately explain how an increase in DA might produce the intriguing psychopathology of schizophrenia. The present hypothesis is not implying that schizophrenia is due to underactivity of Ca^{+2}-activated K^{+} conductance mechanisms in central neurones. It simply states that schizophrenics process information from their environments inappropriately and one site at which pharmacological manipulation might alter such aberrant processing is at the level of Ca^{+2}-activated K^{+} conductance mechanisms in sensory input neurones. The theory takes the focus from the receptor site and directs it on the neurone as an information processing unit.

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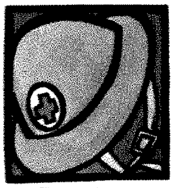

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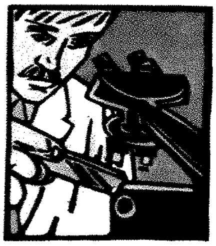

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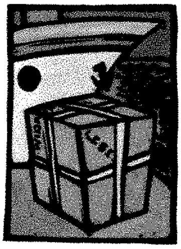

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Commentary on Dinan's Hypothesis*

LESLIE L. IVERSEN

Dinan's paper seeks to question the validity of the now widely accepted hypothesis that neuroleptic drugs of various different chemical categories all act by a common mechanism, namely by blockade of dopamine receptors of the D2 type in brain (for review see Creese *et al*, 1978, 1983; Iversen, 1985). While it is always refreshing to re-examine existing scientific dogma, his criticisms did not appear to me to be very substantial. Having quite fairly reviewed the evidence in favour of the "dopamine hypothesis" for neuroleptic drug action, Dinan summarises his reasons for questioning it. He points out that many neuroleptic drugs have potent actions on a number of other targets, apart from the dopamine receptors in brain. This is indeed so, and the archetypal compound, chlorpromazine, has a particularly rich spectrum of pharmacological activity, being a potent antagonist of serotonin (5-HT_2) receptors, α-adrenoceptors, and histamine (H1) receptors in brain and other tissues. Other neuroleptics also have potent actions on a number of other systems. Indeed, if one were to have studied only chlorpromazine, it is doubtful whether the "dopamine hypothesis" could ever have been developed. The strength of this hypothesis lies in the fact that of all the diverse pharmacological actions which different neuroleptic drugs exhibit, this is the only action that is common to *all* neuroleptic compounds. Furthermore, in a large group of neuroleptics, which differ widely in potency (doses in man ranging from 1 mg/day to almost 1000 mg/day), the potencies of these drugs as dopamine (D2) antagonists correlate significantly with their clinical potencies. Attempts to make such correlations with any other known pharmacological properties of these drugs fail to show significance (Creese *et al*, 1978, 1983).

To suggest, as Dinan does, that "a disease of higher cognitive functioning such as schizophrenia must involve neurones at the highest level and not simply reflect a dysfunction of brainstem neurones . . . " is simple prejudice, not based on any scientific facts. In any case, dopamine neurones do exist in the limbic forebrain, and they send important projections to certain areas of frontal cortex.

Dinan's alternative proposal to explain how neuroleptic drugs act is that they block a particular ionic channel in neuronal membranes, a calcium-activated potassium conductance. This may be related to the known property of some neuroleptics to act as antagonists of the calcium–calmodulin system. The new findings reported by Dinan *et al* are original and of interest, showing that haloperidol, chlorpromazine and trifluoperazine have this effect

*Commentary on preceding paper by T. G. Dinan.

when added *in vitro* to rat hippocampal slice preparations. However, these are only three of the many neuroleptics that exist; to suggest that this is a proven action for "all commonly used neuroleptics . . . in concentrations similar to those achieved clinically" (as Dinan states in his summary) is to extrapolate wildly beyond the available experimental results. To test whether there was any correlation between this effect and neuroleptic activity, a much larger number of compounds would need to be tested, including pairs of compounds such as (+) and (−) butaclamol and (α) and (β)-flupenthixol, where in a pair of isomers it is known that only one compound possesses neuroleptic activity. The question of drug concentration is also relevant, since the actions described by Dinan *et al* are elicited only at micromolar concentrations, much higher than those found free in body fluids clinically (although larger amounts of neuroleptic may be present in plasma tightly bound to protein), and also 100–1000 times higher than the concentrations required for dopamine receptor blockade.

The author goes on to relate these new findings to Broadbent's hypothesis of a defect in perceptual filtering in schizophrenic illness. The neural mechanisms involved in such a phenomenon, however, are quite unknown, so it is impossible to relate this is in any way to changes in ionic mechanisms as suggested.

Overall, Dinan seems to fall into many of the familiar pitfalls that have lured others in this field, and his arguments failed to convince me. It is inherently difficult to explain the actions of psychopharmacologically active compounds in terms of a molecular mechanism, but the "dopamine hypothesis" for explaining the actions of neuroleptics comes close to this, and has been one of the major achievements of research in this area in recent years.

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The Dopamine Hypothesis Survives, but There Must Be A Way Ahead*

T. J. CROW

Karl Popper has taught us that no hypothesis can be regarded as proved – merely as having survived the most stringent tests which have so far been devised. Dinan makes a further attempt to overthrow the dopamine hypothesis of the mechanism of the antipsychotic effect, and provides an alternative suggestion. As one time critic and later advocate of the theory, I think he underestimates its explanatory power and capacity for survival. I doubt that his alternative yet poses a serious challenge. I propose below a second (and perhaps more idiosyncratic) alternative. I do not claim it yet has the explanatory power of the dopamine theory, but I think it generates some new approaches.

The survival of the dopamine hypothesis

A forerunner of the dopamine hypothesis was the view of Deniker (1960) that the extrapyramidal effects of neuroleptic drugs are related to their therapeutic influence in schizophrenia. This view was widely doubted, principally because the two actions appeared dissociated in some drugs (e.g. thioridazine). With the discovery that dopamine is depleted in the striatum in Parkinson's disease (Ehringer & Hornykiewicz, 1960) and that some neuroleptics act on dopaminergic mechanisms (Carlsson & Lindqvist, 1963), the hypothesis became more explicit and testable in the form that neuroleptic drugs exert their therapeutic effects by interfering with dopaminergic transmission. With the development of the dopamine sensitive adenylate cyclase assay for dopamine antagonist activity, it was established that, for the range of phenothiazine and thioxanthene compounds, dopamine antagonist activity correlates well with therapeutic potency.

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*Commentary arising from preceding paper by T. G. Dinan

However, two serious problems for the hypothesis emerged. First, the butyrophenone compounds were less effective in this assay than was predicted from their therapeutic effects. Secondly, discrepancies between dopamine antagonist activity in behavioural assays and therapeutic activity were apparent (Crow & Gillbe, 1974). For example, thioridazine appeared to be much less effective than chlorpromazine in an assay of dopamine antagonist activity (inhibition of amphetamine-induced turning behaviour in rats with nigrostriatal lesions), while the two drugs were approximately equipotent in reducing locomotor activity, an action which therefore seemed more closely related to their therapeutic effectiveness.

The first problem was resolved when the butyrophenone binding assay (for what is now described as the D2, or non-adenylate cyclase-linked receptor) was developed (Seeman *et al*, 1976). On this receptor the potency of the butyrophenone compounds relative to the phenothiazines and thiaxanthenes was shown to be comparable to their therapeutic effects.

The second problem was removed when it was demonstrated in the turning rat model that cholinergic antagonism reverses apparent dopaminergic blockade (Muller & Seeman, 1974). It appears that some neuroleptic drugs (e.g. thioridazine) also possess muscarinic cholinergic antagonist activity (Miller & Hiley, 1974). This accounts for their low incidence of extrapyramidal effects and low potency in the turning-rat model of DA activity.

The dopamine hypothesis thus was able to explain what previously appeared as discrepant observations. This increases one's confidence in its explanatory power.

A further stringent test of the hypothesis was made possible by the observation that DA antagonist activity of the thiaxanthene compounds is limited to the *cis*-stereoisomers (Miller *et al*, 1974). In a clinical trial in patients with acute episodes of illness we were able to show (Johnstone *et al*, 1978) that the *cis*-isomer of flupenthixol is significantly more active than the *trans*-isomer, which itself is no more active than placebo. For several pharmacological actions (e.g. noradrenergic, cholinergic and opiate antagonist activity) the two isomers do not differ. Therefore the antipsychotic effect presumably is unrelated to these receptors. The two isomers differ with respect to dopamine and serotonin antagonist activity. However, the serotonin antagonist potency of psychotropic drugs correlates poorly with antipsychotic efficacy. Therefore dopamine antagonism explains the stereospecificity of the antipsychotic effect and no ready alternative explanation is available.

Therefore one must ask concerning Dinan's hypothesis that actions on calcium-activated potassium conductance are relevant to the anti-psychotic effect: Is thioridazine approximately equivalent to chlorpromazine in this respect, and are both these drugs significantly more effective than promazine? Are the butyrophenones more potent than the phenothiazines and thiaxanthenes? Are the *cis*-isomers of the thiaxanthenes more potent than the *trans*-isomers? Unless the answers to each of these questions is yes, it seems that the Dinan hypothesis lags behind dopamine antagonism as an explanation of the antipsychotic effect.

Is there dopamine overactivity in schizophrenia?

Another origin of the dopamine hypothesis lies in the observation that the amphetamine psychosis closely resembles paranoid schizophrenia (Connell, 1958) and that the amphetamines exert their major behavioural effects by enhancing dopamine release in the brain (Randrup & Munkvad, 1966, 1967). Randrup & Munkvad (1965) were able to show that the behavioural changes induced in rodents by the amphetamines are selectively reversed (i.e. with a return of previously suppressed normal behaviours) by administration of neuroleptic drugs, but not by other sedative agents. These observations rather strongly suggest that it is the selectivity toward dopaminergic mechanisms which confers upon neuroleptics their particular properties; they also suggest the more ambitious formulation of the hypothesis that some form of dopaminergic overactivity underlies the positive symptoms of the disease (Randrup & Munkvad, 1972).

Compelling though these observations are, a caveat should be entered. Although everything seems consistent with the view that the amphetamines exert their psychotogenic effects via dopamine release, it remains puzzling why, if this is so, direct dopamine receptor agonists (e.g. apomorphine, bromocriptine) are not found to be psychotogenic in the same way.

The relative lack of psychotic potency of direct dopamine agonists remains something of an embarrassment for the dopamine theory of psychosis.

The theory would be greatly strengthened if it were possible to demonstrate an abnormality of dopaminergic transmission in patients. There is no compelling evidence of increased activity of dopamine neurones either from cerebrospinal fluid studies or from assessment of post-mortem brain samples for metabolites of dopamine (Crow *et al*, 1984). Investigations of the status of the D2 dopamine receptor, however, are at an interesting stage. Studies on post-mortem brain samples agree in finding an increase of approximately 80–100% in numbers of D2 receptors (Lee *et al*, 1978; Owen *et al*, 1978; Mackay *et al*, 1982; Cross *et al*, 1981), but the question of whether or not this change can be attributed to past neuroleptic medication is unresolved. There are now a number of techniques for studying the D2 receptor *in vivo*, and some of these have already been applied to patients whose drug-free status can be securely established. In one study with ^{77}Br-spiperone using single-photon emission tomography (Crawley *et al*, 1986), an increase ($P<0.05$) in striatum/cerebellum ratios of 11% by comparison with controls was observed in 12 patients who had been free of neuroleptics for at least 6 months. In a positron emission tomography (PET) study, a much larger increase (approximately 150%) was demonstrated in drug-free and drug-treated schizophrenics in maximum binding of ^{11}C-methylspiperone estimated by a method which involves administration of haloperidol between a first and second scan (Wong *et al*, 1986). Thus there are indications that D2 receptors may indeed be increased. However, there are problems with both techniques and it will be of particular interest to see whether the increase is confirmed by the PET technique using ^{11}C-raclopride (Farde *et al*, 1986). If it can be established that D2 receptor changes are associated with some component of the disease process, this will provide an explanation that will be difficult to resist of why it is that dopamine antagonist drugs ameliorate symptoms.

Is dopamine a plausible substrate for schizophrenic symptoms?

An element in Dinan's critique of the dopamine hypothesis is the suggestion that dopamine neurones are an unlikely location of the primary disturbance, because as a component of the basal ganglia they are not involved in higher cognitive activity. However, this argument ignores a number of considerations. First, the dopamine systems arising from the ventral mesencephalon project not only to the striatum but also to the nucleus accumbens (which has direct connections with hippocampus and other limbic structures) and the frontal cortex. In these sites, if not in the striatum, it may be supposed that dopamine release has an influence on higher cognitive processes.

Secondly, there is evidence that dopamine neurones, even in the striatum, have more than the simple motor function which is suggested by observations on Parkinson's disease. For example, Marshall *et al* (1974) and Ljungberg & Ungerstedt (1976) have drawn attention to the phenomenon of sensory inattention after unilateral nigrostriatal lesions.

Thirdly, there is a strong case that activation of dopamine neurones has rewarding as well as activating effects, as was first suggested on the basis of a study in which the rat mesencephalon was mapped for electrical self-stimulation responding (Crow, 1972).

These observations can be accommodated within the concept that dopamine neurones function as an 'incentive motivational' system which energises and directs the organism's responses toward significant environmental stimuli (Crow, 1973; Crow & Deakin, 1985). It is of interest that an early behavioural interpretation of the mechanism of action of neuroleptics is compatible with effects on such a system. On the basis of their experiments on chlorpromazine, Olds & Travis (1960) suggested that the drug "acts against a system which controls operant or 'voluntary' behaviour" and speculated that "counteraction of positive feedback processes subserving positive reinforcement mechanisms may be a key to the control of certain psychotic episodes".

Thus, in addition to the evidence from amphetamine psychosis that dopaminergic overactivity is correlated with psychosis, it seems that the functions of dopamine neurones are not inconsistent with their having a role in the pathogenesis of schizophrenic symptoms. One of the problems with Dinan's theory is that it appears to make no predictions about the selectivity of the effects of neuroleptics. Thus one might expect calcium activation of changes in potassium conductance to be characteristic of a wide range of neuronal elements (e.g. motor neurones and Purkinje cells as well as cortical neurones). Without further development it is difficult to see how the theory can account for the specificity of the antipsychotic effect.

An alternative to the dopamine hypothesis? – antiviral activity

Whether or not there is a disturbance of dopaminergic transmission in schizophrenia, the theory that neuroleptic drugs act by blocking dopamine receptors appears by far the best explanation we have of their effectiveness in treatment. Nevertheless, not least because their therapeutic effects are limited and no great advance in treatment is in prospect, we have to consider alternative possibilities.

One such alternative that intrigues me is that these drugs act by an antiviral mechanism. A number of recent authors (Torrey & Peterson, 1976; Crow, 1978; Hare, 1979) have thought that schizophrenia might be caused by a virus. I considered the possibility that the disease might be due to an exogenous pathogen (Crow, 1983) but dismissed this on the grounds that age of onset appears to be intrinsic to predisposition to the disease, i.e. it is not environmentally determined (Crow & Done, 1986). This consideration narrows down the type of viral agent that could be responsible to the class of agents that are either acquired *in utero* or integrated in the genome, i.e. transmitted as a gene (Crow, 1984). Included in the latter category are some types of retrovirus (Teich, 1982). In mice, integrated retroviruses are known to cause specific diseases, e.g. mammary tumours and leukemias. It is of considerable interest that actions of some neuroleptic drugs on diseases caused by retroviruses in mice have been demonstrated. For example, lytic actions of a variety of neuroleptic drugs on retroviruses *in vitro* have been reported (Wunderlich & Sydow, 1980), and suppression of disease manifestations caused by retroviruses has been reported in the case of mammary tumours by reserpine (Wunderlich & Zotter, 1982) and Rauscher leukemia virus by haloperidol (Wunderlich *et al*, 1980).

We recently investigated possible antiviral effects of a limited range of psychotropic drugs (including chlorpromazine and lithium) on herpes simplex virus infectivity and synthesis *in vitro* (Patou *et al*, 1986). Reductions in virus titre were observed with several compounds, including lithium. However, among the neuroleptics tested there was no relationship with antipsychotic efficacy; for example, promethazine had effects but trifluoperazine did not, and *trans*-flupenthixol was somewhat more effective than the *cis*-isomer. It remains possible that more specific effects would be observed on integrated pathogens, particularly those expressed in the central nervous system. This line of investigation seems worth pursuing.

If an antiviral mechanism of action is postulated, the question of what role dopamine plays must also be considered. One possibility is that the relation between antipsychotic potency and dopamine antagonism is no more than a coincidence. This cannot be excluded, but for the reasons given above it seems unlikely. Two other possibilities come to mind. The first is that changes in dopaminergic function are secondary to pathological changes elsewhere. There is increasing evidence for a focus of structural change in the temporal lobe (Brown *et al*, 1986; Bogerts *et al*, 1985). Czernansky *et al* (1983, 1985) have shown in animal experiments that an epileptic focus in the amygdala can cause changes in numbers of dopamine receptors in the striatum. Similarly Pycock *et al* (1980) describe how an increase in receptor numbers in the striatum follows lesions to the frontal dopaminergic projection. Along these lines one might propose that episodes of positive symptoms (assuming these to be dopamine related) are only an indirect consequence of the primary pathological change. In this case the effects of neuroleptics would be entirely symptomatic, a possibility which is not seriously challenged by the existing literature.

The second possibility is that there is some more direct relationship between the pathological agent and the dopamine receptor. For example, one might consider whether the agent gains access to particular cells by way of the dopamine receptor in the way that the human immunodeficiency virus gains access to lymphocytes via the T4 receptor. Alternatively, one could hypothesise that the virus is integrated in the genome at a site which is related to the transcriptional control of the receptor.

Both these possibilities concerning the role of dopamine deserve consideration on the grounds that they might open up new approaches and also perhaps explain the prophylactic efficacy of neuroleptics, which sits somewhat uneasily with the notion that these drugs act by a simple receptor blockade.

Concluding comments

The dopamine theory of the action of neuroleptic drugs remains the most plausible explanation of their therapeutic effects because

(a) it accounts well for the relative potency of the whole range of phenothiazine, butyrophenone and thiaxanthene drugs, including the stereoisomers of flupenthixol

(b) it is consistent with the fact that agents (e.g. amphetamines), acting by dopamine release, have psychotogenic effects, and that both the psychotomimetic effects in man and behavioural effects in animals are reversed by neuroleptic drugs.

Although it is as yet unclear whether there are disturbances of dopaminergic transmission in schizophrenia, an increase in receptor numbers remains a possibility and is supported by recent imaging studies. Moreover, the function of dopamine neurones in sensori-motor integration as revealed by animal experiments is not inconsistent with a disturbance of these systems contributing to psychosis. The two major outstanding difficulties for the dopamine hypothesis are the relative lack of psychotogenic effects of direct dopamine agonists, and the apparent weakness of simple dopamine antagonism as an explanation of the prophylactic efficacy of these compounds.

Notwithstanding its general success, the dopamine hypothesis does not seem to be generating further progress, particularly the introduction of more effective treatments. Therefore alternative hypotheses must be considered. One such hypothesis is that these drugs have actions on pathogens that are integrated in the genome but expressed to cause episodes of disease. While it is conceivable that such a mode of action might bear a relation to dopaminergic transmission (e.g. if the pathogen has an affinity to the D2 dopamine receptor), the hypothesis (like other current theories of the antipsychotic effect) lacks the explanatory power of the dopamine theory in that it has yet to be subjected to the experimental tests that the dopamine hypothesis has survived.

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Is There More Dementia, Depression and Neurosis in New York?

A Comparative Study of the Elderly in New York and London Using the Computer Diagnosis AGECAT

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A computerised diagnosis, AGECAT, is applied to data from random community samples of the elderly in New York and London in order to examine the prevalence of mental illness in the two cities, especially the result reported from the same study, using a different diagnostic procedure, that the proportion of dementia was higher in New York. The greater proportion of dementia in New York is confirmed for all AGECAT's levels of diagnostic confidence, for both sexes and at all half-decades over 65 years. Provided that this difference is upheld and genetic differences can be discounted, these findings would seem to support a contributory environmental cause for dementia. Proportions of depression as a whole are similar in both cities. Syndrome case and sub-case levels of neurotic disorder are generally higher in London.

From a study of the elderly aged over 65 living in the communities of New York and London, the US–UK Cross-National (Diagnostic) Project reported the prevalence of dementia in New York to be significantly higher than that in London (Gurland *et al*, 1983). Because only the London sample had been interviewed and diagnosed entirely by psychiatrists, it had not been possible to compare psychiatric diagnosis between the two cities. An algorithm had therefore been devised by which subjects could be allotted by the interviewer to levels of illness described as 'pervasive' dementia and 'pervasive' depression. Here we re-examine the data by applying a recently developed computerised diagnosis, AGECAT.

Epidemiological studies in the past have suffered from poor method. Representative samples have always been difficult to draw, and early studies employed unstructured methods and intuitive diagnoses. More recently semi-structured interviews have been used and attempts have been made to standardise the diagnostic process. In 1986 Copeland *et al* reported the development of a computerised diagnostic system, AGECAT, which could be applied to data derived from the Geriatric Mental State (GMS) and the CARE interview. In the study described here, both a semi-structured interview and a computer diagnostic system are used to provide a reliable 'yardstick' for comparison between two community samples.

Method

What is being measured?

It is important that a case be defined for the purpose for which it is to be used (Copeland, 1981*a*). However, there are stages in the definition of a case which we have attempted to define elsewhere (Copeland *et al*, 1986). At the point where symptoms group to form a pattern recognised by a psychiatrist as an illness, we identify a 'syndrome' case. Such a case is a profile of symptoms only. If this is the only syndrome present, or if several syndromes reach case level in the same individual and the primary one is selected, the subject becomes a diagnostic case of that syndrome. For the London/New York community study further criteria were introduced: that the severity of the condition merited professional intervention, and that the subject's age be restricted to 65 years and over. As these further criteria were added to that of the pure syndrome we have called these 'criteria cases for intervention'.

The History and Aetiology Schedule (HAS) for an informant, which deals with historical facts, was not developed in time for this community study. Therefore, the AGECAT system as used here is only able to identify cases at the *syndrome* and *diagnostic syndrome* level. In consequence there may be some 'over-diagnosis' of cases which, although fulfilling the criteria for syndrome cases, would not have merited intervention in the judgement of the psychiatrists; for example, states of bereavement, and longstanding and distressing fluctuations of mood, which share a cross-sectional mental-state picture of depression. Nevertheless, we have now shown a satisfactory concordance between AGECAT and psychiatrists' diagnoses in a number of studies (Copeland *et al*, 1986, 1987).

Interview method

In 1976 the development of the GMS, a semi-structured mental-state interview for the elderly, was reported (Copeland *et al*, 1976, Gurland *et al*, 1976). For the community a much shortened version was prepared, omitting many of the psychotic sections, to which was added a large number of social and general medical items, to form the CARE interview. The CARE interviews have since been modified and different forms are now available.

AGECAT

The AGECAT system was developed on a theoretical model based on clinical experience (Copeland *et al*, 1986). It was then tested against samples of patients, readjusted and retested. It brings together items from the GMS and those schedules derived from it into 157 symptom components, which are then formed into eight diagnostic 'clusters', each consisting of a number of groups representing symptoms important for diagnosis. Scores on these groups determine the level of diagnostic confidence for each subject on each diagnostic cluster. The diagnostic clusters are organic, schizophrenic, manic, depressive (psychotic and neurotic), hypochondriacal, obsessional, phobic and anxious.

In stage 2 the levels are compared across clusters, following the above hierarchy, so that levels towards the beginning tends to dominate those towards the end. A principal diagnosis emerges, an alternative diagnosis if appropriate, and for each diagnostic cluster, the corresponding levels of diagnostic confidence or certainty (Dewey & Copeland, 1986). In addition, a decision is made on whether or not the symptoms rank as a syndrome case. For all clusters, levels of confidence 3, 4 and 5 represent syndrome case levels, and 1 and 2 sub-case levels. Symptom profiles can be derived, and organic and depression scores suitable for case-finding can be quickly calculated.

In the full diagnostic system the results of stage 2 are processed with data from the HAS (stage 3) and the diagnostic syndromes are broken down into individual diagnoses. This stage was not available for this analysis.

Interview personnel

All subjects in the London sample were interviewed by research psychiatrists of senior registrar status or above, employed full-time by the project. In New York only half the sample was seen in this way; the rest were interviewed by social scientists who were trained in the method and whose reliability had been satisfactorily assessed against psychiatrists' ratings. Reliability studies were performed between interviewers and between teams and found to be satisfactory. Teams exchanged members so that all principal interviewers interviewed in both cities.

Sample

In London the study area chosen was Greater London. A random sample was taken of 3000 general practitioners serving Greater London. Permission was obtained from each chosen general practitioner, and his patients aged over 65 were random-sampled at the local Family Practitioner Committee's premises. Only one practitioner refused to co-operate with the study and his patients were 'lost'.

In New York a state sample was obtained from the New York State Office for Ageing. In all 1726 households were chosen in metropolitan New York, obtained in 66 dwelling clusters throughout the five boroughs. A series of random sub-samples was drawn in each city, and a comparison of the consistency of the sub-samples provided a check on how well they represented the whole. Each dwelling unit was visited up to four times; if no respondent was found, information was sought from neighbours where possible. The interview response rates were 81% in London and 71% in New York. In all, 396 interviews were completed in London and 445 in New York.

Results

Demography

Table I shows the age distribution of the sample. The mean age was 74 in both cities, and the distribution by half-decades was similar, except that the proportion of males aged 80–89 in the London sample was less than half that in New York. This group would have been aged 18–30 at the time of the outbreak of the First World War. At over 90 years the number of males was very small but the proportions were similar in both cities. The rest of the demographic findings, which have been described in detail by Gurland *et al* (1983), will only be summarised here. In the New York sample 10% were black, 39% Catholic and 35% Jewish, compared with 1%, 10% and 3% respectively in London. In the New York sample 60% had been immigrants, compared with only 10% in London. The distribution on occupational status was much the same in the two cities, with a small preponderance of unskilled workers in London. Marital status showed no significant differences. A census of New York City and one of London, both taken in 1976, showed a close approximation for sex and age with the corresponding city sample described here (New York State, 1976; HMSO, 1976).

TABLE I
Distribution of the samples by age and sex

	New York (n=445)		*London (n=396)*	
Age	*Males (%)*	*Females (%)*	*Males (%)*	*Females (%)*
65–69	43	57	38	62
70–74	39	61	35	65
74–79	37	63	42	58
80–84	43	57	21	79
85–89	39	61	17	83
90+	18	82	17	83

Diagnostic comparison

A greater frequency of depression had been expected in New York because the high crime rate, the problems of health care, shortage of public housing, and less centrally organised welfare were likely to result in more unpleasant life events among the elderly in that city than in London. Whereas, apart from the possibility of a wider racial and genetic mix, there seemed little reason to assume that the level of dementia would vary between the two cities.

The findings did not fulfil these expectations. Table II shows the distribution of AGECAT diagnostic cases by sex. AGECAT finds almost twice as much organic disorder in the New York sample as in the London sample for both sexes ($P<0.02$), whereas the levels for depression and the neuroses are not significantly different. Table III sets the AGECAT diagnostic cases beside 'pervasive illness', psychiatrists', and the DSM-III (American Psychiatric Association, 1980) diagnoses made 'blind' by a psychiatrist (JRMC) reviewing the AGECAT symptom components. In the original study, Gurland *et al* (1983) defined 'pervasive' and 'limited' dementia and depression (the 'hand-applied' algorithm they provide has no relationship to the AGECAT method, which at this stage does not take social factors into consideration), and reported a point (past month) prevalence rate for 'pervasive depression' of 13% in New York and 12.4% in London, whereas the rates for 'pervasive dementia' were 4.9% in New York and 2.3% in London. Rates for 'limited dementia' were also higher in the New York sample.

TABLE II

Comparison of the samples by sex using AGECAT diagnostic cases (New York n=445, London n=396)

	Males		*Females*	
	New York (%)	*London (%)*	*New York (%)*	*London (%)*
Depression	13.0	13.1	18.3	22.8
Mania	0.0	0.0	0.4	0.0
Organic illness	5.7	2.2	10.1	5.4
Neuroses	1.7	1.5	1.1	2.3
Total ill	20.4	16.8	29.9	30.5

Table III also shows the comparison between New York and London for types of depressive illness. AGECAT at this stage splits the depressive category into neurosis (mental state similar to 'reactive') and psychosis (mental state similar to 'endogenous'), which is here a qualitative distinction. DSM-III uses a mixed method, partly qualitative in the distinction between major affective disorders and dysthymic disorders, and partly aetiological in the removal of bereavement from the depressive rubric. AGECAT separates bereavement and other depressive states of supposed separate aetiology on the basis of HAS data, not available in this study. DSM-III diagnoses are only available for the London sample. There is similarity in the proportions of AGECAT depressive psychosis (3.3%) and DSM-III 'major affective disorder' (4.6%). The proportion of 12.4 for 'pervasive depression' is similar to that for all types of DSM-III 'depression' taken together. Case levels for AGECAT depressive neurosis and DSM-III dysthymic disorder are bound to cause difficulties, as such conditions presumably lie on a continuum with normality.

As expected, AGECAT, identifying at the syndrome case level, selects the largest number of cases, but nevertheless agrees closely with the London interviewing psychiatrists. The proportions for 'pervasive' and DSM-III illness also agree closely; both methods are more restrictive than AGECAT and the psychiatrists.

Organic diagnosis

Table IV shows the distribution of AGECAT diagnostic organic states by levels of confidence. As explained above,

TABLE III

Comparison of the samples using AGECAT, pervasive illness, psychiatrists' and DSM-III diagnoses where available

	New York (n=445)		*London (n=396)*			
	Pervasive illness (%)	*AGECAT diagnostic cases (%)*	*AGECAT diagnostic cases (%)*	*Pervasive illness (%)*	*Interviewing psychiatrists (%)*	*DSM-III (%)*
Depression (all forms)	13.0	16.2^1	19.4^2	12.4	17.2	12.7^3
Mania	—^{4}	0.2	0.0	—^{4}	0.0	—^{5}
Organic illness	4.9	8.3*	4.3	2.3	4.3	2.8
Neuroses	—^{4}	1.4	2.0	—^{4}	4.1	—^{5}
Alcoholism	—^{4}	—^{4}	—^{4}	—^{4}	1.3	—^{5}
Total ill	—^{4}	26.1	25.8	—^{4}	27.2	—^{5}

1. Depressive psychosis 1.8, depressive neurosis 14.4.
2. Depressive psychosis 3.3, depressive neurosis 16.2.
3. Major depression 4.6, dysthymic disorder 6.3, bereavement 1.8.
4. Not applicable.
5. Not available.

*$P<0.02$.

TABLE IV
Distribution of AGECAT diagnostic organic syndromes (sub-case and case) by levels of confidence of diagnosis; numbers New York 63 (14.2%), London 25 (6.3%)

	Sub-case		*Case*		
	O1	*O2*	*O3*	*O4*	*O5*
New York: % of full sample ($n=445$)	0.5	5.4	4.1	4.3	0.0
London: % of full sample ($n=396$)	0.3	1.8	2.8	1.5	0.0

it is possible for a subject to achieve confidence levels of 3 and above (case levels) on more than one diagnostic cluster. For example, a subject may achieve both organic and depression syndrome case levels, but emerge finally as a diagnostic case of depression. Psychiatrists might call such a case 'pseudo-dementia', where the organic syndrome would be regarded as secondary to the depression. Such secondary organic syndromes are not included in this table. 'Diagnoses' also refers not only to cases (levels O3 to O5), but also to sub-case levels (O1 and O2). Table IV shows that a preponderance of organic 'diagnoses' in New York is not confined to case levels 3 and above. Such preponderence is evident at all levels of confidence except 5.

There are two principal types of diagreement between AGECAT and psychiatrists' diagnoses. One is the disagreement on category; that is, AGECAT designating a case as organic but the psychiatrists diagnosing it as depression. The other is where AGECAT agrees with the psychiatrists on the diagnosis, such as an organic syndrome, but disagrees on whether or not the subject is a case. Clearly, psychiatrists can vary in their concept of case levels and therefore, in simple case/non-case decisions, prevalence figures can differ widely. One of the merits of AGECAT lies in its consistency both in choosing diagnostic categories and in choosing case and sub-case levels. Sub-case levels of 2 would probably contain subjects whom some psychiatrists would recognise as cases and others not. Similarly, some AGECAT cases at level 3 might not be recognised by some psychiatrists. In practice, AGECAT appears to agree with most psychiatrists on its selection of cases, but allows investigators to record the proportions of subjects at each confidence level and thus to determine which choice of levels best suits their study. Studies are now being undertaken to determine the outcome of AGECAT sub-case levels 1 and 2, and whether or not future cases can be identified among them.

Figure 1 shows the distribution of AGECAT diagnostic syndrome cases of organic disorder by age in half-decades for the two cities. There is a greater or lesser preponderence of organic cases in New York at all six age periods. AGECAT cases are shown as a percentage of the sample at that age for each city. Proportions of community-dwelling subjects aged 80 or more suffering from dementia were 21.2% in New York and 15.7% in London. After age 85 numbers were comparatively small even for these samples, so that less reliance can be placed on the accuracy of findings at these age periods. The increasing proportions of dementia with age are statistically significant in the two cities (London $P<0.05$, New York $P<0.02$).

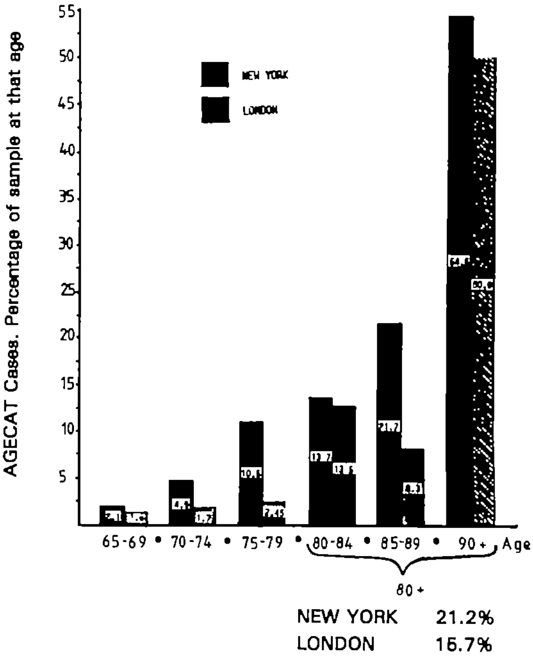

FIG. 1 Distribution of AGECAT diagnostic organic syndrome cases by age.

Depressive diagnosis

Where the distinction between case and non-case is drawn usually rests on the arbitrary judgement of the psychiatrist. AGECAT tries to simplify this problem by distributing subjects on case and sub-case levels (Table V). Here there is comparatively little overall difference between the proportions of these cases for the depressive confidence levels, across both cities. The tendency for there to be more depressive psychosis in London than in New York is seen to be mainly at the higher level (DP4) (not significant).

Figure 2 shows the distribution of diagnostic syndrome cases of depression by age. Like the previous figure for organic diagnostic syndromes, it excludes sub-case levels (D1 and D2). Taken overall, differences between the two cities are not striking and there is no consistent preponderance of depression cases in any one city. Again, findings for those aged over 85 are less certain because of small numbers than for the other age groups. For subjects aged 80 years and over the proportion of AGECAT diagnostic syndrome cases of depression is similar in both cities: New York 15.3%, London 14.3%.

TABLE V

Distribution of AGECAT diagnostic depressive syndromes (sub-case and case) by levels of confidence of diagnosis; numbers New York 135 (30.3%), London 129 (32.6%)

	Sub-case		*Case*				
			Depressive neurosis		*Depressive psychosis*		
	D1	*D2*	*DN3*	*DN4*	*DP3*	*DP4*	*DP5*
New York: % of full sample ($n=445$)	5.8	8.3	11.7	2.7	1.1	0.7	0.0
London: % of full sample ($n=396$)	5.1	8.1	14.4	1.8	1.3	2.0	0.0

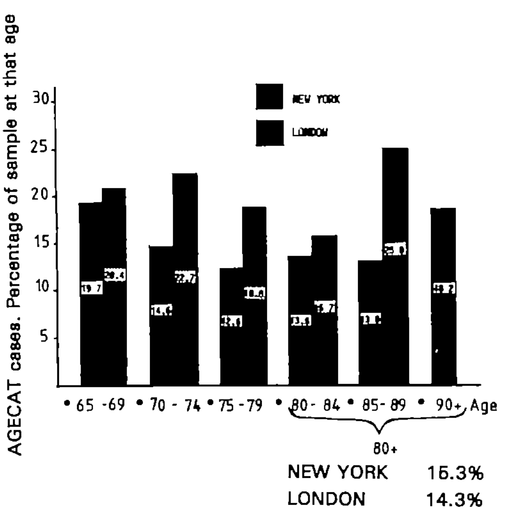

FIG. 2 Distribution of AGECAT diagnostic depression syndrome cases by age.

TABLE VI

Distribution of AGECAT diagnostic neurotic syndromes (case and sub-case) by levels of confidence of diagnosis; numbers New York 87 (19.6%), London 120 (30.3%)

	Hypochondriacal neurosis			
	Sub-case		*Case*	
	HC1	*HC2*	*HC3*	*HC4*
New York: % of full sample ($n=445$)	0.2	0.0	0.7	0.0
London: % of full sample ($n=396$)	0.0	0.0	0.5	0.0

	Obsessional neurosis			
	Sub-case		*Case*	
	OB1	*OB2*	*OB3*	*OB4*
New York: % of full sample ($n=445$)	0.2	0.5	0.0	0.0
London: % of full sample ($n=396$)	5.3	4.0	0.3	0.3

	Phobic neurosis			
	Sub-case		*Case*	
	PH1	*PH2*	*PH3*	*PH4*
New York: % of full sample ($n=445$)	0.0	1.4	0.0	0.0
London: % of full sample ($n=396$)	0.0	1.8	0.0	0.0

	Anxiety neurosis				
	Sub-case		*Case*		
	AN1	*AN2*	*AN3*	*AN4*	*AN5*
New York: % of full sample ($n=445$)	0.7	15.3	0.2	0.5	0.0
London: % of full sample ($n=396$)	1.5	15.7	0.8	0.3	0.0

Neurotic diagnosis

Table VI shows AGECAT diagnostic syndrome cases and sub-cases of neurotic disorders in the New York and London samples. If the symptoms of neurotic illness also lie on a continuum with normal behaviour, so that the differences between cases and non-cases are primarily quantitative, it will be difficult to draw the distinction. AGECAT finds remarkably few cases of pure neurosis in the total sample of 841 subjects in both cities. However, the interviewing psychiatrists themselves found only 4.1% of neuroses in London. Table VI shows that where the level for case identification is drawn will make a considerable difference in prevalence rates, at least for anxiety and obsessional neurosis. Case and sub-case levels of obsessional neurosis taken together are strikingly more common in London than in New York ($P<0.001$).

Figure 3 shows the distribution of AGECAT diagnostic syndrome cases and sub-cases of neurotic disorders by age for the two cities. The proportions of cases of neurosis are so small that they are shown here added to the sub-cases. If the division between cases and sub-cases is quantitative, the distribution of cases would be expected to follow that of sub-cases, although in Fig. 3 the proportion of cases to sub-cases is not constant between the age groups. The

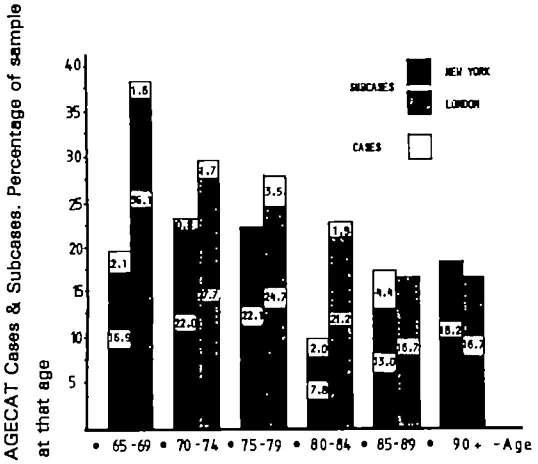

FIG. 3 Distribution of AGECAT diagnostic neurotic syndrome cases and sub-cases (levels 1 and 2) by age.

consistent tendency for there to be a higher proportion of sub-cases in London than in New York is demonstrated ($P<0.001$), although this is not necessarily reflected in case levels. There is also a tendency (not significant), at least in London, for sub-case levels to decline with age, which is not mirrored by the proportions of cases until after age 85, when findings are less certain because of smaller sample numbers.

The GMS symptom components were compared to reveal those significantly more common in one city than the other, even when allowance was made for the large number examined. Consistent with the other findings, the symptoms more common in London ($P<0.05$) reflect neurotic problems ('free-floating anxiety', 'tension', 'loss of interest', 'irritability', 'persistent thoughts') and other obsessional symptoms ('checks, washes repeatedly, excessively and cannot resist'), while those in New York were organic in type, concerning memory performance ('interviewer's name wrong on the first and second occasion', 'disorientation in place' and 'gives incorrect or incomplete address') ($P<0.05$).

Discussion

Levels of organic illness

The higher proportions of organic illness in New York are confirmed by AGECAT comparisons. These higher proportions are evident at all case and sub-case levels, for both sexes and across all age groups (half-decades) 65 and over. We have discussed possible explanations for this surprise finding elsewhere (Gurland *et al*, 1983; Copeland & Gurland, 1985). In short, a number of possibilities have been examined and rejected. When all five random sub-samples in each city were examined, the mean 'rational score' for dementia (Gurland *et al*, 1983) was higher in each New York sub-sample than for any London sub-sample except one, where the proportions were the same. A one-year follow-up study conducted in both cities (response rate in New York 72%, in London 63%) showed that the ratio for the diagnoses of dementia, assessed as 'pervasive dementia', between New York and London remained the same in both direction and magnitude, even when response rate at follow-up varied between sub-samples. Follow-up also tended to confirm the initial diagnosis. All nine cases in London diagnosed by the psychiatrists as dementia and followed up (the interviewer had no knowledge of the initial diagnosis) were confirmed as dementia one year later by the research psychiatrists. One-third of all those cases diagnosed as dementia in New York had died by the time of the one-year follow-up, compared with none in London, thus making it unlikely that the higher prevalence in New York resulted from longer length of illness, or earlier death in London. Examination using latent class analysis to classify dementia showed that the proportion of new cases arising in the sample during the year of the follow-up was higher in New York (2.4%) than in London (0.4%). A similar proportion of demented subjects was found to have entered institutions during the one-year follow-up in both cities (New York 9.5%, London 11.1%). When the populations were adjusted for age, sex, race and country of origin, non-white and foreign-born, the diagnostic proportions were unchanged.

There was some correlation between depression and dementia in New York. AGECAT is helpful here as it records separately each syndrome confidence level. The first possibility is that organic cases were under-represented in London because that proportion associated with depressive symptoms was incorrectly diagnosed as depression. However, there are no depressed cases in the London samples which are also syndrome organic cases. There are five associated with level O2. Three of these were followed up at one year. One had, indeed, become a case of dementia, moving to level O3 while the depressive levels had weakened, but of the other two, one was fully recovered and the other had become a pure case of depression. There would, therefore, seem little evidence to support this view.

The alternative possibility is that organic cases in New York were over-represented because depressive pseudo-dementias were misdiagnosed as organic. There are five organic cases associated with syndrome levels of depression, all but one of neurotic depression. Unfortunately, follow-up data are not available for these subjects, but if all five were assumed to be misdiagnosed it would still leave 32 organic cases in New York to 17 in London.

The possibility that the higher proportion of organic cases in New York was due to higher proportions of multi-infarct dementia seems unlikely, as the proportion of subjects with either history or evidence of a stroke was identical in both cities. The possibility that hospitals, nursing homes or social service accommodation might admit higher proportions of dementing clients in London also seemed unlikely, partly because of the very small proportion of the elderly population in such facilities, and because a subsequent study comparing psychiatric diagnoses in a random sample of New York and London hospitals, nursing and other homes (Gurland *et al*, 1979) showed the proportions of organic illness to be higher in New York institutions.

The finding of more dementing illness in New York among the elderly requires replication. So far we can advance no adequate explanation for it, although tentative suggestions have ranged from the high consumption of contaminated alcohol during the years of prohibition to possible dietary deficiency among the high immigrant New York population. We found no relationship between race and dementia in the general analysis. If genetic factors can be excluded and the difference upheld, these findings would seem to go some way towards supporting a contributory environmental cause for dementia.

Levels of depressive illness

It is also of interest that the expectation of more depression and neurotic illness in New York was not borne out. Depression seems no more common in the elderly than in the young. The psychiatrists' figure of 17% in London mirrors the 16% found by the same project in an earlier study in the old London borough of Camberwell, in collaboration with Brown & Harris (1978), using the Present State Examination in a population of younger women (aged 19–65). However, this overall figure for those aged 65 and more conceals higher proportions for both sexes before age 70, followed by a gradual decline for women to the tenth decade. The initial fall for men occurs between 65 and 74, with a steady increase in depression from then onwards. However, once again, findings are less certain in these upper age groups where numbers in the sample are small. It should be noted that although levels of crime against individuals were shown to be higher in New York than in London, complaints of fear of crime and of general neighbourhood deterioration were similar in both cities. Actual differences did not, therefore, seem to reflect their perception. Levels of physical illness, disability and dependence were similar in both cities. We have suggested (Copeland, 1981*b*) that unpleasant life events which have a causal role in depression may have to be perceived not only as painful and unpleasant but also as inevitable and unchangeable, representing for the subject an unpleasant stress before which he or she feels helpless. It seemed likely that the elderly of this generation, although they perceived many life events as unchangeable, did not necessarily perceive them as painful or unpleasant. To be perceived as painful, social events or conditions may have to fall short of expectations. Although living appeared to the interviewers to be much more difficult and stressful in New York, and this was amply borne out by the evidence, it probably did not fall short of expectation, especially for this largely immigrant population. Certainly there were instances in London when the interviewees expressed gratitude for their social conditions, recollecting how their parents had suffered at their age, even though to the interviewers, social standards were unsatisfactorily low.

We have already recorded (Gurland *et al*, 1983) that only 3% of those with 'pervasive depression' in New York and 14% in London were receiving anti-depressant medication. Just as disturbing was the high proportion of benzodiazapines prescribed, often in the opinion of the interviewers inappropriately, without concurrent anti-depressant therapy (31% of depressed subjects in New York and 18% in London). In New York, 93% of the depressed and 81% of the total sample had seen a doctor in the previous year before interview, and 82% and 74% in London.

Levels of neurotic illness

The low proportions of neurosis in the two cities, both on psychiatrists' diagnosis in London and on all AGECAT levels, was a surprise finding. If the level for anxiety neurosis is lowered to between AGECAT levels 1 and 2 rather than between levels 2 and 3, the proportion rises to over 16% in both cities. Nevertheless, there are very few subjects with moderate or severe anxiety which is not also associated with other mental illness. The preponderance of obsessional symptoms in London, particularly at the sub-case level, is presumably a cultural difference in the subjects. If, as is sometimes suggested, obsessional symptoms reflect states of insecurity, then we might have expected greater insecurity among the immigrant population of New York. It may be argued that AGECAT case levels for neurosis are pitched too high, but in replication samples, the proportions of neurotic cases identified by AGECAT have been similar to those identified by the psychiatrists. As pointed out earlier, psychiatrists are not skilled in distinguishing between cases and non-cases. A much earlier study (Copeland *et al*, 1975) showed that psychiatrists tended to rate lower levels of psychiatric pathology than general practitioners when viewing the same video-tapes of psychiatric patients. It was suggested at that time that psychiatrists would tend to rate examples of mental illness 'down' in relation to their experience of more severe illness, while those without such experience would tend to rate symptoms 'up', against their concept of normal behaviour. Such rating practices might lead psychiatrists to identify fewer cases of neurosis than general practitioners. A study comparing AGECAT diagnoses of neurotic disorders against general practitioner diagnoses could provide further information on the way mental illness in the elderly is perceived by different groups of doctors. Nevertheless, apart from anxiety and depressive neurosis, and, possibly in London, obsessional neurosis, other forms of neurotic illness appear to be rare in the elderly at any level of diagnostic confidence.

We have attempted to demonstrate the use of the AGECAT computerised diagnostic system for comparing mental illness in two samples of elderly subjects. We believe it is helpful to have a standardised method of diagnosis which can be applied reliably and consistently to different populations, which allows quantification of the levels of confidence in diagnosis, and especially identifies and grades sub-case levels.

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Detention of 'Psychopathic Disorder' Patients in Special Hospitals

Critical Issues

A. T. GROUNDS

The detention of offenders in the legal category 'psychopathic disorder' in special hospitals for treatment raises a number of critical issues. There are doubts about the nature of the disorder; what constitutes treatment; who is 'treatable'; the effectiveness of treatment; and whether evidence of psychological change implies reduced risk of reoffending. In view of these uncertainties, it is argued that indeterminate hospital orders may provide an unrealistic and unjust legal framework for treating 'psychopaths' in special hospitals, and the use of powers under the Mental Health Act to transfer such patients to hospital during the course of prison sentences is a more appropriate alternative. This provision could be used more frequently, subject to improved safeguards of the right of release at the expiry of sentence.

In general penal policy during recent decades there has been a growing disillusion with rehabilitation and treatment as objectives of sentencing, firstly because of doubts about their effectiveness (Martinson, 1974; Brody, 1976), and secondly because they are held to be unjust (Lewis, C. S., 1953; von Hirsch, 1976; Bowden, 1985). Nevertheless, a small number (under 30 a year) of offenders in the Mental Health Act category 'psychopathic disorder' continue to be sentenced by restricted hospital orders and admitted to special hospitals. They represent a tiny minority – less than 1 in 2000 – of those sentenced in the criminal courts for violent and sexual offences. A current approach to the assessment and treatment of 'psychopaths' in one of the special hospitals has been described by Grounds *et al* (1987). Medical recommendations for their admission under restricted hospital orders may rest on an implicit belief that indeterminate sentencing for psychiatric treatment is appropriate for such individuals, and that treatment can reduce the risk of reoffending. When this view is set against the current climate of opinion in criminology and penal policy it looks anachronistic: it maintains the optimistic spirit of the East & Hubert (1939) report in its assumption that psychotherapy may be effective in reducing the risk of future antisocial behaviour in selected cases.

This paper will consider six critical questions concerning the detention of 'psychopathic' offenders in hospital for treatment: Do such offenders have a definite mental disorder? What constitutes treatment? Who is 'treatable'? Is treatment effective? Does psychological change imply reduced risk of reoffending? Is indeterminate sentencing for treatment a fair exchange for sentencing based on principles of criminal justice?

The category of 'psychopathic disorder'

The legal term 'psychopathic disorder' in the 1983 Mental Health Act is defined as "a persistent disorder or disability of mind (whether or not including significant impairment of intelligence) which results in abnormally aggressive or seriously irresponsible conduct on the part of the person concerned". The troubled historical origins of the legal definition and of the underlying clinical concepts – "semantic variations on a dubious theme" – are documented by Walker & McCabe (1973) and Lewis (1974). Walker & McCabe's (1973) examination of how the legal term is used led them to conclude that as a 'diagnosis' 'psychopathic disorder' had no explanatory, descriptive, prognostic or therapeutic function. In practice it is used as a pseudo-diagnosis; a label attached to offenders with a variety of abnormalities to help the psychiatrist "get his patient through the customs-barrier of the courts if he wants to." The Butler committee (Home Office, 1975) also concluded that: "The class of persons to whom the term 'psychopathic disorder' relates is not a single category identifiable by any medical, biological or psychological criteria"; and studies of 'psychopathic disorder' patients in special hospitals (Blackburn, 1982) confirm the variety of clinical features subsumed under this term. The category of 'psychopathic disorder' "remains ill-defined, circular and does not embrace a homogeneous group of individuals" (Blackburn, 1983).

It is therefore a shaky legal foundation on which to select some offenders for diversion from the penal system into indeterminate hospital care.

What constitutes treatment?

There is little consensus about what constitutes treatment for 'psychopaths'. The interpretation of the term 'medical treatment' in the Mental Health Act is general: "'medical treatment' includes nursing, and also includes care, habilitation and rehabilitation under medical supervision". Traditionally in special hospitals the equally non-specific term 'milieu therapy' has been described as treatment (McGrath, 1966; Royal College of Psychiatrists, 1983); but as Gunn (1978) has pointed out: "it is difficult to argue that in hospitals of any kind a structured disciplined environment is a *medical* strategy; it is simply a prerequisite for making sure that patients stay where they are told to stay." McGrath (1968) also questioned to what extent "a benevolent custodial and expectant regime" could be justified as 'treatment'.

If treatment is defined in more specific terms, what counts as adequate treatment for 'psychopaths' will vary in different contexts. In an out-patient setting it may be justifiable to regard measures such as crisis intervention, and long term support without expectation of substantial personality change, as appropriate psychiatric treatment; but in a hospital setting where 'psychopaths' are detained for an indefinite period in maximum security this is not sufficient. Treatment is expected to produce personality change: the Home Office seeks evidence of this before agreeing to release, and for the offender too this is the promise implied by a hospital order. The therapeutic aims therefore have to be higher; and if they cannot be achieved the patient is being deceived.

'Treatability'

In the 1959 Mental Health Act, the term 'psychopathic disorder' included the clause that the disorder "requires or is susceptible to medical treatment." In the 1983 Mental Health Act the 'treatability' clause was changed and made a condition that had to be met for a long term treatment order to be made. For this purpose it must be held that medical treatment for the patient "is likely to alleviate or prevent a deterioration of his condition".

Medical recommendations for hospital orders under the 1959 Act included a section filled in by the reporting doctors, headed "Information to establish that the disorder or disability of mind requires or is susceptible to medical treatment". The specific items cited in the preadmission medical reports of the 'psychopathic disorder' patients ($n = 13$) in one ward in Broadmoor Hospital on 1 March 1986 were examined, and two main features were evident: first the wide variety of items mentioned, and secondly their lack of specificity. Some reports merely asserted that treatment was required, without reasons being given. Factors most frequently cited as evidence that the patient's disorder was susceptible to treatment were, first, the patient's self-concern or wish to have treatment; and secondly, being of a young age.

Even the fullest reports were expressed in general terms. For example: "He is a young man of good intelligence who is potentially capable of benefiting from psychotherapy and other forms of psychological treatment. He is anxious to have treatment". Others were more half-hearted: "In view of his young age it is worth trying treatment and the consultant at Broadmoor Hospital is prepared to undertake his treatment". The contents of the reports suggest there was little agreement about specific factors that indicate a likelihood of benefit from treatment, and they do not indicate what specifically distinguished this tiny group of serious offenders from others.

Lewis (1974) noted that the most obvious criticism of the legal definition of 'psychopathic disorder' centered on the 'treatability' clause. "The opinion of psychiatrists or other competent experts as to who requires or is susceptible to treatment will inevitably tend to be subjective and inconsistent." In an earlier paper, when discussing the role of medical diagnosis in a social context, he argued that, "Unless the criteria of ill health are independent and clear it is difficult or unsafe to use data based on them for purposes of selection" (Lewis, A., 1953). The force of this conclusion was ignored by the Butler committee (Home Office, 1975) and others since (e.g. Ashworth & Gostin, 1984) who, having recognised the uncertainty about what constitutes 'treatability', have nevertheless proceeded to argue that the legal criterion should be tightened up even further, so that doctors would be required to make more specific judgements and predictions about this issue. The proposal is self-contradictory and impracticable.

These difficulties are unlikely to be fundamentally altered by the use of the new Interim Hospital Orders (S.38, Mental Health Act 1983), but it is arguable that they may be useful in enabling prospective patients and hospital staff to make more realistic and informed assessments of whether a treatment alliance is possible than could be done prior to the 1983 Act.

Effectiveness of treatment

The general conclusions drawn from surveys of psychotherapy outcome research (Bergin & Lambert, 1978; Prioleau *et al*, 1983) and research on the effectiveness of corrective and rehabilitation programmes for offenders (Martinson, 1974; Brody, 1976) are gloomy. The available follow-up studies of special hospital patients do not enable us to relate outcome to specific treatment received (Black, 1984); however, such studies do indicate that patients in the category of 'psychopathic disorder' have a poorer outcome in terms of reconvictions than the mentally ill, and a history of prior criminal convictions is the factor most strongly associated with reconviction after release from special hospitals (Bowden, 1981; Black, 1982; Tennent & Way, 1984). These results suggest that, as is the case for offenders in the penal system, it is the pre-admission criminal profile that is the best predictor of reoffending; and it seems unlikely that the effect of hospital treatment is in general sufficient to offset its influence. Thus there is little empirical basis for confidence that, in detaining 'psychopaths' for treatment in special hospitals with the aim of reducing likelihood of reoffending, there is evidence to back up our therapeutic hopes and aspirations.

Does psychological change imply reduced risk?

The next issue is related. If, following treatment, some 'psychopaths' do show evidence of psychological and personality change, does such change imply that the patient's risk of reoffending is reduced? Medical recommendations to the Home Office or to mental health review tribunals for discharge or transfer have to be framed as if such an inference was valid, but there is currently a lack of any detailed empirical study confirming whether this is the case. The evidence from follow-up studies so far is not encouraging. If pre-admission variables such as previous convictions are the best predictors of reoffending, as is case for offenders in penal settings, there has to be doubt about the validity of inferring reduced likelihood of dangerous behaviour from evidence of personality or behavioural change in hospital.

Treatment instead of justice – a fair exchange?

The doubts surrounding the above issues lead to the last question. Is the admission of a 'psychopathic' offender to hospital on an indeterminate treatment order a fair exchange for a sentence determined by the sentencing court on criminal justice principles? The effect of psychiatric involvement leading to a hospital order is to remove the offender's right to have the length of time he spends in custody determined by the court on the principle of proportionality. Apart from the possibility of discharge by mental health review tribunals, discretion over the release of restricted patients lies with the executive, in the form of the Home Secretary, rather than the courts. Furthermore, tribunals have to base their decisions on clinical criteria, and cannot take tariff considerations into account. In general sentencing policy there is a weight of legal opinion opposed to the principle of indeterminate sentencing with release dependent upon the progress of the offender. Scarman (1978), for example, has argued that the determination of how long an offender remains in custody should be a matter for the sentencing court rather than the executive, in the absence of evidence that rehabilitation as a penal policy is effective. The balance of justice between the offender and the public "is best struck in open court". The indeterminate hospital order, under which continued detention depends on clinical criteria, operates on quite different principles. But in the case of 'psychopaths', where are the specific clinical features that justify treating this tiny group of offenders differently from the many thousands of others passing through the criminal justice system each year? The question can be answered more easily in the case of the mentally ill and handicapped than in the case of the 'psychopath'.

The admission of 'psychopaths' on indeterminate treatment orders cannot be validly justified on the sole basis that special hospitals provide more humane environments of care than prisons, or that hospital-based research offers the prospect of major breakthroughs in understanding and treatment. The reality of indefinite detention in conditions of maximum security is not so benign. Critics such as Goffman (1961), describing the destructive social dynamics of psychiatric institutions; Kittrie (1971), with his legal critique of "therapeutic tyranny"; and earlier still C. S. Lewis (1953), in his dramatic argument against the "humanitarian theory of punishment", all perhaps overstated their cases, but they have left a legacy of insights that are too incisive and unsettling to be easily dismissed.

These considerations should not be taken to imply that psychiatric help should not be offered to personality-disordered offenders. Patients in the legal category 'psychopathic disorder' have psychopathology from which they suffer (Craft, 1966; Blackburn, 1975, 1982); further descriptive research is required (Lewis, 1974); and psychiatric care and treatment should continue to be offered (Gunn, 1978). The question is, in the light of current knowledge about treatment and its effectiveness, what is the most realistic and just legal framework within which to carry out these tasks? It would seem preferable to find a way of ensuring that those who require care and treatment receive it but without forfeiting their rights to sentencing according to normal criminal justice considerations. The medical and penal issues need to be separated. This would also protect doctors from having unrealistic roles placed upon them as "medical underwriters of preventive detention" (Chiswick, 1982), with the attendant responsibilities of making judgements about future dangerousness and duration of custody: responsibilities which involve carrying the mantle of the sentencing court. Gunn (1978) has argued that compulsory indeterminate treatment for 'psychopaths' in special hospitals is "not entirely ideal because the same person has to act as custodian and physician in an area where there is doubt as to whether a medical disorder is present and where the criteria for custody may be quite different from the criteria for psychiatric treatment and response". Dell & Robertson (1986) reached similar conclusions in their comparative study of treatment and discharge decisions about mentally ill and 'psychopathic disorder' patients in Broadmoor Hospital.

Transfer to hospital during sentence

One way of enabling psychiatric treatment within the framework of normal sentencing is available under the Mental Health Act provision to transfer prisoners to hospital for treatment during sentence (S.47 of 1983 Act). This allows a proper separation of psychiatric and criminal justice considerations in dealing with such offenders, and Mawson (1983) has argued that it is a preferable option. Until recent years only a minority of 'psychopaths' have been transferred to special hospitals by this means, and most have been admitted under hospital orders (Grounds *et al*, 1987).

However, current legal provisions for the transfer of mentally disordered sentenced prisoners, although preferable, are not ideal from a criminal justice point of view. Those transferred during the course of determinate sentences can remain detained under 'notional hospital orders' at the end of sentence without any formal re-examination and renewal of detention at that time. Most of the 'psychopaths' who were transferred during sentence to Broadmoor under the 1959 Mental Health Act eventually left Broadmoor by being discharged to the community or transferred to local hospitals, but the majority of these cases were detained in Broadmoor beyond the expiry date of their original sentences before being released. Recent proposals by a Home Office & DHSS (1986) working party on 'psychopathic offenders' did not address this issue, and were motivated primarily by a concern with how to specify minimum rather than maximum periods of detention before discharge.

There is a case either for a change in clinical practice or a change in the law to ensure that sentenced prisoners given determinate sentences and transferred to hospital for treatment should normally be entitled to release at the expiry of their sentences. If continuing treatment in conditions of maximum security is required beyond this time, this should be carried out by means of making a new civil order under the Mental Health Act (S.3). This would require a new application and two medical recommendations, and would safeguard against passive continuation of detention under a 'notional hospital order' without recertification as occurs at present. Such a reform was advocated by Gostin (1977) but not incorporated into the new Mental Health Act, although it was agreed that, in the case of restricted patients, Home Office restrictions on discharge should be lifted at the "earliest date of release" (which allows for remission) rather than the "latest date of release".

Discussion

Psychiatric help and care should continue to be offered to serious offenders with personality disorders. The question raised in this paper is what is the most appropriate, realistic and just legal framework for doing so. Provisions in the Mental Health Act to transfer 'psychopathic disorder' offenders to hospital during the course of prison sentences are preferable to the use of indeterminate hospital orders, subject to improved safeguards of the right of release at the end of sentence, which are necessary from a criminal justice point of view. Psychiatric help for such offenders needs to be available, but without involving doctors in an inappropriate role and without the offenders themselves losing the right to sentencing according to 'just deserts' principles. As C. S. Lewis (1953) argued, the consequences can be inhumane if we seek to *substitute* mercy for justice. "Mercy detached from justice grows unmerciful, that is the important paradox."

In the case of 'psychopathic disorder', if there was evidence, first, that we were dealing with a well-defined disorder; secondly, that we had specific treatments for it; thirdly, that we knew who was likely to benefit from treatment; fourthly, that such treatments were effective in producing psychological change; and fifthly, that psychological change implied a reduced risk of dangerous behaviour; then there would be justification for preferring to deal with such offenders by indeterminate treatment orders rather than tariff sentencing. But the evidence at each step is lacking and is insufficient to justify the practice of recommending hospital orders for 'psychopaths'. The view that this practice is appropriate continues to be held within the English special hospital system, but it has to be questioned to what extent the beliefs that sustain this practice can be empirically supported.

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The Dismantling of the Mental Hospital?

Glenside Hospital Surveys 1960–1985

M. FORD, C. GODDARD and R. LANSDALL-WELFARE

Results of the sixth quinquennial survey of the resident population of Glenside Hospital, Bristol, are reported. The total population continues to fall, but the rate of decline has slowed; the implications of this are discussed. Many patients live in an emotionally impoverished state, friendless and rarely leaving the hospital. Few in-patients are employed, even within the hospital. Considerable provision is made, however, for the employment of day-patients. To effectively resettle and support in the community those currently remaining in hospital will require increasingly extensive provision.

Every 5 years since 1960, the population of Glenside Hospital, Bristol has been reviewed with reference to in-patients of more than 3 months' duration, and their needs for accommodation and employment (Cooper & Early, 1961; Early & Magnus, 1966; Early & Nicolas, 1971, 1977, 1981).

All of the surveys have employed the technique originally developed by Cooper & Early (1961), in which a schedule of 34 items is completed for each patient resident for 3 months or more on the day of the survey. Necessary information for this purpose is obtained from the charge nurse, case-notes and central hospital records. For the 1985 survey an extra question was added in order to identify those patients who had been in-patients at the time of the 1960 survey, the 'Remnant'.

Results and comparison with previous surveys

Figure 1 and Table I show the length of stay of patients. On 1 January 1985 there were 368 patients (175 men, 193 women) resident in the hospital for more than 3 months. Over 25 years the population has fallen from 1012, a fall of 64%.

Between 1980 and 1985 there was a fall in the population hospitalised for more than 5 years and a levelling of the numbers in the 2–5 years group. Patients resident between 3 months and 2 years increased both in percentage and absolute terms.

The population aged progressively from 1960 to 1980 (Fig. 2). Between 1980 and 1985, however, the percentage in each of the four major groups remained much the same. In 1985, 46% of men and 65% of women were over 65 years.

The legal status of patients is shown in Table II. Between 1980 and 1985, the number and percentage of detained patients increased.

Employment data are given in Table III. The latest figures show unemployment affecting over 80% of the resident population. Only a handful were working outside the hospital, despite access to a sheltered workshop. There had been a dramatic fall in those working in the hospital Industrial Therapy Workshop and other hospital departments since 1975.

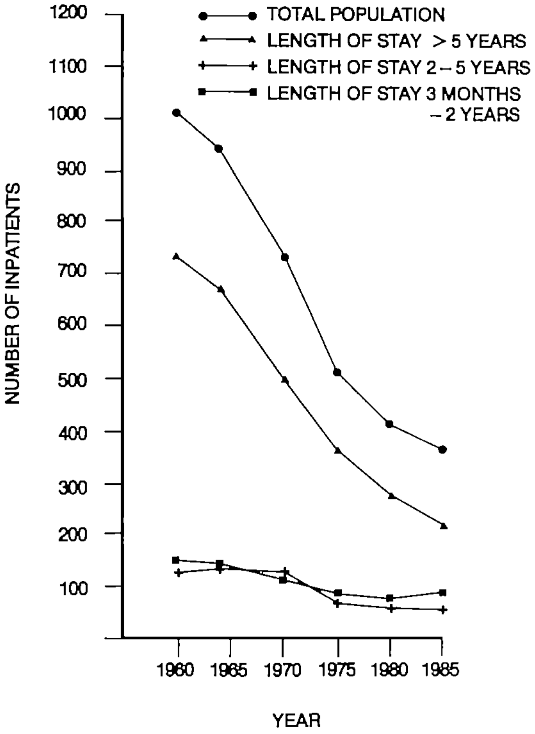

FIG. 1 Length of stay of patients in Glenside Hospital.

Information on contacts is given in Table IV. Between 1980 and 1985 there was no substantial change in the contacts that the patients had; 50% of the patients in

TABLE I
Duration of stay in Glenside Hospital: point prevalence on 1 January

	1960	*1964*	*1970*	*1975*	*1980*	*1985*
>5 years	737 (73%)	672	500	365	279 (67%)	221 (60%)
2–5 years	125 (12%)	136	126	64	59 (14%)	56 (15%)
3 months to 2 years	150 (15%)	140	113	86	78 (19%)	91 (25%)
<3 months	135	131	121	110	72	64

TABLE II
Legal status: patients detained

Section	*1960*	*1964*	*1970*	*1975*	*1980*	*1985*
26/3	52 (5%)	53 (6%)	41 (6%)	9 (2%)	12 (3%)	16 (4%)
60/37, 65/41, 72/47	— —	— —	— —	— —	5 (1%)	7 (2%)

TABLE III
Employment of patients

	1960 (n=1012)	*1964 (n=948)*	*1970 (n=739)*	*1975 (n=515)*	*1980 (n=416)*	*1985 (n=368)*
Working outside hospital	55	341	67	36	21	6
Working in hospital Industrial Therapy Department/Integrated Rehabilitation Workshop	224		178	107	53	12
Working in hospital department	361	283	218	100	87	5
Ward chores					48	42
Unemployed or occupational therapy	372	324	276	272	207	303

TABLE IV
Contacts of patients in hospital for different periods

	<2 years		*2–5 years*		*>5 years*	
	1980 (%)	*1985 (%)*	*1980 (%)*	*1985 (%)*	*1980 (%)*	*1985 (%)*
Goes out regularly	27	32	19	7	8	18
Goes out occasionally	14	14	19	13	14	8
Visited	43	33	42	54	28	24
Rarely or never visited	15	20	20	26	50	50

TABLE V
Nursing care required

	1960 (%)	*1964 (%)*	*1970 (%)*	*1975 (%)*	*1980 (%)*	*1985 (%)*
Nursing care not necessary	12	18	7	5	8	4
Minimal nursing care	35	25	29	23	23	22
Routine psychiatric nursing	53	58	64	66	52	52
Routine geriatric nursing					8	9
Special nursing					9	13

TABLE VI
Type of accommodation required

	1960	*1964*	*1970*	*1975*	*1980*	*1985*
Psychiatric hospital care	558 (55%)	562	380	277	254 (61%)	224 (61%)
Geriatric care	83 (8%)	73	71	46	48 (12%)	40 (11%)
After-care:	321 (32%)	259	270	185	97 (23%)	88 (24%)
supervised					85	79
unsupervised					12	9
No special arrangements	50 (5%)	54	18	7	17 (4%)	16 (4%)

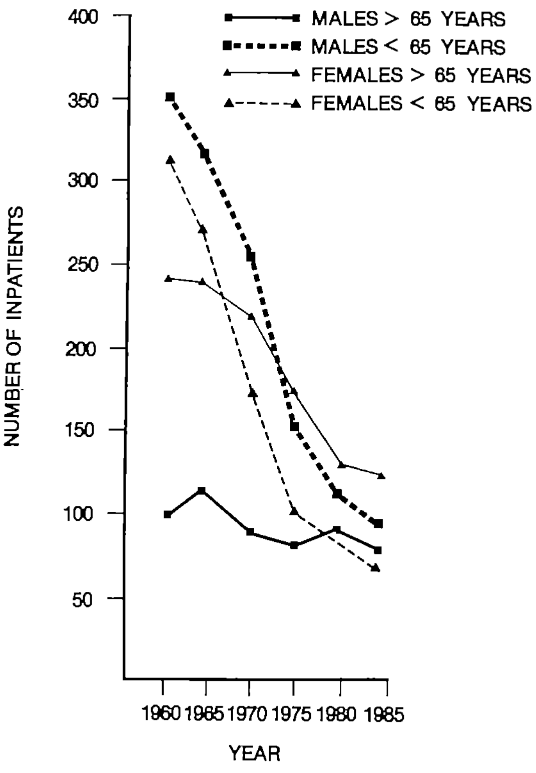

FIG. 2 Age of patients in Glenside Hospital.

hospital for more than 5 years in both 1980 and 1985 did not go out and were rarely visited.

In 1985 a significant proportion (26%) still required little or no nursing care (Table V). The proportion requiring routine geriatric care was 9%, similar to that in 1980.

In line with nursing requirements, accommodation requirements have changed little in percentage terms over the 25 years, although absolute numbers have fallen (Table VI). In 1985, 28% had no need for hospital care.

Discussion

Hospital population

Nationwide, over the period 1960 to 1985, the number of in-patients fell from 128 000 to 64 000 (General Register Office, 1964; DHSS, 1986), a fall of 50%. Glenside has been in line with or slightly in advance of this trend. However, the rate of decline of the total Glenside population appears to be slowing. Between 1960 and 1980 the decline averaged 3% per annum, and from 1980 to 1985 the fall was 2% per annum. These figures have implications for planning and are further evaluated under attrition rate.

The small increase in the number of detained patients may reflect the interests of the consultants and the absence of a medium-secure unit in Avon. (A medium-secure unit is planned for the Glenside site.) Detention is far less used than in 1960.

The age structure of the population remains much the same as in 1980. There continues to be an increase in the very elderly: in 1985, 30% were over 75. The main feature of the period 1975 to 1980 was an increase from 35% to 44% in men aged over 65. This has slowed considerably – 2% increase from 1980 to 1985. Since 1980 there has been a small rise in the under 45 age group: in 1985 it was 14% of the total.

The number in the group resident more than 5 years has fallen, but is still substantial. The diagnostic categories are little changed since 1980: the latest figures give 76% schizophrenic and 5% with senile dementia. This latter figure reflects the life expectancy of severely demented hospital patients who 'because of the high mortality rate . . . are resident for less than five years' (Wing, 1979). However, it should be noted that 10 of the 65 demented patients in our study had lived in hospital for more than 5 years.

In the 2–5 year group, the numbers in 1980 with a diagnosis of dementia or schizophrenia were approximately equal (21:20). The 1985 survey shows a marked change, 30 with dementia and 13 with schizophrenia, and there was no corresponding increase in any other diagnosis. This, if not a random change, could represent more active rehabilitation and discharge of schizophrenics.

The increase in the group resident from 3 months to 2 years is interesting. It is mainly explained by an increase in the number of patients with affective psychosis: from 12 in 1980 to 23 in 1985. There is also an increase from 0 to 6 in 'other psychoses', covering non-organic, non-schizophrenic and non-affective psychoses. These data could suggest a change in diagnostic habits rather than an increased incidence, or increased difficulty in rehabilitating these patients.

Further examination of the group resident for 3 months to 2 years revealed that two-thirds were women. This group was represented in all age brackets, 46 being under 65 years. A large number – 15 – were detained. When compared with the total resident population, a much larger percentage were or had been married. The majority – 56 – had previously been employed. Diagnostic categories were as follows: senile dementia 27, schizophrenia 22, affective psychosis 23, other psychoses 6, neurosis 3, personality disorder 5, and 5 with other diagnoses. They were more likely than other residents to be considered suitable for discharge to supervised and unsupervised accommodation, or to accommodation already available. However, only in 28 was no disturbance thought likely to ensue on discharge. Further, the chronicity of some of the group is indicated by the fact that eight of them had been in-patients in the original census of 1959. Thus, although some of this group will be discharged, they are also likely to be readmitted.

Unemployment

Unemployment is clearly and increasingly the norm. To a large extent, the 1985 figures reflect a redefinition of the role of the Industrial Therapy Department, which has now been renamed the Integrated Rehabilitation Workshops (IRW). It now offers a mixture of industrial training and occupational therapy. In-patients are clearly involved in the latter.

Increasing numbers of day-patients are attending and are considered for industrial training. Thus the IRW and the Bristol Industrial Training Organisation, situated off the hospital campus, are mainly community resources. If patients are capable of industrial training or sheltered work, then, with rare exceptions, they are living in the community. This is in contrast to 1960, when industrial training and sheltered workshops were considered the pathway to discharge for psychiatric hospital patients 'to hold their own in the community' (Cooper & Early, 1961).

By 1971, Early (1978) had commented: 'the continuation of economically viable industrial therapy in hospital will increasingly depend on the attendance of day patients.' Several major factors are operating: the ageing of the hospital population, the increasing level of general unemployment, and partly consequent on these, changing concepts of rehabilitation priorities. As a result, activities of daily living and socialisation are increasingly emphasised, and employment for the resident psychiatric in-patients is rarely an immediate goal. Also, to quote Bennett (1981), 'the appreciation that further long-stay hospital patients are being replaced by long-term patients in the community and the recognition of these disabled patients need for support together with medical and social aid, leads in turn to a reconsideration of the function of a psychiatric unit for disabled patients in a district service.'

Friendlessness

The number of patients who neither go out nor are visited remains scandalous. Factors which had a major bearing on this were male sex, marital status (only 51 were married), increasing age, and increasing duration of stay. Only 27 of 206 patients over 65 years of age went out, and of 131 patients in hospital over 20 years' duration, only 15 went out. Families 'forget' the patient; and 'society seals up its cracks against the mentally disabled' (Morgan & Cheadle, 1981).

Nursing levels are only rarely sufficient to organise trips out. The current state of affairs clearly underlines the implausibility of discharging long-term or elderly psychiatric hospital residents to a friendless world. There is a great need for an increase in staff levels and voluntary aid to re-establish contacts and interests outside the hospital.

Dementia and nursing care

The percentage of demented patients has increased only marginally; the 1985 figure is 18%. However, they have become less mobile and more dependent: the figures show 30 as permanently chairfast, 36 as considered suitable for psychiatric hospital care, and 32 for geriatric hospital care.

There may well be considerable national variation in the number of hospitalised demented patients. Levene *et al* (1985), in their study of all 1087 patients who were in psychiatric beds provided by a large district health authority, calculated that 'one third of the total . . . had a primary diagnosis of dementia.'

This inappropriate use of scarce psychiatric nursing experience continues to be a source of frustration and demoralisation to nursing staff already faced with considerable understaffing and difficulties in recruitment.

Mental handicap

There has been a continuing decline in the numbers with a primary diagnosis of mental handicap: 44 (9%) in 1975, 19 (5%) in 1980 and 5 (1%) in 1985. Of these five, four had no subsidiary diagnosis but had been hospitalised for over 20 years, and therefore were not considered for transfer to a mental handicap hospital. They are, nevertheless, inappropriately placed.

Accommodation

The numbers have fallen, but the pattern of demand for accommodation has changed little over the years. In 1985 there were still 28% who did not require hospital accommodation (37% in 1960). The major hurdle to discharge remains the lack of supervised after-care. Of the 79 patients felt to require supervised after-care, 49 had a diagnosis of schizophrenia, and 14 of affective disorder; 52 were under 65 years of age. Attempts by the hospital social work department to set up hostels under joint funding arrangements have failed, and projects from local social service departments and voluntary agencies have yet to show themselves able to take anything but a small proportion of the problem.

The characteristics of the patients in this survey suggest that they will require not just supervised accommodation but also the full range of day-care facilities. To quote Wing & Morris (1981): 'The very success of the rehabilitation programmes of the 1960's and 1970's has meant that the population left behind in hospital was increasingly handicapped', and resettlement required 'an extensive network of sheltered residential and day units'.

However, the contrast with 1960 remains clear, for the Glenside survey of that year illustrated 'the immediate need for aftercare accommodation for over 300 patients, many of whom have been in hospital continuously for several years and have only mild psychiatric symptoms' (Cooper & Early, 1961).

Moreover, there has been a considerable sum spent on residential accommodation and supportive services for the elderly mentally infirm outside of hospital. Additionally, there has been a recent mushrooming of private-sector accommodation for the elderly. Thus, while the percentage of Glenside patients with dementia has increased, this has only been a fraction of the total in the community. In other areas, as referred to earlier, the psychiatric hospital may be providing accommodation for a much larger proportion of the demented.

The Remnant

The Remnant is a group of 112 patients who in 1985 had been continuously in-patients since the first survey in 1960. These were compared with 38 patients who had been in-patients in 1960 but had had at least one intervening period out of hospital since.

Similarities between the groups include the predominance of schizophrenia, poor social participation, present unemployment, and siblings being the only potential outside support.

Differences include the predominance of males in the continuous group (67 men, 45 women) compared with an equal sex ratio in the discontinuous group. This could lend support to the evidence for a more favourable prognosis for women, which has been consistently noted in modern studies of schizophrenia (Watt & Szulecka, 1979; Watt *et al*, 1983; Hogarty *et al*, 1974).

Compared with the discontinuous group, the remnant were older, more likely to be unmarried, more obviously mentally abnormal, more likely to be considered of below average intelligence, and less likely to have contacts with the outside world.

Interestingly, there was an excess in the Remnant of those who had held regular semi-skilled or unskilled work prior to admission. As to those who had held skilled work, the proportions in each group were similar. In the discontinuous group, 25 of the 38 had been unemployed on admission, compared with only 45 of the 112 of the Remnant as a whole ($P=0.01$). We presume that this reflects the predominance of men in the Remnant, and the effects of previous psychiatric admission in the discontinuous group. It also points to the catastrophic nature of some schizophrenic illnesses, leading some people directly from employment to permanent hospital residence.

The Remnant therefore consists of nearly one-third of the hospital population. They are generally over 55 years old, male schizophrenics, with poor social functioning and few outside contacts. These severely handicapped people continue to live in impoverished ward environments with only minimal staff. They also suffer personal poverty, no longer being able to supplement their income from industrial therapy; they survive on a basic allowance which scarcely provides a packet of cigarettes a day. One could envisage that increased resources would improve their lives, but the prospects of them participating in the 'community' must be limited.

Attrition

One can note that 'in districts which are still served by a mental illness hospital there may be little value in distinguishing between "new long stay" and "old long stay" patients; for planning purposes, what is needed is a statistical calculation of possible future bed requirements from the rate of decline in both groups (either taken separately or together)' (DHSS, 1985).

The current rate of attrition of those in hospital more than 5 years is 12 per annum. At present rates this group will not disappear by death or discharge until 2003. By comparison, a similar calculation in 1980 postulated extinction of the group by 1996.

Calculation of accumulation rate in the 2–5 years group, after removing those with dementia and organic psychosis, most of whom will die within 5 years of admission, reveals a rate of 9 per annum. Life expectancy tables for this group, average age 66 years, indicate a life of 15 years per patient. This group will, therefore, eventually need 135 beds. Excluding dements and those staying less than 2 years, bed requirements for long-stay patients would level out at 135 in 2003. However, both the excluded groups (i.e. the demented (30) in the 2–5 years group and the whole of the 3 months to 2 years group (91)) are currently increasing.

Various predictions of the 1955 population were made by (a) extrapolation of the total population curve visually, (b) dividing into age and sex groups and using similar extrapolations, and (c) using life expectancy tables for the various groups of duration of stay. These methods predicted 284, 301 and 298 respectively. The mean of these is 294 – a fall of 74 patients over 10 years, or approximately 2% per annum. Thus the hospital population will continue to fall, but at a slower rate.

Conclusion

Employment, even sheltered, is no longer a major feature of in-patient care. As the hospital population ages and national unemployment increases, the values and skills developed by occupational therapists are considered more relevant than industrial therapy. However, for patients residing in the community, industrial therapy continues to be regarded as an essential part of rehabilitation.

The hospital population continues to fall, but the rate of decline is slowing. Predictions suggest that this deceleration will not be reversed. This is particularly relevant at a time when the Department of Health and Social Security claim that 'it is increasingly urgent for regions to decide on a regional strategy for psychiatric services, including hospital closure where relevant' (DHSS, 1981).

If the concepts of community are applied to our resident population, their characteristics suggest that they will need supervision, day care, extensive rehabilitation, and friends in the community. Many will also require periods of psychiatric hospital care.

The slowing in hospital population decline suggests that, except for the demented, increasingly appropriate facilities have not been developed. Of central importance 'is the absence of the necessary infrastructure for services and the financial supports without which talk of community care is surely a sham' (Scull, 1983).

Acknowledgements

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The Extent and Nature of Depressive Phenomena in Primary Health Care

A Study in Calcutta, India

BISWAJIT SEN and PAUL WILLIAMS

The extent and factor structure of depressive phenomena were investigated in three primary health care clinics in the city of Calcutta. The Self-Reporting Questionnaire and the Screening for Depression Questionnaire were used as the first-stage instruments, and the Clinical Interview Schedule and the Hamilton Rating Scale for Depression as the second-stage instruments. The implications of the findings are discussed.

Around 3% of the global population were estimated to be suffering from some form of clinically recognisable depression a decade ago (Sartorius, 1974). If the definition of depression is extended to include depressive phenomena of any degree of severity, estimates given by Shepherd (1983) allow one to calculate that around 15% of the (British) population are so affected. Studies quoted by Kapur & Singh (1983) and Venkoba Rao (1984) reveal that clinically recognisable depressive disorders are as common in India as in the Western world; they were estimated to affect 3–5% in community surveys carried out in the last decade.

Naik & Wig (1980), in a study conducted as part of a larger World Health Organisation investigation in a general hospital out-patient service in Chandigarh in North India, concluded that "about 20% of the cases seen in a general medical practice" exhibit depressive phenomena "with or without other physical illnesses". General practitioners carried out the first-stage screening, with the WHO-SADD (Standardised Assessment of Depressive Disorder; WHO, 1983) being applied to detect depression. Though the socio-demographic profile of the clinic attenders was not provided, it may be assumed that they largely belonged to the lower social classes, from the lower middle class downwards (since this is characteristic of attenders at hospital clinics, where no fees are charged).

Most people suffering from depression are not seen by psychiatrists. Many present themselves to primary care clinics (Shepherd, 1983; WHO, 1975), often with vague somatic problems. It is believed that this is particularly true for developing countries like India where there is a shortage of specialist psychiatric facilities. The overwhelming majority of these patients are not recognised as suffering from depression and thus remain untreated. An important reason for this situation may well be the lack of adequate mental health education of primary health care personnel. This in turn is believed to be, at least in part, due to lack of sufficient knowledge on the nature, frequency, and management of depressive disorders in primary care.

The present study was conducted in Calcutta, in the eastern part of India, in areas housing predominantly pre- and semi-literate people belonging to the lowest socio-economic classes. One of the original objectives of the study was to investigate the extent to which health auxiliaries with limited training were successful in detecting psychiatric morbidity in general, and depressive illness in particular (Sen *et al*, 1987). The present paper, however, is concerned with the following aims: (a) to assess the extent of depressive phenomena in this setting; (b) to explore the nature of depressive phenomena with the aid of factor analysis, and their relationship to anxiety.

Method

Study area

Three primary care clinics situated in the periphery of the city of Calcutta were chosen for the study. A notable feature of these clinics was that all three were situated within small catchment areas of between 3000 and 5500 persons, and more than 90% of the residents in each area were registered with the corresponding clinic. The majority of patients attending these clinics were manual workers living in slums or crowded housing, with primitive sanitary conditions.

Research instruments

Screening for Depression Questionnaire

The SDQ was constructed in Bengali for the purposes of this study. The ICD-9, DSM-III, and WHO-SADD were taken into consideration for the development of this questionnaire. It consists of nine questions designed to elicit symptoms commonly found in almost any variety of depression by means of simple Yes/No answers. The SDQ was designed to be either self-administered or, with pre- or semi-literate persons, to be administered by health auxiliaries.

Self-reporting Questionnaire

The SRQ-20 (Harding *et al*, 1980) was also designed to be either self-administered or given by health auxiliaries, and has been extensively used in developing countries (Harding *et al*, 1983).

Clinical Interview Schedule

The CIS (Goldberg *et al*, 1970) is a standardised, semi-structured inventory designed for use in community surveys and general practice studies. Its feasibility and reliability have been tested in many settings across different cultures (Goldberg *et al*, 1970; Campillo-Serrano *et al*, 1981; Cheng *et al*, 1982; Mari *et al*, 1986). It is administered by a psychiatrist trained in its use, and enquiries about ten groups of 'reported' symptoms and twelve 'manifest' abnormalities are rated along five-point scales.

Hamilton Rating Scale for Depression (HRSD)

The Hamilton scale (Hamilton, 1967) is probably the most widely used depression scale in clinical research. It is designed for quantifying the results of a clinical interview with patients diagnosed as depressed. An 18-item version was used, with 'paranoid symptoms' added to the generally used 17-item scale. A score of 20 or above was held to be indicative of depression (Bech, 1981).

Translation

The SRQ and the CIS were translated into Bengali by the senior author and back-translated into English by a schoolteacher not acquainted with the original versions. The back-translation was subsequently compared with the original version jointly with an English psychiatrist for conceptual equivalence of the items. Minor modifications were made to the translated questionnaires to remove discrepancies.

Collection of data

The design of the study is illustrated in Fig. 1. An unduplicated consecutive series of adult patients (15 years and above) attending the three primary care clinics over a period of four weeks (in July and August 1985) were screened by health auxiliaries with the SRQ and the SDQ. Those who scored 8 or above on the SRQ were designated as 'probable cases' and those with scores of 0–7 as 'probable non-cases'. The psychiatric interview was conducted 'blind' (i.e. without knowledge of the questionnaire responses of the individual).

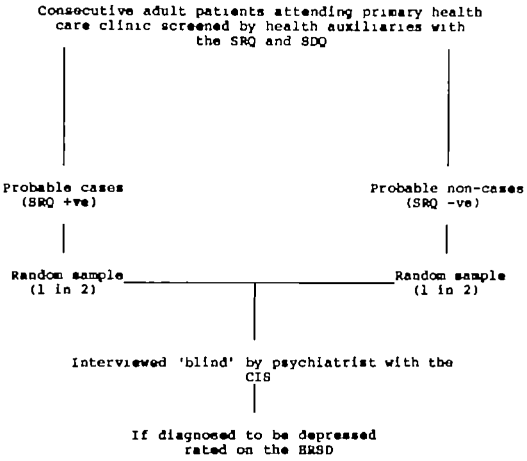
Consecutive adult patients attending primary health care clinic screened by health auxiliaries with the SRQ and SDQ
Probable cases (SRQ +ve)
Probable non-cases (SRQ -ve)
Random sample (1 in 2)
Random sample (1 in 2)
Interviewed 'blind' by psychiatrist with the CIS
If diagnosed to be depressed rated on the HRSD

FIG. 1 Design of the study.

Personnel and training

Three health auxiliaries who had between 8 and 12 years of schooling and were subsequently trained in primary health care were involved in the study. Two were members of the respective communities. They received no particular training apart from a 30-minute orientation session, during which it was ascertained that they themselves understood the questions and the procedure that was required of them. In situations of doubt regarding the presence or absence of the symptom, they were asked to score the symptom as present.

Results

Two patients were so ill that it would not have been feasible to administer the research questionnaires. There were no refusals.

Characteristics of the sample

Out of the total sample of 202 respondents, 48 (24%) were males and 154 (76%) were females. As would be expected in India, almost half the patients were between 15 and 35 years of age. More than a third of the population could not read or write, and only 12% were educated beyond primary school. The average per capita annual income of the population was below Rs1200/-; that is, around $100 (the national average is around $270, according to the 1981 census).

The two questionnaires were administered by the health auxiliaries to 202 consecutive adult attenders, out of whom 114 were interviewed. The interviews were conducted almost immediately after administration of the questionnaires.

Validity of the SDQ

The validity of the SDQ in this sample has been described in detail elsewhere (Sen & Williams, 1987). It was observed that although the sensitivities of the SDQ both against the 'Manifest Depression' items of the CIS and against the Hamilton scale were high (83% and 88% respectively), the specificities (60% and 51%) and positive predictive values (47% and 43%) were low. In search for a better predictor, it was found that a new nine-item questionnaire (SDQ-9; see Table III), constructed from items in the SRQ and SDQ which discriminated most successfully between depressed and non-depressed individuals, had higher validity indices against the two measures of depression (Table I).

TABLE I
Validity indices of SDQ-9

	Manifest Depression	*Hamilton*
Sensitivity: %	77	87
Specificity: %	79	51
Positive predictive value: %	59	43

Extent of depressive phenomena in primary care

Out of 48 males, 13 (27%) scored more than 5 (the cut-off point) on the SDQ-9, 14 (29%) were judged depressed by the Manifest Depression items, and 5 (10%) by the Hamilton scale. Table II shows the estimated number (with percentages) of females of different ages exhibiting depressive phenomena when interviewed with the three scales. It is obvious that an estimate of depressive phenomena varies with the stringency of the applied criteria.

TABLE II
Number of females with scores >5 in SDQ-9, and estimates of the number of females with positive scores in Manifest Depression and in Hamilton, by age (figures in parentheses are age-specific percentages)

Age groups	*Females (n = 154)*		
	SDQ-9	*Manifest Depression*	*Hamilton*
15–24	9 (26)	5 (14)	5 (14)
25–44	38 (54)	42 (59)	23 (32)
45–64	25 (71)	18 (51)	10 (29)
65+	5 (39)	7 (54)	4 (31)
All ages	77 (50)	72 (47)	42 (27)

In the females, it is seen that, as a whole, more than a quarter of all the respondents suffer from marked depression, as judged by the Hamilton scale. It is also observed that the proportion of depressed respondents varies little between the three older age groups, whether judged by the Manifest Depression items or the Hamilton scale. The proportion exhibiting depressive *symptoms* (SDQ-9 score >5), however, increases markedly in the 45–64 age group.

TABLE III
Frequency of positive replies on the SDQ-9 items in males and females

SDQ-9 items	*Males (n = 48)*		*Females (n = 154)*	
	Number	*%*	*Number*	*%*
1. Do you feel unhappy?	28	58	110	71
2. Do you have trouble thinking clearly?	21	44	96	62
3. Do you find it difficult to make decisions?	19	40	89	58
4. Is your daily work suffering?	25	52	76	49
5. Do you see little hope for the future?	20	42	106	69
6. Have you ever thought 'What is the use of carrying on any longer?'	16	33	88	57
7. Are you unable to play a useful part in life?	24	50	93	60
8. Do you feel that you are a worthless person?	19	40	86	82
9. Has the thought of ending your life been in your mind?	6	13	36	23

The frequency of positive replies to the individual items of the SDQ-9 are shown in Table III. A remarkably high proportion of both males and females appear to suffer from each of the nine depressive symptoms. The proportion of females is greater than males in every item except "daily work suffering". However, except for the question on "worthlessness", to which the females answer "yes" twice as often as men, the differences are not as much as would be expected. It is remarkable that more than three-quarters of the females felt "worthless" and more than two-thirds admitted to "unhappiness" and "hopelessness". It is also interesting that almost a quarter of the women admitted to suicidal ideas.

Factor structure of depressive phenomena

A principal components analysis with varimax rotation was carried out to explore the structure of depressive phenomena in the primary health care setting. The procedure yielded three factors for the SDQ (Table IV) and two for the SDQ-9 (Table V). Items with factor loadings of less than 0.5 were disregarded, as it is generally agreed that such items are found less consistently under the same factor across populations.

Among the three factors isolated from the SDQ, factor I may be said to represent a vignette which is familiar to clinicians in India as one of the typical pictures of depression

TABLE IV

Principal components analysis with varimax rotation of SDQ

	Factor loading
Factor I	
(25.7% variance explained)	
Think it is better to die	0.721
Inner restlessness	0.719
Hopeless	0.653
Sad	0.556
Lack in self-confidence	0.532
Factor II	
(12.9% variance explained)	
Guilt	0.655
Crying	0.645
Factor III	
(12.6% variance explained)	
Loss of interest	0.732
Sad	0.550
Weak	−0.504

(Wig *et al*, 1980). However, factors II and III cannot be clinically conceptualised with ease as separate entities, and hence are difficult to interpret in the Indian context as meaningful subgroups of depression. This is not surprising as three of the five items constituting the two factors, namely "guilt", "loss of interest", and "weak", were among the poorest discriminators for depression in an item-by-item analysis.

In contrast, both the factors isolated from SDQ-9 seem capable of a sensible interpretation. Items in factor I are mostly general in nature and may be expected to predict relatively mild depression, perhaps including even 'normal' depression. Items in factor II would be expected, on the other hand, to predict depression of a marked degree. Such an interpretation is suggested by the fact that the positive predictive value of factor II against the Hamilton scale (which is assumed to indicate depression of a marked degree) is the same as that of the SDQ-9 as a whole (52%, calculated for 30% prevalence). In contrast, the positive predictive value of factor I against the Hamilton scale is lower than that of the SDQ-9 (46.9%).

TABLE V

Principal components analysis with varimax rotation of SDQ-9

	Factor loading
Factor I	
(39.1% variance explained)	
Unable to play a useful part in life	0.790
Worthless	0.643
Daily work suffering	0.639
Cannot think clearly	0.584
Difficulty in making decisions	0.573
Unhappy	0.516
Factor II	
(11.4% variance explained)	
Think of suicide	0.824
Think it is better to die	0.792
Hopeless	0.505

Relationship of depressive with other psychiatric phenomena

Since the SRQ contains questions related to psychiatric phenomena other than depression, it was decided to

TABLE VI

Principal components analysis with varimax rotation of the SRQ

	Factor loading
Factor I	
(21.8% variance explained)	
Unhappy	0.745
Anxious/tense/worried	0.680
Unable to play useful part in life	0.507
Factor II	
(7.8% variance explained)	
Lost interest	0.768
Crying	0.623
Think of suicide	0.513
Factor III	
(6.5% variance explained)	
Easily frightened	0.737
Headaches	0.556
Cannot think clearly	0.511
Factor IV	
(6.1% variance explained)	
Cannot enjoy daily activities	0.751
Cannot think clearly	0.507
Difficulty in making decisions	0.506
Factor V	
(6.0% variance explained)	
Poor digestion	0.818
Abdominal discomfort	0.810
Factor VI	
(5.5% variance explained)	
Poor appetite	0.681
Sleep badly	0.680
Factor VIII	
(5.3% variance explained)	
Easily tired	0.812
Constantly tired	0.739

investigate the relationship of these phenomena with depression, with special reference to those related to anxiety. A principal components analysis with varimax rotation isolated seven factors (Table VI).

It may be noted that factor I, which may be conceptualised as an anxiety-depression component, explained 21.8% of the variance. Each of the other six factors explained between 7.8% and 5.3% of the variance. It is observed that there is no clearly defined separation between depression, anxiety, or somatic phenomena in any of the factors.

Since factor I, which contains the most direct of the three questions on anxiety, explained more than a fifth of the variance and also discriminated moderately well between depressed and non-depressed respondents on the Manifest Depression items and the Hamilton scale (Sen & Williams, 1987), it was decided to explore further the relationship between depression and anxiety.

Out of 114 respondents interviewed, 32 (28%) had clinical anxiety, all of a mild nature. All but five of them were judged to be suffering concurrently from depression.

The three anxiety items on the SRQ, namely "anxiety/tension/worry", "easily frightened", and "hand tremor", were taken together, and validity indices of this new scale (termed SRQ-Anxiety) were obtained against the "manifest" anxiety scale of the CIS (termed Manifest Anxiety) as well as against Manifest Depression. Indices of validity were also obtained for SDQ-9 against Manifest Anxiety for comparison. The indices are shown in Table VII.

TABLE VII

Exploring the relationship between depression and anxiety: validity indices of SRQ-Anxiety against Manifest Anxiety and Manifest Depression, and SDQ-9 against Manifest Anxiety

	SRQ-Anxiety v. Manifest Anxiety	*SDQ-9 v. Manifest Anxiety*	*SRQ-Anxiety v. Manifest Depression*
Sensitivity: %	43.8	59.3	52.2
Specificity: %	70.7	72.0	78.6
Positive predictive value (adjusted for prevalence): %	48.7	57.5	60.5

It is evident that the SDQ-9, which does not contain any question to detect clinical anxiety, is superior to SRQ-Anxiety in screening for anxiety. It is also interesting that SRQ-Anxiety performs better in screening for depression than it does for anxiety.

Discussion

Extent of depressive phenomena in primary health care

Studies reporting the extent of depression in primary health care in India are few. As indicated earlier, Naik & Wig (1980), using WHO-SADD as the second-stage instrument, studied the prevalence of depressive symptoms in a general medical practice in Chandigarh and found that around 20% of the clinic attenders had depressive symptoms. Reviewing available literature, Venkoba Rao (1984) estimated that the prevalence of depression in general medical practice varies from 5% to 25%, depression being "four to five times more in northern and eastern India" than in the south and west. In a rural primary health care clinic not far from Calcutta, Banerji *et al* (1987) reported that in a sample of 80 adults who had been attending regularly for at least six months, the prevalence of depression, as judged by clinical interview, was 20%.

In the present study, clinically recognisable depressive phenomena, ranging in intensity from mild to marked, is estimated to occur in 42.6% of the respondents (proportion positive on Manifest Depression), with 23.3% being diagnosed as suffering from depression of marked intensity (proportion positive on Hamilton). Over a quarter of the women (27.3%) of all ages were estimated to be depressed on the Hamilton scale.

Informal enquiry among the 'Hamilton positive' women in the interviewed subsample revealed that none of them were under psychiatric care, a finding which is not surprising in this setting. While they were obviously aware that their 'illness' merited medical attention, they apparently did not consider that the 'illness' was a potentially treatable psychiatric condition. The medical officers in charge of the clinics reported that, while they suspected that a section of their patients might be depressed, they thought this to be due to their adverse socio-economic and, perhaps, family circumstances, factors which they felt to be beyond their control, especially in view of their "chronic patient overload". This situation is believed to be representative of the Indian context. Thus the responsibility for the treatment and care of these individuals may be expected to devolve upon the health auxiliaries (rather than mental health professionals), who, of course, have to be appropriately trained for the purpose.

Among Western studies, Stoeckle *et al* (1964) interviewed 101 "new patients" coming to the medical clinic of the Massachusetts General Hospital and found "depressive reactions" in 59.4% (men 49%, women 69%). Hoeper *et al* (1979) studied the prevalence of psychiatric disorders in Marshfield Clinic in Wisconsin, and applying SADS-L criteria, estimated that 14.2% of a sample 1072 patients who consented to participate in the study (there were 255 refusals) were suffering from depressive disorders.

Parker *et al* (1984) applied PSE criteria to 564 subjects, predominantly women, attending "representative (general) practices" in Sydney: they calculated that at least 18% had depressive disorder. Skuse & Williams (1984) estimated that 26% of all the patients attending a general practice in London were depressed. Their diagnosis was based on joint rating of a structured clinical interview.

Generalising from these studies in India and the West about the prevalence of depression in general practice/primary health care studies may not yield precise estimates because of the cultural, social and demographic differences between the populations, and also because of the different diagnostic criteria used. However, two trends are readily observed. It appears that, with hospital-oriented criteria, between one-sixth and one-fifth of primary health care clinic attenders are diagnosed as depressives. With less stringent criteria, more suited to the primary health care situation, between two- and three-fifths of the attenders are seen to manifest depression.

In Table II, the sudden and marked increase in the proportion of women between the ages of 45–64 years exhibiting depressive symptoms (SDQ–9 score >5) is difficult to interpret. Certainly the phenomenon needs to be replicated in other studies before a proper evaluation can be carried out. A possible explanation may be an exaggerated emotional response to somatic disorders at a period of life when physical stamina is on the decline, with no corresponding reduction in day-to-day responsibilities, which mostly require manual labour. Clinically, this period of life is associated with an increased frequence of consultation for minor physical complaints. If the excess of 'depressive' complaints is seen to be restricted to the clinic setting, the explanation offered would receive support.

It can also be noted in this context that certain life events which may predispose to depressive symptoms are common during this period (e.g. children getting married and leaving home; husband's retirement, with consequent reduction or loss of regular income). It needs to be investigated whether this phenomenon is also observed in community studies in similar settings.

Nature of depression in primary health care and the relationship with anxiety

SDQ–9

The clinical interpretation of factors I and II (obtained from SDQ–9, Table V) as 'mild' and 'marked' depression respectively has been mentioned. It is not claimed, however, that these are distinct categories. On the contrary, the finding that factor I ('mild depression') discriminates between Hamilton positive and negative individuals with a fair degree of accuracy lends support to the dimensional view of depression rather than the categorical. It may be that factor I is representative of individuals with 'distress states' which do not require physical forms of treatment like tricyclics and ECT. Indeed, it is possible that the majority of individuals experiencing similar states discontinue prescribed tricyclics within a few days of commencement because of 'intolerable' side-effects. Factor II appears more likely to be representative of individuals who would benefit from drugs and ECT. It is planned to test this hypothesis in the near future in one of the clinic areas.

The relationship between depression and anxiety has recently been reviewed by Stavrakaki & Vargo (1986). They noted issues such as the presence of mixed anxiety and depression, and contradictory research findings, and suggested that "there is no unequivocal solution to the problem of how anxiety and depression are related". While most of the studies they reviewed were from Western countries, Leff (1981) noted that in India also there is a considerable degree of overlap between patients' concepts of anxiety and depression, a view which finds support in the present study. However, the findings of this study allow this notion to be taken further. It is possible that the reason for the overlap is that anxiety is found most often as a concomitant of depression and is thus necessarily highly correlated with the latter. The inability of respondents to distinguish between depression and anxiety could possibly be attributed to the same cause.

The undifferentiated nature of different psychiatric phenomena

Principal components analyses with varimax rotation of GHQ–30 and GHQ–60 data have been reported in many studies (see the review by Vieweg & Hedlund, 1983). Most of these have revealed an anxiety–depression/dysphoria component which explained 20–25% of the total variance. Two studies, both using the GHQ–60, have reported this component explaining over 50% of the total variance (Worsley *et al*, 1978; Chan & Chan, 1983).

To our knowledge, this is the first occasion on which the factor structure of the SRQ has been reported (Table VI). It is suggested that factor I, which explains 21.8% of the variance, corresponds with the anxiety–depression/dysphoria component of the studies quoted above. While a case may be made out, in the light of this finding, in favour of the universality of an anxiety–depression syndrome in primary health care and community studies, the present situation is seen to be more complex from an examination of the other factors isolated from the SRQ. Whereas in the studies reporting the factor structure of the GHQ the several factors could be conceptualised with relative ease, factors II to VII do not readily lend themselves to clinically meaningful interpretation. The lack of clear separation of the various psychological and somatic dimensions suggests that much of the psychiatric morbidity in primary health care is undifferentiated, a view similar to those expressed by Shepherd (1977).

Clinicians in India are familiar with the generally undifferentiated nature of the complaints expressed by patients, particularly those coming from the lower social strata. Not infrequently, even experienced psychiatrists, thoroughly familiar with the subculture represented by the patient, are hard put to place the subject in an appropriate diagnostic category. This may be appreciated from the coining of a term, possibly arising more out of a sense of desperation than any attempt at clarification, such as 'panneurosis'.

The factor structure of the SRQ, with special reference to the relationship between depression and anxiety, has particular relevance for the classification of minor psychiatric disorders in the primary health care settings in India. The essentially undifferentiated nature of the symptom patterns suggests that narrowly defined criteria of the various neurotic disorders familiar to clinicians will serve little purpose in these settings. This study has shown that health auxiliaries are able to diagnose depressive disorders with some degree of confidence. The detection and nature of other types of psychiatric phenomena must remain, for the present, objects of further epidemiological research.

Conclusion

The essential objective of this paper was to explore the extent and nature of depressive phenomena in the primary health care setting in India, with reference to their relevance to health auxiliaries with limited training. The importance of diagnosing depressive disorders in primary health care clinics has been established. The extent to which health auxiliaries can be successful in treating such disorders now needs to be studied. The long-term view must, of course, be that of exploring the means whereby health auxiliaries can succeed in preventing their occurrence. This, perhaps, is not as distant a goal as it appears at first sight. The societal structure in India is generally held to be orientated strongly towards the extended family, which is held together by strong cohesive forces. The potential for emotional support which exists within the extended family is an important resource for patients with minor psychiatric disorders (Aneshensel & Stone, 1982). And the person who is ideally suited to mobilise this resource, being the health professional closest to the family, is the health auxiliary. With suitable training and education (Wig *et al*, 1981), the health auxiliary should be able to play a cardinal role in the management of psychiatric morbidity in primary health care settings in the foreseeable future.

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Psychological Intervention in Civilian Flying Phobia

Evaluation and a Three-Year Follow-Up

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Thirty-eight people with a moderate to severe degree of aerophobia self-referred themselves to a course designed to help them travel by air. The course involved three long sessions based on giving information, graded exposure without avoidance, and group support with natural coping models. Measures of self-reported anxiety levels were taken at regular intervals. The results showed that being given information or undergoing a simulated flight had little effect on perceived anxiety. Prolonged exposure to flying had a marked effect on reducing anxiety and anticipated apprehension about future flying. At one-year and three-year follow-up, 40% and 60% of participants had flown commercially, although there was some minor restoration of anticipated anxiety associated with flying; 84% showed less anxiety about flying. Psychological intervention, in collaboration with airlines, may have marked benefits in reducing fear of flying.

Many people are apprehensive about air travel. Agras *et al* (1969), in an epidemiological study of phobias, reported fear of flying to be intense in 10% of the population and mild in 20%. For some, the anxiety associated with such travel causes psychological and/or physiological symptoms severe enough to prevent the sufferer from flying. In consequence, social life, vacations or job opportunities may be impaired.

Most of the research into the treatment of phobic reaction to flying has concentrated on military personnel and trained air crew (Anderson, 1948; Morgenstern, 1966; Goorney, 1970; Aitken *et al*, 1970; O'Connor, 1970; Daly *et al*, 1970; Aitken *et al*, 1981). Anderson (1948) believed that "the prognosis is usually bad". Morgenstern (1966) concluded that there was no successful treatment for USAF personnel, although Goorney (1970) did note flying phobia to be a treatable condition. This was confirmed by Aitken *et al* (1970) when, as a result of using a combination of behavioural strategies together with psychotherapy, 9 out of 14 aircrew were able to return to limited flying duties. Each of these studies had discussed flying phobia and its treatment with specific reference to the *acquisition* of such fear in persons whose occupation involved operational flying and which subsequently led to their avoidance of flying.

Denholz *et al* (1974, 1978) described an automated audio-visual programme which combined desensitisation, modelling and positive reinforcement, so allowing 40 out of 51 (78.4%) civilian participants, who undertook the programme, to fly. Twenty-six of those who had flown after treatment were contacted three and a half years later. Twenty-three of them had maintained their ability to fly.

Solyom *et al* (1973), in a study of treatment efficacy for 40 patients with air travel phobia, used three behaviour therapy techniques – aversion relief, systematic desensitisation, and habituation – all of which proved equally effective in significantly reducing the phobia, while group psychotherapy was ineffective. However, at follow-up, the difference between the approaches was no longer significant. The authors suggested that the behaviour therapy techniques achieved a more immediate result because they were more directly symptom-orientated. They also found that previous extensive flying experience tended to reduce the benefits of therapy.

In a study on 56 self-selected but screened civilian volunteers with a fear of flying, Howard *et al* (1983) compared results on small groups allocated to different regimes of therapy. After an eight-week programme of either systematic desensitisation, flooding, implosion, relaxation or no treatment, the subjects and therapists took a plane flight. Measures used during the flight failed to distinguish between the groups, a result which was attributed to the dilution of the comparative groups. We investigated a form of intervention on a larger number of subjects, which involved education, desensitisation and flooding.

Method

Participants

Participants were self-selected to the programme following national television and local radio coverage of a similar, apparently highly successful, course undertaken previously by two of us (JSMcC; NB) working with a local self-help group. In order to select *out* those with a simple apprehension, rather than a true fear of flying, entry requirements to the programme for evaluation were relatively strict. Each enquirer was sent information about the programme together with an enrolment form. All applicants were required to obtain in writing their doctor's certification that they were mentally and physically fit to attend the course. Forty-six people were recruited, 38 of whom completed the programme (11 males; 27 females). Their mean age was 44.3 years (s.d. 12.4 years).

Comparison group

Twelve people enquired about the programme too late to attend. They formed a comparison group (3 males; 9 females). Their mean age was 50.5 years (s.d. 10.6 years).

Programme

The principles of the intervention strategy included preparation by information, graded exposure over long sessions without avoidance, and group support with natural coping models. The course consisted of three sessions, each approximately three hours long, separated by one to two weeks.

The first session took the form of a group meeting at which talks were given by a psychologist on the physiology and behavioural control of fear; and by an experienced pilot on flight procedure and safety.

Session two was held at a nearby airport with the co-operation of its Director and British Midland Airways. It was designed to simulate all aspects of civilian flying without actually taking off. Participants were asked to check in and wait in the departure lounge until their 'flight' was called. During the waiting period help and support were given, where appropriate, by the course organisers although generally participants were encouraged to feel in control and to be personally coping. After being given an hour to become accustomed to the airport atmosphere, the group went through customary security checks and boarding procedures. On board, the passengers were seated, seat belts fastened, the aircraft doors closed, the steps removed and the engines started. Each procedure was explained carefully over the intercom by the pilot, and a simulated flight then took place. During this time standard announcements concerning altitude, weather, positional checks etc. were made. The atmosphere of realism was further reinforced by the serving of light refreshments by the cabin crew.

At the third session, smaller subgroups of between six and eight people undertook a scheduled return flight (courtesy of the airline) lasting approximately one hour each way. They were accompanied by two of the course organisers. During a 30-minute turn-around period participants remained on the aircraft. After disembarking, the course organisers reinforced the participants' coping strategies and emphasised the positive aspects of their experience.

Evaluation techniques

On enrolment in the programme, participants gave general demographic details of themselves and indicated whether they had flown previously, what worried them about flying and if they had ever received treatment for 'nervous tension'.

A series of 10 cm visual analogue scales was constructed. These were designed to monitor the severity of anxiety each participant felt they would experience in a number of situations connected with flying. The scales were completed before the first session, two days after finishing the course and at a one-year follow-up. The comparison group completed these scales one month apart, only.

All participants completed Spielberger's State Anxiety Inventory (SSAI) (Spielberger *et al*, 1970) during the introductory meeting and before boarding the aircraft at the second session. The SSAI was also completed on a further three occasions during the last session: one prior to boarding; one on board shortly prior to take-off and one mid-way through the return flight.

At follow-up, one year later, participants were asked whether they had flown during that period, and if so, how often. They were asked to comment on their experience and whether the programme had influenced their views about flying. At a three-year follow-up, participants were again contacted in order to identify a total number who had subsequently flown.

Results

Ill health, business pressures or the intervention of holidays prevented five people from completing the programme, three of whom did subsequently fly. A further three participants were too fearful to attempt the final session. This left a total of 38 people who completed all three sessions.

What worried people about flying?

Fourteen (37%) of the participants were worried about 'being enclosed'. That was the most common fear expressed. Thirteen people (34%) were afraid of crashing. Five people (13%) were frightened of heights. The remainder were fearful either of loss of control or air-sickness, or had multiple worries.

Anxiety profile across the programme

The median self-rated state anxiety scores are shown in Fig. 1. Analysis by the Wilcoxon matched-pairs test shows that there was no statistically significant difference between the scores obtained in the introductory session and those given before the simulated flight. Compared with initial levels, anxiety scores rose significantly before the actual flight ($P<0.001$) and, although falling significantly after take-off ($P<0.001$), remained higher during the outward flight ($P<0.01$) than at the initial meeting. On the return flight, however, anxiety levels fell markedly and were significantly less than they had been at the first meeting ($P<0.002$).

The Mann–Whitney U test identified less anxiety in the claustrophobic group in comparison with the group fearful

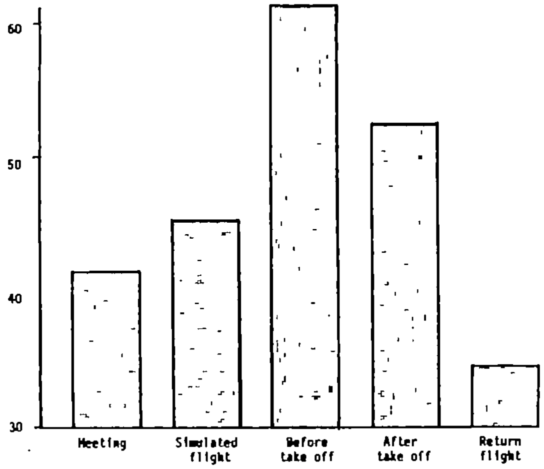

FIG. 1 Median Spielberger Anxiety Scores measured at points throughout the course.

of heights both after take-off ($P<0.05$) and on the return flight ($P<0.05$). The claustrophobic group, too, in comparison with those fearful of crashing, was less anxious after take-off ($P<0.05$) and on the return flight ($P=0.05$).

Scores on the visual analogue scales, completed two days after the programme, were compared with those obtained before the first meeting. After completing the course, participants felt less anxious about looking at photographs of aircraft ($P<0.04$), meeting people at an airport ($P<0.002$), watching a film of an aircraft taking off ($P<0.04$), waiting to board an aircraft ($P<0.001$), sitting in an aircraft ($P<0.001$), taking off ($P<0.001$), and flying over land ($P<0.001$) or water ($P<0.001$). They also rated aircraft as being less often in the news ($P<0.001$).

Comparisons were made of the analogue scores given by the untreated comparison group and those given by the course participants. The Mann–Whitney *U* test showed that the two groups did not differ initially on any scale, except one. The comparison group rated aircraft to be less often in the news than did the experimental group ($P<0.02$). After intervention and compared with the comparison group, the experimental group thought they would feel calmer when waiting in the departure lounge ($P<0.001$), boarding an aircraft ($P<0.001$), sitting in an aircraft ($P<0.001$), taking off ($P<0.001$), and flying over land ($P<0.001$) or water ($P<0.001$) (Fig. 2).

The mean anxiety scores of those who enrolled on the course but who were unable to complete it either because they were too fearful or because of ill-health, business or social reasons are shown in Table I. Small numbers preclude formal statistical analysis but it does appear that there is no marked difference in anxiety scores between those too fearful to undertake the final session, and those who either did undertake it, or for other reasons were unable to do so.

One-year follow-up

Thirty-six (95%) of the participants returned the follow-up questionnaire and analogue scales sent to them one year later. Compared with their preliminary completion of the scales, all subjects were significantly less anxious about all the aspects of flying covered by the questions. However, when compared with the scales completed immediately after the course, there was a significant rise in perceived anxiety associated with sitting in an aircraft ($P=0.001$), flying over land ($P=0.004$), and flying over water ($P=0.005$) (Fig. 3).

Two people did not return the questionnaires. One of them had previously communicated that she had undertaken a flight. Thus fifteen (39%) of the group had flown in the intervening year. Of the remainder, eleven people said they had not had the opportunity to fly; eight others had definite plans to do so and three more were too anxious to consider flying. One only was lost to follow-up. Thirty-two (84%)

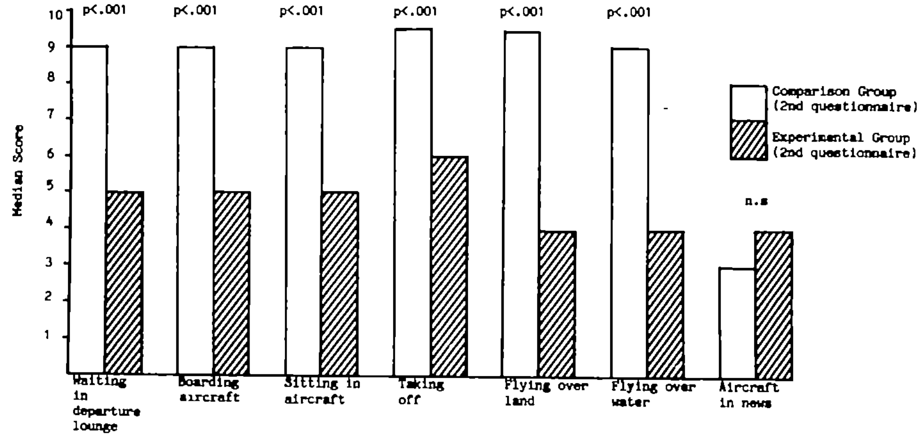

FIG. 2 Median score for fear of flying, visual analogue scale (after intervention): comparison group *v*. experimental group

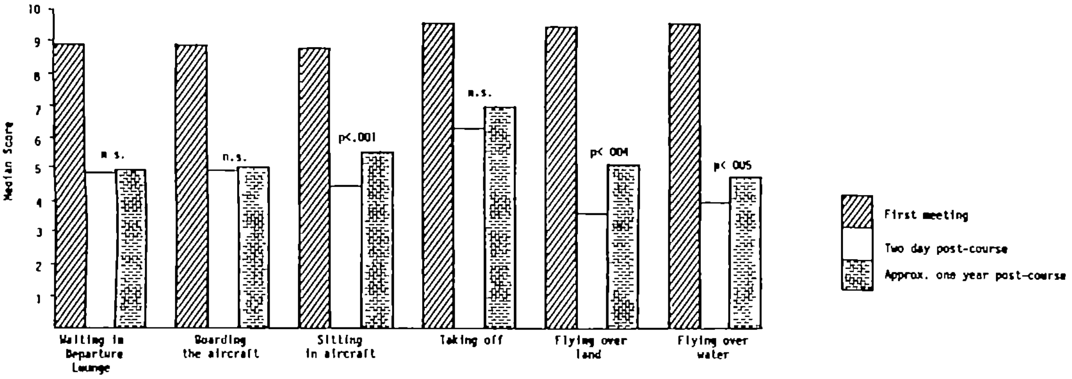

FIG. 3 Median score for fear of flying, visual analogue scale: the *P* values show the differences between the two-day post-course and the one-year post-course measurements

TABLE I
Mean SSAI scores for participants

	First meeting			*Simulated flight*		
	Mean	*s.d.*	*n*	*Mean*	*s.d.*	*n*
Subjects too fearful to complete the course	36.66	10.115	3	51.66	21.571	3
Subjects not completing the course for other reasons	45.00	13.416	5	46.75	0.957	4
Experimental subjects	43.44	14.157	36	46.37	12.661	37

of the participants reported themselves to be less anxious about flying.

The only variables to discriminate between those who had flown during the year and those who had not were those linked to changes in anxiety during the course. Participants who had flown after the course were those who had shown greatest reduction from the first session to the post-course evaluation in their rated anxiety while in the departure lounge ($P = 0.01$), boarding an aircraft ($P = 0.004$) and sitting in an aircraft ($P < 0.005$).

Three-year follow-up

Three years after the course, a total of 23 participants reported that they had flown (61%), ten had not flown (26%), and from five there was no reply to a questionnaire (13%).

Discussion

The aim of the course was simply stated as one of offering applicants the necessary degree of help and encouragement needed to enable them to travel by air. Since over 60% of the participants subsequently flew, it seemed that a fair degree of success could be claimed. However, since the subjects were self-selected, it is possible that they did not represent the most extreme cases of aerophobia. Nevertheless, two factors suggest that they did represent a group of people who were particularly fearful of flying. Firstly, nearly half the group had never flown before; and of those who had, the majority had not flown for many years. Secondly, there was the commitment needed to fulfil the requirements of the course. Participants had to obtain permission from their general practitioner and provide a certificate of fitness. Many had to travel substantial distances in winter weather conditions to attend the three long and stressful sessions. In short, they were a particular group but we thought them to be fairly typical of people with a moderate to severe fear of flying.

If subjective ratings are considered, the course also appears to have been successful. Although the confidence that was evident immediately after the course-flight had diminished slightly at the one-year follow-up, subjects did rate themselves as significantly less anxious about flying than they had been prior to the course. Of those who completed it, 84% claimed to be subsequently less anxious about flying. It was the apparent success of the course which made it difficult to assess which people had benefited most from it. It had been assumed that its educational element, together with the reassuring presence of an experienced pilot, would particularly benefit the inexperienced passenger and those who were anxious about crashing. This hypothesis was not, however, borne out by our findings. Similarly the claustrophobic group, who we feared would find the experience particularly stressful, was significantly less anxious during the flight than the rest of the group.

A secondary aim of the study was to attempt to identify those people who would benefit most from this form of treatment. In view of previous findings by Gelder *et al* (1973) that different types of phobias do not respond differently to flooding with the feared stimulus, it was perhaps not surprising that we failed to identify those who would do particularly well as a result of our intervention. The claustrophobic group was significantly less anxious than other groups during the course-flight but that was not reflected in their willingness to fly after the course.

Those who did subsequently fly were noted to have shown the greatest reduction from the first session to the post-course evaluation in their anxiety levels while in the departure lounge, boarding an aircraft and sitting in one.

From our results, it would seem unlikely that the personal characteristics of the phobic subject were important in determining the success of the treatment programme. The results, however, do seem to show that this form of group treatment, in civilian aerophobics, can be effective. Furthermore, in contrast to previous studies, which have involved numerous sessions, required specialised equipment and the time of psychologists skilled in behavioural therapy, our strategies were relatively economical (Solyom *et al*, 1973; Denholz & Mann, 1974; Denholz *et al*, 1978). Since we showed little evidence for an anxiolytic effect of giving phobics information about flying, it may be that more emphasis on exposure to the airport environment and aircraft and less emphasis on educational aspects might improve the therapeutic response and reduce the number of sessions needed.

Gelder *et al* (1973) have reported that prolonged exposure to the feared stimulus is as effective as systematic desensitisation in relieving phobias. Continuous exposure to the feared situation also seems more effective than continued brief ones (Stern & Marks, 1973), the latter of which may paradoxically increase fear (Stone & Borkovec, 1975). Our participants remained on the aircraft during the turn-around period in the third session, and that continuous exposure may have contributed to the marked fall in anxiety levels observed during the return flight.

Exposing phobics *in vivo* to the feared situation needs careful regulation: there are ethical problems in indiscriminately encouraging phobics, especially those with previous traumatic experiences of flying, to attempt air travel without appropriate psychological support. Nevertheless, with air travel becoming an increasingly routine activity, it does appear possible that airlines, co-operating with psychologists, could attempt some of the principles used in the present study in order to help some of their more fearful potential passengers to fly.

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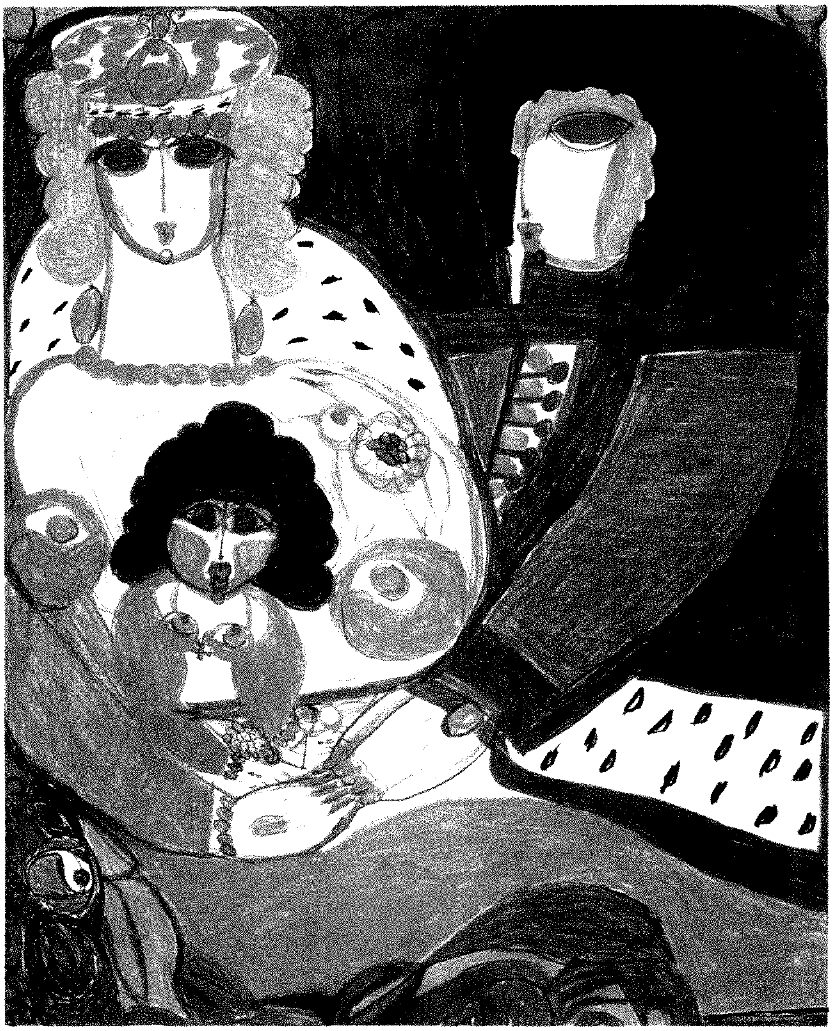

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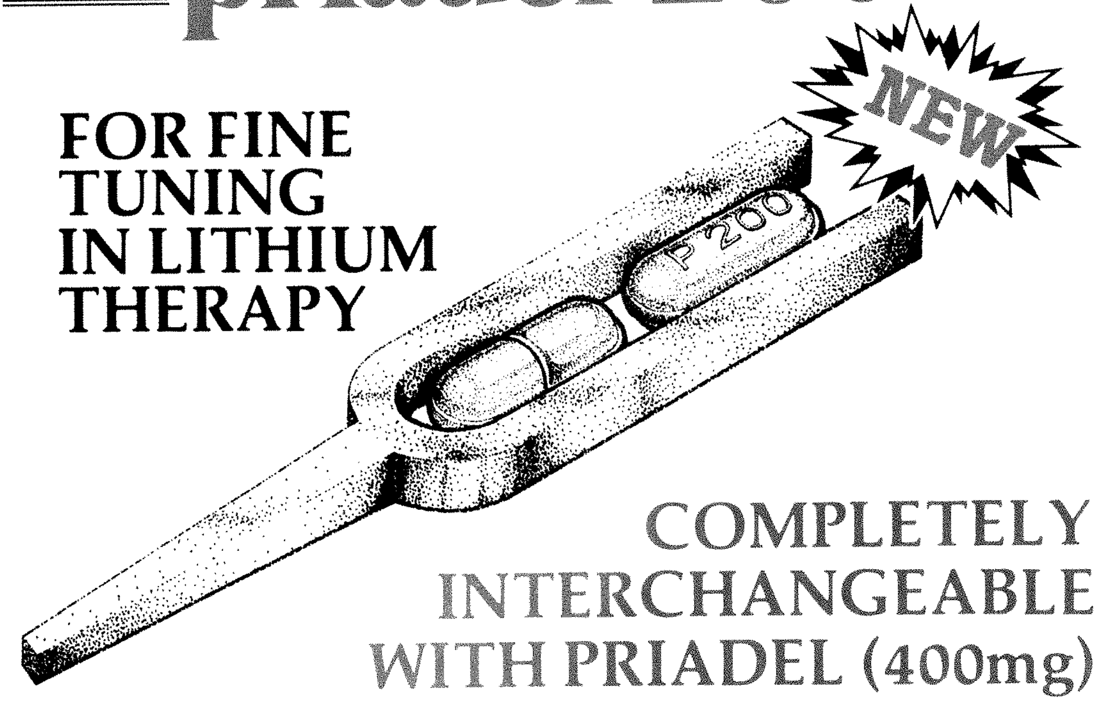

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British Journal of Psychiatry (1987), **151**, 499–505

Winter-Born *v* Summer-Born Schizophrenics

R. E. KENDELL and I. W. KEMP

Data from two sources – the Edinburgh Psychiatric Case Register and the psychiatric in-patient records of the Scottish Health Service – were used to compare large populations of first-admission schizophrenics born in winter (January to March) and in summer (June to October). Parallel comparisons were carried out for affective psychoses. Comparison of the months of birth of the Scottish patients with those of the general population indicated that there was a 9% excess of schizophrenic births and a 3% excess of affective births in the first 3 months of the year. In the Edinburgh material, winter-born schizophrenics were more likely than the summer-born to receive a diagnosis of paranoid or schizoaffective schizophrenia and less likely to receive diagnoses other than schizophrenia on readmission, but neither of these differences emerged in the much larger Scottish material. There were no differences between winter- and summer-born schizophrenics in age of onset, sex ratio, or prognosis in either data set, nor were any significant differences found between winter- and summer-born affectives. We have therefore failed to demonstrate any convincing differences between winter- and summer-born schizophrenics.

In many countries in the temperate latitudes of the northern hemisphere, schizophrenics are born in the winter months more often than other people. In Sweden (Dalen, 1968), Denmark (Videbech *et al*, 1974), Norway (Ødegaard, 1974), England and Wales (Hare *et al*, 1974) and the United States (Torrey *et al*, 1977), large representative populations of schizophrenics have been compared with general population controls closely matched for year and place of birth. In all of these countries a modest but statistically significant excess of schizophrenic births was found in the winter months, particularly January, February and March. Similar well controlled studies have not yet been carried out in the southern hemisphere, but the indications are that there is a similar excess of winter births (i.e. in July to September) in South Africa (Dalen, 1975) and Australia (Parker & Neilson, 1976; Jones & Frei, 1979). Although Hare *et al* (1974) found an excess of winter births in manic–depressives as well, most investigators have found this season-of-birth effect only in schizophrenics. It also seems likely that the birth dates of the siblings of schizophrenics do not differ from those of the general population (Buck & Simpson, 1978), although the evidence on this score is conflicting (Hare, 1976). This all suggests that some environmental influence acting *in utero* or in the early months of life and varying with the season of year is contributing to the aetiology of at least some forms of schizophrenia. A viral infection is one of many possibilities, and receives some support from the finding that the excess of schizophrenic births is greater in years with particularly cold winters (Hare & Moran, 1981).

If some agent making an important contribution to the aetiology of schizophrenia acts only, or mainly, in the winter months, there might be differences between the schizophrenic illnesses to which this seasonal agent had and had not contributed. If so, there might be detectable differences between summer-born and winter-born schizophrenics – in age of onset, perhaps, or in symptoms, treatment response or prognosis. However, even if one assumes that this seasonal agent acts only in the three winter months January to March, and that it makes a major contribution to aetiology, the fact that the observed excess of winter births was only about 8% in most of the studies referred to above implies that, even in these winter months, this seasonal agent is either making no contribution at all to the aetiology of over 90% of future schizophrenic illnesses or, if it is making a contribution, no more than it does at other times of the year. This means that very large populations of summer- and winter-born schizophrenics would have to be compared before significant differences between the two could be expected to emerge, even if illnesses to whose aetiology the seasonal agent had contributed were appreciably different in some respect from other schizophrenic illnesses. However, Dalen (1975) and Pulver *et al* (1983) have both reported that the excess of winter births is greater in, or restricted to, schizophrenics with a relatively good prognosis, and Watson *et al* (1984) have reported that it may be greater in unmarried schizophrenics, which suggests that there may be important differences to be found.

This study reports two attempts, one based on 571 schizophrenics from the Edinburgh Psychiatric Case Register, the other on 2653 schizophrenics from the whole of Scotland, to detect differences between those born in winter and summer months.

Method

Edinburgh Psychiatric Case Register (EPCR) data

The EPCR covers the 470 000 inhabitants of the city of Edinburgh and has recorded data on all psychiatric admissions since 1 January 1970 and on all psychiatric contacts, as in-patient or general hospital ward referral, since 1 January 1978. Information was extracted from the register files in March 1983 on all first-admission schizophrenics (i.e. first admissions to the EPCR since 1 January 1970, including the resident population of the Royal Edinburgh Hospital on that date, with a discharge diagnosis of ICD 295) who were aged 15–39 inclusive at the time of admission and whose date of birth was known.

The 222 schizophrenics fulfilling these criteria with a birth date between 1 January and 31 March were then compared with the 349 born between 1 June and 31 October on the 13 variables listed in Table I. An analogous comparison was also carried out for affective psychosis first admissions; that is, between the 198 first admissions aged 15–39 with a discharge diagnosis of affective psychosis (ICD 296) who were born in January to March and the 277 born in June to October. The reason for this parallel comparison of first-admission affective psychotics was to check that any difference found between winter- and summer-born schizophrenics was specific to schizophrenia.

The reason for comparing the 3-month period January to March with the 5-month period June to October was more complicated. Scotland happens to be one of the few countries in north-west Europe in which the birth dates of schizophrenics have not been compared with those of the population as a whole, so the months with an excess of schizophrenic births are unknown. In England and Wales and most northern-hemisphere countries these are, in most years, January to March, but in Norway the excess extends from January to May (Ødegaard, 1974), in Eire the excess is in the second quarter of the year (O'Hare *et al*, 1980) and in the United States it extends from December to May (Torrey *et al*, 1977). Omitting patients born in November, December, April, and May therefore minimises the chances of the summer-born population being contaminated with patients who actually represent part of the winter peak.

TABLE I

EPCR items on which winter- and summer-born patients were compared

1. Age on admission
2. Sex
3. Marital status
4. Admission status (formal or informal)
5. Place of birth
6. Social class
7. Employment status before admission
8. Living group before admission
9. Duration of initial admission
10. Number of readmissions 1970–1982
11. Cumulative duration of all admissions 1970–1982
12. Discharge diagnosis (initial admission)
13. Discharge diagnosis (all readmissions)

There was no significant difference between winter- and summer-born schizophrenics on any of the first eleven items listed in Table I (see Table II). There was, however, a significant ($\chi^2=14.6$ and $P=0.044$) difference in the distribution of the original discharge diagnoses of the two, more of the winter-born patients having a diagnosis of paranoid or schizoaffective schizophrenia and more of the summer-born patients a diagnosis of latent schizophrenia or acute schizophrenic episode (Table III). The distributions of their readmission diagnoses were also very different (Table IV), with a far higher proportion of the readmission diagnoses of the summer-born patients being diagnoses other than schizophrenia (33% *v.* 15%). Comparison of these two distributions using the chi-squared test suggests that they are dramatically different ($\chi^2=38.5$ and $P<0.0001$ with 2 d.f.), but this very low chance probability is misleading, for some patients were readmitted more often than others and so had a greater influence on the overall distribution of readmission diagnoses. In fact, the striking difference obtained is largely due to the fact that, by chance, a small number of summer-born schizophrenics were readmitted with diagnoses other than schizophrenia a very large number of times. When the distributions of the different patterns of readmission diagnoses observed in individual patients were compared in the summer-born and the winter-born (i.e. counting each patient once only), the difference between the two was much less striking ($\chi^2=6.31$ and $P=0.043$). However, there is still a significant difference between the readmission diagnoses of summer- and winter-born schizophrenics, as there was for their first-admission diagnoses. There was no significant difference between winter- and summer-born affectives on any of the 13 items. In particular, there was no difference in the distributions of either their initial or their readmission diagnoses.

Scottish national data

Since 1963, information has been collected on all admissions to and discharges from all psychiatric hospitals and units in Scotland by the Common Services Agency of the Scottish Health Service. This information (SMR4) is less extensive than that recorded by the EPCR but covers a population ten times as large – the 5 200 000 inhabitants of Scotland. Comparisons between winter- and summer-born schizophrenics and winter- and summer-born affectives were carried out in exactly the same way as with the EPCR patients. That is, both comparisons were restricted to patients aged 15–39 inclusive at the time of admission, with known date of birth and with no known previous in-patient psychiatric care; schizophrenia was defined as a discharge

TABLE II
Comparisons between winter- and summer-born Edinburgh patients

	Schizophrenics		*Affectives*	
	Winter-born	*Summer-born*	*Winter-born*	*Summer-born*
Number of patients	222	349	198	277
Mean age on first admission	27.9	27.5	29.5	29.4
Sex (% male)	60.8	62.8	44.4	40.8
Marital status				
Never married (%)	76.6	74.8	45.5	41.9
Married (%)	14.9	19.2	46.0	46.6
Other (%)	8.6	6.0	8.6	11.6
Admission status (% informal)	81.1	78.8	85.9	89.2
Duration of first admission (weeks)				
Median	5.9	8.3	5.0	5.2
Mean	151.1	146.4	7.4	16.9
Number of readmissions				
Median	0.0	1.0	0.0	0.0
Mean	1.5	1.8	1.1	0.9
Cumulative duration of all admissions (weeks)				
Median	12.0	16.4	8.1	8.1
Mean	179.7	168.1	13.8	24.0

Notes. None of the differences between winter- and summer-born patients is statistically significant. For age, student's t test was used. For duration of first admission, number of readmissions and cumulative duration of all admissions, the non-parametric Kolmogorov-Smirnov Two Sample Test was used because of their skewed distributions. For all other variables the χ^2 test was used.

The great disparity between the median and mean durations of schizophrenic admissions is due to a small number of patients whose first admission had lasted for up to 30 years.

TABLE III
Discharge diagnoses of winter- and summer-born Edinburgh patients: first admissions

Schizophrenics[1]	*Winter-born (n = 222)*	*Summer-born (n = 349)*
Simple (%)	7.7	5.7
Hebephrenic (%)	6.3	7.7
Catatonic (%)	1.8	1.7
Paranoid (%)	32.0	25.5
Acute episode (%)	5.9	10.6
Latent (%)	0.0	2.0
Schizoaffective (%)	9.0	4.6
Other (%)	9.0	11.7
Unspecified (%)	28.4	30.4

Affectives[2]	*Winter-born (n = 198)*	*Summer-born (n = 277)*
Manic-depressive, manic (%)	38.9	31.0
Manic-depressive, depressed (%)	48.0	56.0
Other (%)	8.6	10.1
Unspecified (%)	4.5	2.9

1. $\chi^2 = 14.6$ and $P = 0.044$ with 7 d.f.
2. $\chi^2 = 3.79$ and $P = 0.4$ with 4 d.f.

Note. The number of diagnostic categories shown in Tables III, IV, VI and VII do not always correspond with the number of degrees of freedom shown in the χ^2 comparisons. This is because some diagnostic categories have been combined for ease of presentation.

diagnosis of ICD 295 and affective psychosis as a discharge diagnosis of ICD 296; and winter-born was defined as having a birth date in January to March and summer-born as having a birth date in June to October.

The comparisons were based on SMR4 data for the 12-year period 1970–1981 and involved ten of the 13 items listed in Table I (all except 5, 6 and 8). Comparison of the number of readmissions, the cumulative duration of all admissions (first admissions and readmissions combined) and the discharge diagnoses of readmissions was more difficult than it had been for the Edinburgh patients because, unlike the EPCR returns, the SMR4 returns for the successive admissions of a single individual are not normally linked together. A special linkage exercise therefore had to be mounted using surname, date of birth, sex and the hospital's case reference number as identifying data. By this means almost all readmissions to the hospital to which the patient was originally admitted would have been identified. The majority of admissions to other psychiatric hospitals or units in Scotland should also have been identified, though women whose surname changed in the interim, and men or women whose surname was misspelt or whose date of birth was wrongly recorded might well have been missed. Admissions to hospitals in other parts of the United Kingdom would, of course, also have been missed. In order to restrict to manageable numbers the total number of SMR4 returns needing to be scanned, only admissions with a discharge diagnosis of schizophrenia (ICD 295) or affective psychosis (ICD 296) were included in the linkage exercise. As a result, readmissions with other diagnoses, which in the EPCR pilot study had amounted

TABLE IV

Discharge diagnoses of winter- and summer-born Edinburgh patients: readmissions

Schizophrenics[1]	*Winter-born*	*Summer-born*
Total number of readmissions	337	637
Diagnostic distribution of these:		
schizophrenia (%)	84.9	67.0
affective psychoses (%)	5.3	16.8
other diagnoses (%)	9.8	16.2
Number of individual patients	112	188
Readmission diagnoses of these:		
schizophrenia only (%)	72.3	58.5
schizophrenia and other diagnoses (%)	17.9	23.4
other diagnoses only (%)	9.8	18.1

Affectives[2]	*Winter-born*	*Summer-born*
Total number of re-admissions	212	248
Affective psychosis:		
manic–depressive manic (%)	34.9	34.3
manic–depressive depressed (%)	18.4	24.6
other affective (%)	19.3	11.3
Schizophrenia (%)	6.6	6.0
All other diagnoses (%)	20.7	23.8

1. $\chi^2 = 6.31$ and $P = 0.043$ with 2 d.f.
2. $\chi^2 = 7.51$ and $P = 0.11$ with 4 d.f.

to 14% of all the schizophrenics' readmissions, were not identified.

Results

The results are shown in Tables V to VII. None of the comparisons set out in these three tables involves a difference between summer- and winter-born patients which is statistically significant even at the 10% level. In particular, there is no significant difference between the first-admission diagnoses of winter- and summer-born schizophrenics. The proportion of patients with a diagnosis of paranoid or schizo-affective schizophrenia is marginally higher in the winter-born (23.% *v.* 21.5%), but even this modest difference is entirely due to the 274 Royal Edinburgh Hospital patients in the Scottish sample. When the comparison was repeated after removing these 274 patients (10.3% of the total), the diagnoses of winter- and summer-born patients were virtually identical ($\chi^2 = 4.01$ and $P = 0.91$ with 9 d.f.). There is no significant difference between the readmission diagnoses of summer- and winter-born schizophrenics either. So again the findings of the EPCR study are not confirmed. However, when Royal Edinburgh Hospital and other Scottish patients were examined separately, there was a striking tendency in the former for a higher proportion of the readmissions of summer-born than of winter-born patients to be given a diagnosis of affective psychosis (19% *v.* 5%). So this does at least confirm that the EPCR finding described above was not generated by a programming error.

Although the season-of-birth effect has never been studied in Scottish schizophrenics, its existence can be demonstrated and its approximate magnitude calculated from these data. For schizophrenia the 90.25 days between 1 January and 31 March produced 1057 first admissions,

TABLE V

Comparisons between winter- and summer-born Scottish patients

	Schizophrenics		*Affectives*	
	Winter-born	*Summer-born*	*Winter-born*	*Summer-born*
Number of patients	1057	1596	933	1487
Mean age at first admission	26.3	26.2	29.3	29.2
Sex (% male)	62.1	59.5	32.6	34.9
Marital status				
Never married (%)	68.4	67.6	32.0	34.0
Married (%)	25.0	26.4	60.9	58.6
Other (%)	6.6	6.0	7.1	7.5
Admission status (% informal)	77.8	77.1	87.8	89.4
Duration of first admission (weeks)				
Median	5.9	5.7	4.1	4.1
Mean	10.3	10.4	6.1	6.1
Number of readmissions				
Median	0.0	1.0	0.0	0.0
Mean	1.4	1.4	0.7	0.7
Cumulative duration of all admissions (weeks)				
Median	10.9	10.6	6.0	5.6
Mean	29.6	31.9	13.0	12.3

Notes. None of the differences between winter- and summer-born patients is statistically significant. For sex and for marital, admission and employment status the χ^2 test was used. The Kolmogorov-Smirnov Two Sample Test was used for age, duration of first admission, number of readmissions and cumulative duration of all admissions, because of their skewed distributions.

TABLE VI

Discharge diagnoses of winter- and summer-born Scottish patients: first admissions

Schizophrenics[1]	*Winter-born (n = 1057)*	*Summer-born (n = 1596)*
Simple (%)	5.0	5.3
Hebephrenic (%)	6.1	6.5
Catatonic (%)	2.0	1.9
Paranoid (%)	18.7	18.0
Acute episode (%)	4.8	6.0
Latent (%)	0.3	0.6
Schizoaffective (%)	4.3	3.5
Other (%)	3.8	4.7
Unspecified (%)	54.9	53.6

Affectives[2]	*Winter-born (n = 933)*	*Summer-born (n = 1487)*
Manic–depressive manic (%)	22.1	20.0
Manic–depressive depressed (%)	62.9	66.6
Other (%)	5.3	4.4
Unspecified (%)	9.8	9.1

1. $\chi^2 = 6.14$ and $P = 0.73$ with 9 d.f.
2. $\chi^2 = 5.44$ and $P = 0.61$ with 7 d.f.

TABLE VII

Discharge diagnoses of winter- and summer-born Scottish patients: readmissions

Schizophrenics[1]	*Winter-born*	*Summer-born*
Total number of readmissions[3]	1409	2216
Diagnostic distribution of these:		
schizophrenia (%)	89.3	89.9
affective psychosis (%)	10.7	10.1
Number of individual patients	499	781
Readmission diagnoses of these:		
schizophrenia only (%)	85.6	86.0
schizophrenia and affective psychosis (%)	8.4	8.2
affective psychosis only (%)	6.0	5.8

Affectives[2]	*Winter-born*	*Summer-born*
Total number of readmissions	646	1004
Affective psychosis:		
manic–depressive manic (%)	23.4	23.1
manic–depressive depressed (%)	36.8	37.7
other (%)	9.6	10.2
unspecified (%)	11.5	11.4
Schizophrenia (%)	18.7	17.6

1. $\chi^2 = 0.05$ and $P = 0.98$ with 2 d.f.
2. $\chi^2 0.49$ and $P = 0.97$ with 4 d.f.
3. These totals do not include the 76 readmissions still in hospital on 31 December 1981, nor do they include any readmissions with diagnoses other than schizophrenia or affective psychosis.

while the 153 days between 1 June and 31 October produced 1596. The admission rate of winter-born relative to that of summer-born patients is therefore

$$\left(\frac{1057}{90.25}\right) + \left(\frac{1596}{153}\right) = 1.1228$$

For affectives the 90.25 days in January to March produced 933 first admissions while the 153 days in June to October produced 1487. The admission rate of the winter-born relative to the summer-born is therefore

$$\left(\frac{933}{90.25}\right) \div \left(\frac{1487}{153}\right) = 1.0637$$

Patients admitted to hospital in the 12-year period 1970–1981 who were 15–39 at the time of admission would have been born after 1929 and before 1967. Since 1931, the annual reports of the Registrar General for Scotland have given tables of the number of births registered each month, and it is apparent from these that for most of the 37 years between 1930 and 1966 the birth rate was higher in January to March than in June to October. Taking the 3 years 1940, 1950 and 1960 as a sample of that 37-year period (and remembering that 1940 and 1960 were leap years), it was calculated from these tables that the ratio of the Scottish birth rate in January to March to that in June to October was 1.033. The excess admission rate of winter-born schizophrenics relative to the summer-born is therefore

$$\left[\left(\frac{1.1228}{1.033}\right) - 1\right] \times 100 = 8.7\%$$

and the excess admission rate of winter-born affectives relative to the summer-born is

$$\left[\left(\frac{1.0637}{1.033}\right) - 1\right] \times 100 = 3.0\%$$

Discussion

The comparison of the months of birth of Scottish patients and those of the general Scottish population carried out here suggests that there is a 9% excess of schizophrenic births and a 3% excess of affective psychosis births in the first 3 months of the year. The season-of-birth effect is therefore demonstrable in Scotland, as in England and Wales and most other countries in the temperate latitudes of the northern hemisphere. The main purpose of the study, however, was to detect differences between winter- and summer-born patients rather than to demonstrate yet again an excess of winter-born schizophrenics.

The ratings used to compare winter- and summer-born patients were routine statistical returns with the familiar shortcomings of such data. They were of unknown reliability, sometimes incomplete, and the diagnoses they incorporated were made, often by junior medical staff, to unspecified and almost certainly varying criteria. However, all these sources of low reliability and bias should affect winter- and summer-born patients to the same extent. The deficiencies might obscure genuine differences, but it is difficult to see how they could generate spurious differences between groups of patients born at different times of the year.

Although winter- and summer-born patients were compared on several different variables, no statistically significant differences were found between winter- and summer-born affectives, and the only significant differences between winter- and summer-born schizophrenics were differences in the distributions of both first-admission and readmission diagnoses in the Edinburgh patients. These diagnostic differences were only significant at the 5% level; they were not accompanied by any difference in age of onset, readmission rate or total duration of hospitalisation; nor are they easily reconciled with one another. Moreover, no analogous differences were found in the second study based on patients drawn from the whole of Scotland, despite the considerably larger numbers of patients available (there were 1057 winter-born Scottish schizophrenics, compared with 222 in the EPCR study). It seems likely, therefore, that these diagnostic differences were generated by chance. It is true that in the Scottish study diagnoses other than schizophrenia or affective psychosis were not identified by the linkage program used to detect readmissions, and that if they had been a significant difference in the range of readmission diagnoses might have emerged. But the major component of the difference between summer- and winter-born patients in the EPCR comparison had been in the proportion of diagnoses changed to affective psychosis, and there was no hint of any difference in this respect in the Scottish material.

Dalen (1975) and Pulver *et al* (1983) have reported previously that the excess of winter births is greater in, or restricted to, schizophrenics with a relatively good prognosis. However, there is no evidence here, in either the EPCR or the Scottish material, that winter-born patients have a better prognosis than the summer-born. The duration of the original admission, the number of readmissions and the cumulative duration of all admissions of winter- and summer-born patients are all indistinguishable. For all three variables, Kolmogorov–Smirnov tests failed to reveal a difference significant even at the 10% level. It is true that, on average, our patients were only followed for a period of 5–6 years after their original admission, and that significant differences might have emerged if the observation period had been longer. Even so, the combination of these three variables ought to provide a fairly sensitive index of prognosis, certainly more sensitive than those used by Dalen (1975) and Pulver *et al* (1983), who both relied entirely on the duration of the original admission, and in the Scottish study at least patient numbers were substantial. Nor do our data provide any support for the suggestion of Watson *et al* (1984) that the season-of-birth effect predominates in unmarried schizophrenics.

We conclude, therefore, that we have failed to demonstrate any convincing difference between winter- and summer-born schizophrenics, or even to find any strong suggestions that such differences are there to be found. But for a variety of reasons this does not necessarily mean that there are none. In particular, it is important that the possibility, raised by the findings of Kinney & Jacobsen (1978) and Shur (1982), that there is no excess of winter births in schizophrenics with a positive family history should be investigated further.

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P300 and CT Scan in Patients with Chronic Schizophrenia

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Twenty chronic male schizophrenic subjects aged 30–50 years were examined by an auditory event-related potential procedure for the evaluation of the P300 component, a CT scan and a neuropsychological test battery. The P300 latency was increased and its amplitude was reduced. CT scan measures showed lateral and third ventricle enlargement, and there was a global neuropsychological impairment. Poor neuropsychological performances were consistently associated with delayed P300 latencies, but not with CT scan measures. Ventricular enlargement was more pronounced among subjects with a negative family history for major psychiatric disorders.

There is consistent evidence (Heaton *et al*, 1978) that chronic schizophrenic subjects perform poorly on neuropsychological tests. Whether there may be specific patterns linked to a deteriorating course and affecting all diagnostic subgroups remains unclear, although some authors have suggested that characteristic features are present (De Wolfe *et al*, 1971; Davis *et al*, 1972; Liddle & Crow, 1984), and others have even proposed anatomo-functional models which could explain the spectrum of the 'negative' symptomatology (Levin, 1984). It has been long suspected that schizophrenic cognitive impairment may not have an exclusively 'functional' origin. 'Soft' neurological signs have been repeatedly described in this condition (Rochford *et al*, 1970), as well as histopathological (Fisman, 1975), pneumoencephalographic (Haug, 1962), electroencephalographic (EEG) (Itil *et al*, 1972) and more recently, computerised tomographic (CT) scan (Johnstone *et al*, 1976) and regional cerebral blood flow (Ariel *et al*, 1983) abnormalities.

Brain morphology as investigated with CT scan has been the subject of many studies, which have given inconclusive results regarding the localisation of abnormal findings: enlargement of lateral ventricles (Andreasen *et al*, 1982*b*; Owens *et al*, 1985), third ventricle enlargement (Tanaka *et al*, 1981), fourth ventricle enlargement (Lippman *et al*, 1982) and the association of structural abnormalities with clinical variables (Andreasen *et al*, 1982*a*; Nasrallah *et al*, 1982; Rieder *et al*, 1983). Some authors have reported normal findings (Jernigan *et al*, 1982; Benes *et al*, 1982).

Another approach to the problem of cognitive deficit in schizophrenia has been advanced by some authors (Baribeau-Braun *et al*, 1983; Pfefferbaum *et al*, 1984), who studied the late positive component of event-related potentials (ERPs) – P3 or P300 – and described an increase in latency in schizophrenics, which is thought to be a characteristic of demented patients (Goodin *et al*, 1978; Leppler *et al*, 1984).

In the present study a multimodal approach has been used to investigate possible relationships between schizophrenic deterioration and clinical, neuropsychological, neuroradiological and neurophysiological parameters.

Method

Subjects

Twenty male subjects were selected among the long-stay patients (at least 5 years' hospitalisation) of San Colombano al Lambro Fatebenefratelli Psychiatric Hospital according to the following criteria.

The first criterion was diagnosis of 'schizophrenic disorder – chronic course' according to DSM-III criteria (American Psychiatric Association, 1980). Five subjects were classified as 'paranoid type', four as 'disorganised type' and eleven as 'residual type'. Eight subjects had a positive family history (first-degree relatives) for schizophrenia (six) or major depression requiring hospitalisation (two). They were evenly distributed among diagnostic subtypes.

The second inclusion criterion was monotherapy with haloperidol 4–6 mg/day p.o. for at least 6 months preceding the investigation. The third criterion was absence of other psychiatric or neurological diseases.

The fourth criterion was ability to co-operate with the test procedures. A preliminary study showed that most chronic schizophrenics with a memory quotient (MQ) score of 70 or less on the Wechsler Memory Scale were not able to perform the counting task of the ERP paradigms. An MQ score of at least 75 was therefore established as an inclusion criterion.

The patients were aged between 30 and 50 years (mean, 42 years); they had had 5–8 years of education.

Control groups

It was not possible to perform the ERP procedures, the neuropsychological tests and the CT scan on a single control group. Therefore two control groups, each of 20 age-matched male subjects, were selected. The first group consisted of healthy volunteers who were submitted to the ERP procedures and to the neuropsychological test battery. The educational status of this group (6–8 years education) was similar to that of the patients. The second group consisted of hospitalised patients diagnosed as having chronic tension headache (six), migraine (eight), Bell's palsy (three) or neuroses (three). CT scans were performed on the second group.

Procedure

The patients were submitted to the ERP procedures in the late hours of the morning. Within 7 days they were tested with the neuropsychological battery, and within a period of 6 months, 18 of them received a CT scan. One patient did not give his informed consent for the CT scan, and the other died because of pneumonia.

Event-related potentials

The ERP procedure took place in a sound-attenuated, electrically shielded cabin. Surface electrodes were placed as follows:

Channel 1: vertex – linked earlobes
Channel 2: internal chantus – infraorbital region (eye movement and blink contaminated trials were automatically excluded)
Fpz was grounded.

The EEG was processed by an OTE 1239 Neuroaverager (512 words; band width 0.5 – 50 Hz; time base 500 ms; bin width 0.98 ms). The stimuli (800 Hz tone bursts; duration 30 ms; rise/fall time 10 ms) were delivered by an Alvar Pluristim IV stimulator through two small loudspeakers placed 20 cm away on the right and left of the subject's head.

The auditory ERP procedure included the following four paradigms, three of which (2–4) required selective attention. In paradigm 1 the subject was instructed to count binaurally presented tones. Stimulus intensity was 70 dB SL and inter-stimulus interval (ISI) was 2 s; 20 stimuli were presented.

In paradigm 2 the subject was instructed to count only the stimuli presented to the right ear. In this paradigm 22 (rare) tones presented to the right ear alternated in a pseudo-random order with 110 (frequent) tones presented to the left ear. Stimulus intensity was 70 dB SL and ISI was 2 s.

Paradigm 3 was specular to paradigm 2: the subject had to count only the rare stimuli presented to the left ear.

In paradigm 4, the subject was instructed to count only the 'loud' stimuli. In this paradigm 22 binaural (rare) tones with an intensity of 70 dB SL alternated in a pseudo-random order with 110 (frequent) tones of 50 dB SL. ISI was 2 s.

In paradigm 1, responses to all stimuli were averaged, while in paradigms 2–4, only responses to rare stimuli were averaged.

The averaged signals were plotted by means of a Phillips 8040 X–Y recorder.

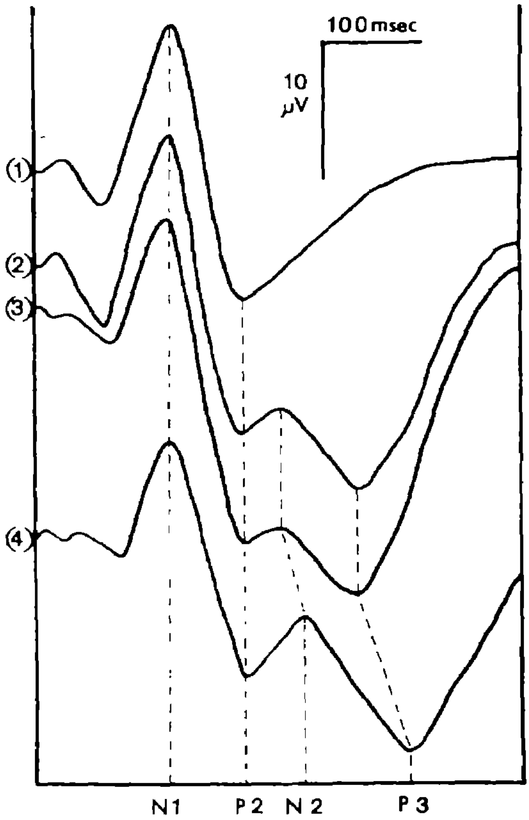

FIG. 1 Example of event-related potentials obtained in paradigms 1–4 in a control subject. The dashed lines identify the peaks N1, P2, N2 and P3. Negativity upwards.

Peaks were visually identified by adopting the following criteria: N1 was the first large negative peak falling in the interval 75–150 ms; P2 was the subsequent positive peak falling in the interval 150–250 ms. These two peaks were evident in all paradigms (Fig. 1). In selective-attention paradigms two further negative–positive waves, usually labelled N2 and P3 (or P300), appeared (see traces 2–4 of Fig. 1). P3 latency fell in the interval 320–480 ms, and N2 was the greatest negative peak between P2 and P3. Latency values of peaks N1, P2, N2 and P3, as well as peak-to-peak amplitudes N1–P2, P2–N2, N2–P3 and N1–P3, were considered for data analysis.

Neuropsychological battery

The following psychological tests were used: Wechsler Memory Scale, form I (WMS); Wechsler Bellevue I (WB); Raven Progressive Matrices '38 (PM 38); and the Toulouse-Pieron (TP). The TP is a test of attention in which the subject is presented with a large grid of eight similar geometric figures and asked to indicate all occurrences of four particular figures by drawing lines through them. Only quantitative performance has been analysed, according to Hiltmann (1960).

The analysis of data was performed on raw scores of single items and on age-corrected indices.

CT scan

In the CT scan, slices of 10 mm were performed from base to vertex (scanner EMI 1010). The following dimensions were measured: (a) the distance between the most lateral portions of the frontal horns; (b) the width of the lateral ventricles at the level of the caudate nuclei; (c) the dimensions of the four largest cortical sulci visible in the three upper slices; (d) the width of the third ventricle at the level of Monroe's foramina.

As described by Huckman *et al* (1975), the two ventricular dimensions were added and a single measure of ventricular size (V-index) was derived; the sum of measures of the four largest sulci supplied the degree of cortical atrophy (C-index).

Neurophysiological and neuroradiological measures were scored independently by two of the authors (A.R. and L.G. respectively), blind to the identities and diagnostic categories; neuropsychological tests were performed by one of the authors (S.M.), who was unaware of the results obtained by CT scan and EEG.

Statistical analysis

Statistical analysis was performed by comparing mean values of independent samples (Student's t test), by calculating Pearson's (r) and Spearman's (r_s) correlation coefficients and by linear regression analysis. A principal component analysis (PCA) (Lebart *et al*, 1977) was performed on Spearman's coefficients obtained from correlations of neuropsychological scores, and the main factors that were derived were entered as independent variables in multiple regression analysis.

Results

ERPs

Both in control subjects and in schizophrenic patients, well defined N1-P2 shaped potentials were obtained in paradigm 1, and N1-P2-N2-P3 shaped potentials were obtained in paradigms 2–4 (Fig. 1). In some cases N2 was recognisable only as a step on a large N1-P3 wave. In these cases P2-N2 amplitude was zero.

Mean latency and amplitude values are reported in Table I. A highly significant latency increase was observed only for peak P300, but it was also evident for N2. Latencies of N1 and P2 did not differ between schizophrenics and controls.

TABLE I

Mean values of peak latencies and peak-to-peak amplitudes of schizophrenic (S) and control (C) subjects

Peak	*Subjects*	*Latencies (ms)* *Paradigm 1*	*Paradigm 2*	*Paradigm 3*	*Paradigm 4*
N1	C	112	122	120	121
	S	119	129	127	128
P2	C	207	213	209	201
	S	212	214	210	215
N2	C	—	254	253	265
	S	—	271*	273**	286**
P3	C	—	326	334	364
	S	—	369***	377***	418***

Peak to peak	*Subjects*	*Amplitudes (μV)* *Paradigm 1*	*Paradigm 2*	*Paradigm 3*	*Paradigm 4*
N1-P2	C	20.4	22.2	23.1	20.0
	S	16.9*	17.4*	16.5**	15.1**
P2-N2	C	—	4.2	5.5	9.9
	S	—	6.4	7.7	10.4
N2-P3	C	—	9.8	10.6	13.4
	S	—	9.7	9.9	10.8
N1-P3	C	—	28.7	27.2	23.7
	S	—	20.9***	19.3***	16.0***

*$P<0.05$; **$P<0.01$; ***$P<0.001$.

The most consistent ($P<0.001$) amplitude reductions in schizophrenics were found for N1-P3, but N1-P2 amplitude was also reduced in the selective-attention paradigms (2–4), as well as in the simple counting paradigm (1). P2-N2 and N2-P3 amplitudes did not differ significantly. This may be at least partially attributed to a greater relative variation. The coefficients of variation (CV) of these two amplitudes had values ranging from 45% to 75%, whereas for N1-P2 and N1-P3 the CV values ranged from 25% to 40%. This greater variability of N2-related interpeak amplitudes is probably connected with interindividual differences in N2 morphology (separate peak or only step on N1-P3 deflection). Therefore P2-N2 and N2-P3 amplitudes were excluded from further analysis.

Eleven patients exhibited prolonged (+2 s.d.) P300 latencies in paradigm 4, and nine of them had prolonged latencies of P300 also in paradigm 2 and/or 3.

In control subjects, P300 latency in paradigm 4 was the only measure which correlated with age (latency = 249 + 3.05 × age, $P<0.025$). No correlation with age was found in the patient group.

Neuropsychological battery

Controls performed better than patients on each item and scored higher on each index of the neuropsychological battery. The significance was always very high ($P<0.001$), except for WB digit span and block design ($P<0.01$).

Since most of the items were correlated with each other, the scores were submitted to a principal component analysis (PCA) separately for the schizophrenic and the control group in order to obtain the main orthogonal factors to be correlated with other variables. A detailed analysis of these results is beyond the aim of the present paper and is presented separately (Romani *et al*, 1987).

Briefly, both in control subjects and in the schizophrenic group, the first two factors accounted for about 50% of the variance or more. The first factor (C1), which explained

38% of the variance in the control group and 37% in the schizophrenic group, was in both cases a general, unipolar factor which correlated significantly with almost every item of the neuropsychological battery.

The second (C2) was a bipolar factor, the relevance and meaning of which showed some differences between the two groups. In the control group, where it could explain 26% of the variance, it had the largest positive loadings on non-verbal items such as WMS picture reproduction, WB picture arrangement, picture completion, block design, object assembly and PM 38, while the largest negative loadings lay on more verbal-related items such as WMS associations, WB information, comprehension and vocabulary. TP also had a high negative loading. Among schizophrenics the meaning of C2 (which accounted for only 12% of the variance) was less evident. The largest positive loadings lay on WB digit-symbol and object assembly, while WMS associations, logic memory and WB picture arrangement had the largest negative loadings. This factor seems to tap a dimension related on the one hand to spatial-perceptive functions (in a manner similar to but not as evident as that seen in controls) and on the other hand to a verbal as well as non-verbal ability to use logic relationships in more integrated contexts.

CT scan

Mean values of CT scan measures are reported in Table II. Among the patient group, abnormal (+2 s.d.) values of both V-index and third ventricle width were found in three subjects, abnormal values of V-index alone in two subjects, and abnormal values of C-index in one subject. In the control group there was no correlation between the three measures, whereas in the patient group the measures were correlated as follows: V-index with C-index, $r_s = 0.64$, $P < 0.01$; C-index with third ventricle, $r_s = 0.67$, $P < 0.01$; V-index with third ventricle, $r_s = 0.78$, $P < 0.001$.

TABLE II
Mean values of CT scan measures for schizophrenics (S) and controls (C)

Subjects	*V-index*[1]	*C-index*[2]	*3rd vent.*[3] *(mm)*
C	12.8	2.8	1.1
S	14.3*	3.3	1.6**

1. Ventricular index.
2. Cortical index.
3. Width of third ventricle.

*$P < 0.05$; **$P < 0.01$.

Relations between variables

ERPs and neuropsychological battery

Multiple regressions were performed between the factors C1 and C2 (derived from the PCA of the neuropsychological scores) and single ERP measures, which were considered as dependent variables. Possible relations with neuropsychological measures could be expected in the case of late components only, (N2, P3 latencies, N1–P3 amplitudes), and not earlier, primarily stimulus-related components (N1, P2 latencies, N1–P2 amplitudes). Therefore two opposite hypotheses were tested. Multiple determination coefficients (R^2) were considered significant when greater than 0.42 ($P < 0.01$). Also, R^2 values greater than 0.30 ($P < 0.05$) are reported, as they may show 'borderline' significant associations. As C1 and C2 are orthogonal factors, R^2 can be expressed as the sum of their single contributions.

For control subjects, no significant relation was found between ERPs and neuropsychological measures.

For schizophrenic subjects, significant R^2 values were found with P3 latency in paradigm 4 ($R^2 = 0.58$; 0.32 explained by C1, and 0.26 by C2), and with P3 latency in paradigm 2 ($R^2 = 0.45$; 0.35 explained by C1, and 0.10 by C2). There was only a 'borderline' association of P3 latency in paradigm 3 with neuropsychological measures ($R^2 = 0.35$; 0.22 explained by C1, and 0.13 by C2), and similar associations were observed for N1–P2 amplitude in paradigm 4 ($R^2 = 0.39$; 0.30 explained by C1, and 0.09 by C2) and in paradigm 2 ($R^2 = 0.39$; 0.33 explained by C1, and 0.06 by C2). In each of these relations a prolonged P3 latency or a reduced N1–P2 amplitude was associated with low values of the general factor C1 (globally poor neuropsychological performance) and with positive values

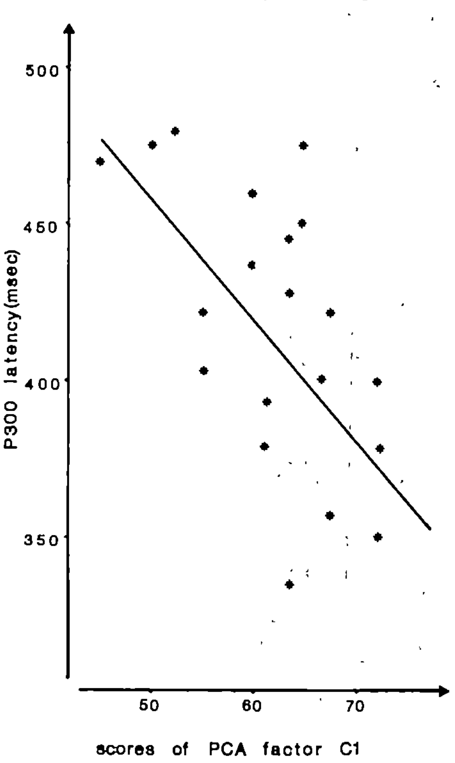

FIG. 2 Relation of P300 latency in paradigm 4 to neuropsychological performance expressed by PCA general factor C1. The regression line (latency = 630–3.44C1; $P < 0.01$) is also shown.

of the bipolar factor C2 (better on spatial–perceptive tasks than on tasks requiring more integrated verbal and non-verbal logic abilities).

It appears that the PCA factor which was most consistently associated with neuropsychological measures was the general factor, C1. For C2, the only significant relationship ($P<0.01$) was with P300 latency in paradigm 4. The relationship of P300 latency in paradigm 4 with factor C1 is represented in Fig. 2 as a simple regression. Considering the correlation coefficients between neurophysiological measures and scores of single items as indices, the most significant values ($P<0.001$) were found between P300 latency in paradigm 4 and WMS MQ ($r=0.78$), logic memory ($r_s=0.76$) and WB performance IQ ($r=0.70$).

ERPs and CT scan

Only 'borderline' significant ($0.01<P<0.05$) correlations between ERPs and CT scan were observed, and only in the case of N1–P2 amplitudes. In paradigm 1 this amplitude measure correlated with V-index ($r=0.49$) and third ventricle ($r_s=0.50$), and in paradigm 4 it correlated with C-index ($r_s=0.47$) and third ventricle ($r_s=0.52$). The correlation coefficients were always positive.

CT scan and neuropsychological battery

No correlation between CT scan and neuropsychological battery was found.

Differences between diagnostic subgroups

ERPs

P300 latency in paradigm 4 was significantly greater in disorganised schizophrenic subjects (474.4 ms) than in either paranoid (410.5 ms, $P<0.02$) or residual schizophrenics (395 ms, $P<0.001$), in spite of the fact that the disorganised subjects were significantly younger (37 years) than paranoid (43.5 years) and residual subjects (44 years). Disorganised schizophrenics also had smaller amplitudes than residual schizophrenics: N1–P2, 12.8 μV *v.* 19 μV ($P<0.05$) in paradigm 1 and 11 μV *v.* 18.2 μV ($P<0.02$) in paradigm 2; N1–P3, 14.2 μV *v.* 23.2 μV ($P<0.02$) in paradigm 2 and 11 μV *v.* 18.9 μV ($P<0.02$) in paradigm 4.

Neuropsychological battery

Disorganised schizophrenics performed worse than residual schizophrenics on the following items: WMS MQ 88 *v.* 109 ($P<0.01$); information 4.2 *v.* 5.3 ($P<0.05$); WB verbal IQ 99 *v.* 107 ($P<0.02$); performance IQ 85.4 *v.* 94.6 ($P<0.01$); full scale IQ 87.2 *v.* 101.5 ($P<0.01$); vocabulary 9.6 *v.* 14 ($P<0.01$); digit span 7.2 *v.* 8.8 ($P<0.05$); arithmetics 5.4 *v.* 6.7 ($P<0.05$); picture arrangement 4.4 *v.* 8.2 ($P<0.01$); picture completion 8.4 *v.* 9.7 ($P<0.01$). Also the general factor C1 derived from the PCA was lower in disorganised than in residual schizophrenics: 54.5 *v.* 64.8 ($P<0.01$). The bipolar factor C2 did not differ significantly between diagnostic subgroups.

CT scan

There was no difference in CT scan measures between diagnostic subgroups.

Differences between schizophrenics in terms of family history

ERPs and neuropsychological battery

When subjects were divided according to whether their family history was positive or negative, no differences in ERP and neuropsychological measures were found between the groups.

CT scan

The mean V-index was greater ($P<0.05$) among subjects with a negative family history (15.5) than among subjects with a positive family history (12.8). There was no difference in mean values of C-index and third ventricle width, but the three subjects with a dilated third ventricle all had a negative family history.

Discussion

A global impairment of intellectual function seems to be a feature of our long-stay schizophrenic patients and confirms previous reports (Heaton *et al*, 1978). Items primarily related to attention (i.e. Toulouse–Pieron), memory (digit span) and more integrated verbal (logic memory, arithmetics) and non-verbal (Raven's Progressive Matrices) functions are equally compromised; moreover, patients included in the diagnostic subgroup of disorganised schizophrenia obtained the lowest scores.

Although the selection criteria excluded the possibility that our patients might have suffered from mental subnormality, and the education of patients and controls did not differ, a control group composed of healthy volunteers might not reveal illness-specific patterns, as the long period of hospitalisation would itself represent an interfering variable (Smith, 1964), possibly more relevant than neuroleptic treatment itself (Small *et al*, 1972). Indeed, Taylor & Abrams (1984) reported in a recent study on cognitive impairment in schizophrenia that the differences in neuropsychological performance were unrelated to neuroleptic and other treatments. Also, Robertson & Taylor (1985) found neither significant differences nor even trends that could differentiate the neuropsychological performance of treated and untreated schizophrenics.

ERP measures confirm previous observations of reduced amplitudes (Roth *et al*, 1972; Verleger & Cohen, 1978; Duncan-Johnson *et al*, 1984) as well as more recent findings of prolonged latencies (Pfefferbaum *et al*, 1984) in schizophrenics. The latter authors examined patients both on and off neuroleptic medication and did not report any difference; therefore neuroleptic treatment is unlikely to account for these ERP abnormalities. Amplitude reduction does not seem to be a specific feature of the P300 component, as N1–P2 is also reduced in the three paradigms involving selective attention; and this phenomenon does not seem to be restricted to such complex situations, as it is evident in paradigm 1, in which only one kind of stimulus is presented. An analogous finding of N1 amplitude reduction in schizophrenic patients in auditory ERP paradigms which did not involve selective attention has been described by Roth *et al* (1980) for relatively long interstimulus intervals.

In contrast, latency increase involves exclusively the late components (P300 and, to a lesser degree, N2), necessarily linked to the more complex paradigms. ERPs obtained from paradigms involving different selective-attention tasks show some specific features (see also Romani *et al*, 1984). P300 latency is some 10% greater in the intensity discrimination paradigm (4) than in paradigms where a right–left discrimination is requested (2 and 3), both in control subjects and in schizophrenics (in each group $P<0.001$). Moreover, in controls a significant correlation of P300 latency with age was observed only in paradigm 4. Finally, this paradigm seems to be the one which is most sensitive in discriminating schizophrenic subjects from controls. These observations are probably not independent and suggest that mechanisms involved in discriminating this relatively small intensity difference (20 dB) on the one hand, and the side of origin of the stimuli on the other, may be partially different, the first process being more time-consuming and more influenced by age and by schizophrenic pathology.

Our CT scan measures are in agreement with the majority of reports about lateral ventricle dilatation in schizophrenia (Johnstone *et al*, 1978; Weinberger *et al*, 1979; Reveley *et al*, 1984; Owens *et al*, 1985). Although statistically significant, the dilatation was never severe (highest value of V-index 21) and involved about one-quarter of our subjects. Third ventricle dilatation was also present in our sample and confirms similar findings of other authors (Tanaka *et al*, 1981; Nybäck *et al*, 1982; Okasha & Madkour, 1982). Moderate cortical atrophy (C-index 7) was present in one patient.

The correlations between CT scan measures, and particularly between V-index and third ventricle width, observed exclusively among schizophrenic subjects, confirm similar findings described by Rieder *et al* (1983), although this author does not report third ventricle measures and does not make any statement about possible correlations in control subjects. The presence of these correlations suggests the possibility that only a subset of patients may exhibit a rather diffuse atrophy, involving primarily deep structures.

The observation that ventricular atrophy seems to be a specific feature of schizophrenic subjects with negative family history for major psychiatric disorders emerged as a collateral finding of our investigation, but is particularly intriguing considering recently published studies (Reveley *et al*, 1984; Schulsinger *et al*, 1984). These authors hypothesise on the basis of similar findings that genetic as well as environmental factors may lead to the development of schizophrenia. The subjects who have a high 'genetic load' (positive family history) may become schizophrenic with little or no environmental contribution, whereas those with a lighter 'genetic load' may require greater environmental influence (e.g. birth trauma), leading also to cerebral damage, as evidenced by signs of atrophy on CT scan. In our sample we were able to find a history of birth trauma in only one subject, who had ventricular enlargement, but other pre- or post-natal events may constitute environmental predisposing factors (Torrey & Peterson, 1976).

The analysis of the relations between measured variables revealed a consistent association between neuropsychological performance and P300 latency. The general neuropsychological factor derived from a principal component analysis – and correlated with almost every single item – accounts for the largest part of this relationship. The highest R^2 value is found in the multiple regression having P300 latency of paradigm 4 as dependent variable. This suggests that this intensity-discrimination paradigm may be more valuable in exploring cognitive functions than right–left discrimination paradigms.

The association of poor neuropsychological performance with prolonged P300 latency has been found exclusively in schizophrenics. We feel that this association is unlikely to be due to treatment, since neuroleptics have been shown to have little effect on both neuropsychological test results and ERPs (see above). Furthermore, drug treatment of our group was rigorously uniform. This relation between neuropsychological performance and P300 latency may be partially due to heterogeneity among diagnostic subgroups, since disorganised schizophrenics had the longest latencies of P300, and the lowest amplitudes, and scored lowest on most neuropsychological tests. The trend towards an association between neuropsychological PCA factors and N1–P2 amplitudes (lower amplitudes in patients with poorer neuropsychological performance) is only partially surprising, since N1 amplitude is known to be in some way related to attention (Baribeau-Braun *et al*, 1983).

The relationship between CT scan signs of atrophy and poor performances on neuropsychological tests reported by some authors (Golden *et al*, 1980; Donnelly *et al*, 1980) was not confirmed by other authors (Weinberger *et al*, 1979; Nasrallah *et al*, 1982) and was not found in our sample. This may be due to a different neuropsychological approach as well as to differences in patient population.

The few positive correlations found between N1–P2 amplitude and some CT scan measures are somewhat difficult to explain and need replication. Nevertheless, one has to consider that analogous findings have been described for the visual evoked potential of elderly and demented patients (Cosi *et al*, 1982).

In conclusion, our findings suggest that the two different instrumental approaches to the problem of schizophrenic cognitive function are clearly not equivalent. Both show some abnormal features, but while ERP measures, and particularly P300 latency, clearly parallel the actual neuropsychological performance, this is not the case with cerebral atrophy measures. The latter may be more related to the pathogenesis of the disease.

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Selective Decreases in MAO-B Activity in Post-Mortem Brains from Schizophrenic Patients with Type II Syndrome

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The activities of the A and B forms of the enzyme monoamine oxidase (MAO, E.C. 1.4.3.4) have been assessed with the substrates 5-hydroxytryptamine and benzylamine respectively in seven areas of the brains of 39 patients with schizophrenia and 44 control subjects. Whereas previous studies have found the enzyme unchanged in brain in schizophrenia, in this study there was a modest but significant decrease in the activity of MAO-B in frontal and temporal cortices and in amygdala. This decrease could not be accounted for by neuroleptic medication, age, sex or post-mortem variables. In a series of 22 patients who had been assessed in life, the reduction in MAO-B activity was found to be associated specifically with the presence of negative symptoms (flattening of affect and paucity of speech). The findings are therefore consistent with other evidence for structural and neurochemical change in the temporal lobe that have been associated with the type II (defect state) syndrome of schizophrenia. The change in enzyme activity is unlikely to be related to a change in monoamine metabolism but may reflect a disturbance in glial function. The change in MAO-B activity in brain in this study is confined to particular areas of brain and a subgroup of patients; it is thought to be entirely unrelated to earlier reports of reductions of enzyme activity in platelets, which are probably attributable to prolonged neuroleptic medication.

Monoamine oxidase (MAO, monoamine O_2 : oxidoreductase, E.C. 1.4.3.4) has been classified into MAO-A and MAO-B on the basis of sensitivity to inhibition by specific inhibitors. Type A MAO is relatively sensitive to inhibition by clorgyline (Johnston, 1968) and Type B MAO to inhibition by deprenyl (Knoll & Magyar, 1972). In this classification of MAO, 5-hydroxytryptamine (5HT) has been described as a specific substrate for MAO-A (Johnston, 1968) and benzylamine a specific substrate for MAO-B (Christmas *et al*, 1972).

The interest in MAO in relation to schizophrenia arose from reports that the enzyme in platelets has features in common with the enzyme in liver and brain (Robinson *et al*, 1968), and that activity in platelets is reduced in patients with chronic schizophrenia (Murphy & Wyatt, 1972). The latter finding stimulated much subsequent work (see Wyatt *et al*, 1979, 1980; DeLisi *et al*, 1982; Gattaz, 1983) but the meaning has remained unclear. Because platelet MAO was found to be unchanged in populations of patients who had not received neuroleptic drugs (Friedman *et al*, 1974; Takahashi *et al*, 1975; Owen *et al*, 1976; Mann & Thomas, 1979) and particularly because, more recently, enzyme activity has been found to decline progressively but slowly with continued neuroleptic medication (Takahashi *et al*, 1975; Friedhoff *et al*, 1978; DeLisi *et al*, 1981; Owen *et al*, 1981) it has appeared increasingly likely that this variable accounts for the low levels sometimes observed in patients with schizophrenia and for the discrepancies in the literature (Belmaker, 1984).

The possible implication of a reduction in enzyme activity in platelets is that it might reflect low MAO activity in the brain. However, this suggestion has not been confirmed in the investigations of brain MAO activity reported to date. Most studies have reported MAO activity unchanged in brains of schizophrenics (Vogel *et al*, 1969; Domino *et al*, 1973; Wise *et al*, 1974; Schwartz *et al*, 1974; Cross *et al*, 1977; Crow *et al*, 1979; Reveley *et al*, 1981). However, Birkhauser (1941) reported MAO activity to be increased in the pallidum of schizophrenics and Utena *et al* (1968) reported an altered regional distribution in MAO activity in brain. In addition Fowler *et al* (1981) reported an increased MAO-B : MAO-A ratio in the pons of patients with chronic schizophrenic and non-schizophrenic psychoses. However, it is arguable that in none of these studies has a sufficient number of brains or a sufficient number of brain regions been studied to identify selective MAO changes in schizophrenia or to identify a possible subgroup of the illness with abnormal MAO activity. In the present study, therefore, we have investigated MAO-A and MAO-B activity in seven brain regions from 44 controls and 39 schizophrenics. The results were analysed with respect to age, sex, storage time at 4°C prior to autopsy, storage time deep-frozen in liquid nitrogen, neuroleptic medication, and diagnosis.

Materials and methods

Chemicals

Benzylamine and 5-hydroxytryptamine creatinine sulphate were obtained from the Sigma Chemical Company. [^{14}C]-benzylamine and [^{14}C]-5-hydroxytryptamine were obtained from the Radiochemical Centre, Amersham. All other chemicals used were from British Drug Houses Ltd, and were of the highest grade available.

Post-mortem tissue

Brains from schizophrenics were obtained at autopsy from hospitals in north-west London and occasionally further afield in southern England. They were included in the study only if the information in the case-notes fulfilled the criteria for definite schizophrenia both as defined by Feighner *et al* (1972) and as defined in the Present State Examination (PSE; Wing *et al*, 1974). For statistical analyses the schizophrenic group was divided according to whether the Nuclear Syndrome was present ($n=28$) or absent ($n=11$) as defined in the PSE syndrome check-list. In addition 22 patients in the schizophrenic group were assessed during life for the presence of positive (hallucinations, delusions, thought-disorder) and of negative (affective flattening, poverty of speech) symptoms using the rating scale devised by Krawiecka *et al* (1977). These patients were then subdivided (Crow, 1980) on the basis that they exhibited either positive symptoms alone (the type I syndrome) or negative symptoms (possibly with positive symptoms also) (the type II syndrome).

Brains were deep-frozen as soon as possible after removal from the cranium, in polystyrene moulds that retained approximate brain shape to facilitate later dissection. At dissection brains were sliced into coronal sections about 0.5–1.0 cm thick. The sections were laid flat on aluminium foil in a cooling tray and the required areas dissected out according to the atlas of DeArmond (1974) and stored over liquid nitrogen. Control brains were obtained from a large district general hospital and dissected and stored as described for schizophrenics. The data relating to the brains is presented in Table I. The schizophrenic group was a little younger, had spent less time at 4°C prior to autopsy, and slightly less time stored over liquid nitrogen than the control group.

TABLE I
Post-mortem brain data

	n	*Sex* *M*	*F*	*Age*	*Time at 4°C: h*	*Time deep-frozen: weeks*
Controls	44	33	13	75.5±1.7	53.7±3.6	233±8.6
Schizophrenics	39	24	17	68.0±2.5	41.2±4.3	201±11.8

Values as mean±s.e.

Assay of MAO activity

The activity of MAO in brain samples was determined by a technique based on that described by Robinson *et al* (1968). Brain samples were homogenised in 30 vol. of 50 mmol/l phosphate buffer, pH 7.2, using a MSE motor-driven homogeniser with a Teflon pestle. Reactions were carried out in plastic Eppendorf tubes in a total volume of 200 μl consisting of 50 μl of homogenate, 20 μl of radioactive substrate and 130 μl of 50 mmol/l phosphate buffer, pH 7.2. Initial studies were carried out on samples of putamen and hypothalamus from eight controls and six drug-free and six drug-treated schizophrenics to determine the effect of neuroleptic medication on the Michaelis Constants (K_m) and maximum reaction velocities (V_{max}) using 5HT and benzylamine as substrates for MAO-A and MAO-B respectively. Drug treatment had no apparent effect on K_m or V_{max} with either substrate. Therefore, MAO activity was assessed in seven brain regions with substrate concentrations of 1.0 mmol/l for both 5HT and benzylamine. Reactions were carried out at 37°C for 30 minutes and then terminated by the addition of 50 μl of 6 mol/l HCl to the reaction mixtures. Deaminated benzylamine products were then extracted into 500 μl of toluene and the deaminated products of 5HT into 500 μl of toluene/ethylacetate (1:1 v/v). After brief centrifugation (8000 *g*, for 2 minutes) 250 μl of the organic phase was transferred to scintillation vials, followed by 10 ml of a Triton X100/Toluene-based scintillant, and the radioactivity determined by liquid scintillation spectrometry. Homogenate proteins were determined by a phenol reagent method (Sutherland *et al*, 1949), and results were expressed as nanomoles of product formed per milligram of protein per 30 min. Extraction efficiencies for the deaminated products of benzylamine and 5HT were 95% and 83% respectively and the results presented have been corrected for recovery.

Results

An initial analysis of covariance showed that there were significant decreases in MAO-B but not MAO-A in frontal cortex, temporal cortex and amygdala of schizophrenics compared with controls. However, there were significant effects of age and sample storage time deep-frozen on both MAO-A and MAO-B activities in most brain regions. The relationship between age and MAO-A and MAO-B obtained by linear regression analysis is presented in Table II. Storage time at 4°C prior to autopsy, sex of the subjects and neuroleptic medication were without significant effect on MAO-A or MAO-B activities. The results of the regional distribution of MAO-A and MAO-B activities presented in Figs 1 and 2, therefore, are adjusted by analysis of covariance with enzyme activity as the dependent variable

TABLE II
Correlations between MAO-A, MAO-B and age

	Controls		*Schizophrenics*		*Controls + schizophrenics*	
	r	*P*	*r*	*P*	*r*	*P*
Caudate						
MAO-A	0.350 (44)	<0.05	0.377 (39)	<0.05	0.353 (83)	<0.001
MAO-B	0.590 (44)	<0.05	0.562 (39)	<0.001	0.590 (83)	<0.001
Hippocampus						
MAO-A	0.218 (40)	NS	0.190 (32)	NS	0.200 (72)	NS
MAO-B	0.320 (40)	<0.05	0.121 (32)	NS	0.236 (72)	<0.05
Frontal cortex						
MAO-A	0.177 (31)	NS	0.297 (34)	NS	0.286 (65)	NS
MAO-B	0.474 (31)	<0.01	0.398 (34)	<0.05	0.458 (65)	<0.001
Temporal cortex						
MAO-A	0.188 (44)	NS	0.293 (37)	NS	0.276 (71)	<0.05
MAO-B	0.370 (44)	<0.05	0.397 (38)	<0.05	0.438 (72)	<0.001
Putamen						
MAO-A	0.368 (44)	<0.05	0.444 (39)	<0.01	0.427 (83)	<0.001
MAO-B	0.555 (44)	<0.001	0.437 (39)	<0.01	0.507 (83)	<0.001
Hypothalamus						
MAO-A	0.103 (44)	NS	0.185 (39)	NS	0.150 (83)	NS
MAO-B	0.479 (44)	<0.001	0.252 (39)	NS	0.329 (83)	<0.001
Amygdala						
MAO-A	0.157 (41)	NS	−0.115 (39)	NS	−0.030 (80)	NS
MAO-B	0.526 (41)	<0.001	0.452 (39)	<0.01	0.504 (80)	<0.001

Numbers in brackets are numbers of brains assayed.

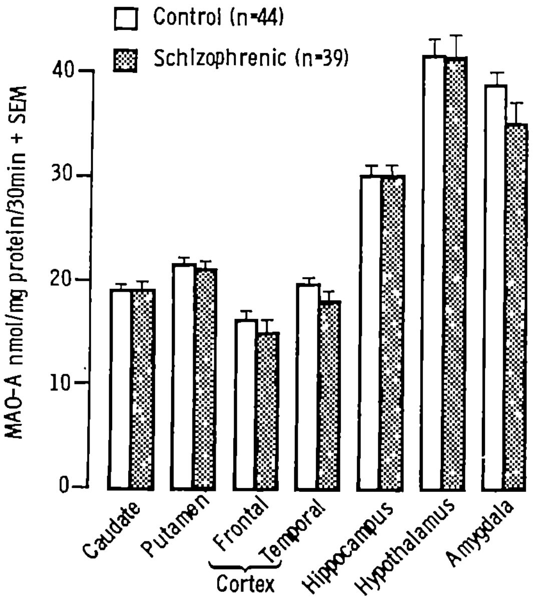

FIG. 1 Regional distribution of MAO-A activity in brains of controls and schizophrenics.

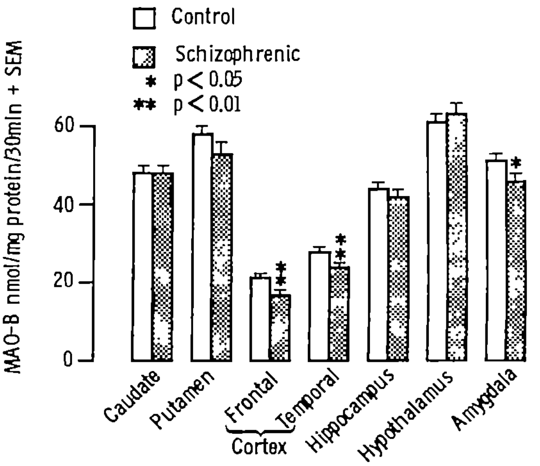

FIG. 2 Regional distribution of MAO-B activity in brains of controls and schizophrenics.

and age and deep-frozen storage time as covariates. This analysis confirmed that there were significant reductions in MAO-B activity in frontal cortex, temporal cortex and amygdala in the schizophrenic patients. Further analysis showed that this MAO-B deficit was specific to type II

TABLE III

MAO-B activity in type I and type II schizophrenics

	Controls	*Type I*	*Type II*	*P*
Frontal cortex	21.2 ± 1.1 (31)	21.9 ± 2.5 (10)	15.3 ± 1.8 (10)	<0.01
Temporal cortex	28.7 ± 0.83 (43)	28.8 ± 2.5 (9)	22.9 ± 1.4 (12)	<0.01
Amygdala	52.6 ± 1.9 (40)	48.0 ± 4.3 (10)	43.3 ± 3.8 (12)	<0.06

Values (mean ± s.e.) as nanomoles of product formed per milligram of protein per 30 min.
Numbers in brackets represent number of brains studied.

schizophrenics (Table III), although the decrease just failed to reach significance in the amygdala, and unrelated to the presence or absence of the Nuclear Syndrome as defined in the PSE.

Discussion

The reductions in MAO-B activity found in the present study in frontal cortex, temporal cortex and the amygdala of schizophrenic post-mortem brain has not been found in earlier studies (e.g. Cross *et al*, 1977; Reveley *et al*, 1981). This result only emerged after an analysis of covariance and may well depend upon the larger sample size of the present as compared to previous studies. However, the reduction in MAO-B activity observed in the present study can hardly be advanced in support of the suggestion that low MAO activity favours the development of schizophrenia (Murphy & Wyatt, 1972). The decrease was selective to patients with negative symptoms and unrelated to the presence or absence of the Nuclear Syndrome as defined by Wing *et al* (1974). In addition, Greene & Grahame-Smith (1978) have demonstrated that MAO is not a rate-limiting enzyme in monoamine metabolism, and the relatively small decreases in MAO-B observed in the present study would not be sufficient to alter central monoamine concentrations. Moreover, it is thought that MAO-B is mainly extraneuronal and MAO-A mainly intraneuronal in rat and human brain (Oreland *et al*, 1983). Thus the MAO-B deficits are most unlikely to be relevant to neurotransmitter metabolism. For the reasons given in the introduction, the deficit is also unlikely to be related to changes in MAO activity in platelets, which are probably secondary to neuroleptic medication.

MAO-B activity, but not MAO-A activity, has been reported to be increased in brains from patients dying with Alzheimer's disease (Adolfsson *et al*, 1980) and also after hemitransection of the brain in the rat (Oreland *et al*, 1980), in both of which cases there is proliferation of glia in the degenerating parts of the brain. Similarly, Fowler *et al* (1981) reported increased MAO-B : MAO-A ratio in schizophrenics compared with controls in samples from the pons (but not other areas), and suggested that the increased ratio resulted from neuronal loss. The results of the present study are not consistent with Fowler's suggestion. It can be seen by inspection (Figs 1 and 2) that MAO-B : MAO-A ratios were decreased rather than increased in most brain regions studied.

MAO-B activity, measured with B-phenylethylamine as substrate, has been reported to be decreased in 12 out of 13 brain regions studied in samples from suicide victims (Gottfries *et al*, 1974). The selective decrease in MAO-B activity in certain brain regions of patients with the type II (defect state) syndrome, therefore, is unlike the more general decrease found in brains of suicides.

The initial kinetic studies on samples from controls and drug-free and drug-treated schizophrenics revealed, firstly, that there were no gross differences between controls and schizophrenics in the V_{max} or K_m of MAO with either 5HT or benzylamine as substrate, and secondly, that neuroleptic medication had no effect on V_{max} or K_m values. An analysis of covariance in the larger series of samples confirmed the lack of effect of neuroleptic medication on MAO-A or MAO-B activity in any of the seven brain regions studied. This is consistent with a previous report (Eckert *et al*, 1980).

A positive correlation between MAO-B and age (Table III) was found, as previously reported for human (Robinson *et al*, 1972; Fowler *et al*, 1981) and rat brain (Benedetti & Keane, 1980). When controls and schizophrenics were considered together, MAO-B activity correlated significantly with age in all brain regions. MAO-A activity was less strikingly related to age, although significant correlations were found in the caudate and putamen of both controls and schizophrenics, and in caudate, putamen and temporal cortex when controls and schizophrenics were considered together. It is unlikely that the relatively high substrate concentrations used in the assay procedure resulted in a loss of substrate specificity, since it has been clearly demonstrated that 5HT and benzylamine remain specific substrates for MAO-A and MAO-B respectively over a very wide range of concentrations (Suzuki *et al*, 1979). Moreover, in the initial kinetic study the ratio of MAO activity towards 5HT and benzylamine did not alter over the range of concentration used. It may be that a large sample is necessary to demonstrate this weak relationship between MAO-A and age. In the present study the schizophrenic group was a little younger than the control group but this age difference was taken into account in the analysis of covariance.

MAO activity was unaffected by the sex of samples or the length of time cadavers were at 4°C in the mortuary prior to removal of the brain from the skull. The post-mortem stability of brain MAO has been demonstrated with experimental animals (Fahn & Cote, 1976). However, there was a significant decline in MAO activity after prolonged storage over liquid nitrogen in all brain regions studied in both controls and schizophrenics. The effect of storage time on MAO activity was also taken into account in the analysis of covariance.

The selective MAO-B changes in frontal and temporal cortex and amygdala in patients with the type II syndrome found in the present study are unlike any previous findings in brain samples from individuals dying from various neuropsychiatric conditions. These changes either reflect, rather than mediate, changes in monoamine function, as suggested by Gottfries *et al* (1974) to explain the decreased MAO activity in brains of suicides, or are part of a more general biochemical pathology in these particular brain regions. Monoamine oxidase is situated in the outer mitochondrial membrane, and an investigation of other mitochondrial markers in brain samples from schizophrenics seems worthwhile. The findings add to other evidence of structural (Brown *et al*, 1986) and neurochemical (Ferrier *et al*, 1983; Reynolds, 1983) changes in the temporal lobe in schizophrenia and emphasise that such change may be particularly associated with poor outcome.

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A Comparison of Scales for Assessing Rehabilitation Patients

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Twenty-four chronic schizophrenic patients were assessed by the Morningside Rehabilitation Status Scale, the Krawiecka Scale, and the Social Adjustment Scale by Self-Report. Inter-rater correlations suggest that the MRSS can be used by a rater with little previous knowledge of the patient. Between-scale correlations suggest the three scales assess somewhat different dimensions. A standard approach to rehabilitation assessment is suggested.

Assessment of patients who pass through rehabilitation services is necessary if progress is to be accurately charted. A new assessment scale, the Morningside Rehabilitation Status Scale (MRSS) (Affleck & McGuire, 1984) covers a wide range of rehabilitation problems, yet can be readily used in a busy NHS rehabilitation service. The scale examines rehabilitation status over the previous month in the dimensions of independence/dependence; activity/inactivity; social integration/isolation; and the effect of symptoms. However, once a scale is published, further work should be undertaken to learn more about its properties, its suitability for use with various populations, and how it compares with other scales (Snaith, 1981). We therefore wish to report, firstly, inter-rater reliability results in the final published version of the scale; and, secondly, a comparison of the MRSS with two established rating scales, namely the Social Adjustment Scale by Self-Report (SAS–SR) (Weissman & Bothwell, 1976) and an assessment of mental status, the Krawiecka Scale (Krawiecka *et al*, 1977).

Method

Twenty-four chronic schizophrenic day patients, 10 females and 14 males, 88% of whom fulfilled the Feighner Criteria for definite or probable schizophrenia (Feighner *et al*, 1972), were rated independently by two psychiatrists using the MRSS; one psychiatrist was in thrice-weekly contact with the patients, the other had little or no previous contact. Assessment, which took 20–30 minutes, was through interviews with the patients and discussion with senior nurses at the day hospital. The sort of information needed to complete the scales is shown in Appendix 1. On the same day, a third psychiatrist assessed patients' mental status over the previous week using the Krawiecka Scale, and within the same week patients assessed their own social adjustment over the previous two weeks using the SAS–SR. The Krawiecka Scale assesses eight schizophrenic and other symptoms: depression, anxiety, flattened or incongruous affect, retardation, delusions, hallucinations, incoherence, and poverty of speech. An assessment can be made in about 15 minutes. Its content allows the classification of patients in accordance with the Wing Scales (Wing, 1961). The SAS–SR, which takes about 10 minutes to complete, is a 54-item questionnaire which yields an overall adjustment score and a rating of six role areas: work as a worker, housewife or student; social and leisure activities; relationships with extended family; and marital roles as a spouse, parent, and member of the family unit.

Results

Correlations between raters on MRSS total and subscale scores ranged from +0.62 (for dependence) to +0.83 (total score) (Table I). The range of correlations was broadly similar to that previously found in Krawiecka scales but somewhat greater than in the SAS–SR (Table I). There were no significant between-rater differences in magnitude of scores except in the inactivity subscale of the MRSS (*t*-test; $t=2.97$, $P<0.01$).

When scales were compared (Table II), correlations between total MRSS scores and both the Krawiecka and SAS–SR scores were statistically significant, but there was no significant correlation between the Krawiecka and SAS–SR scores. With MRSS subscales, the only correlations that were statistically significant in both raters were between dependence and symptoms and Krawiecka total scores.

Discussion

Inter-rater correlations in total and subscale MRSS scores were high and suggest that an accurate rating of patients' rehabilitation status can probably be made by an external rater; although he could obtain details from staff or relatives, he had little previous knowledge of the patient. Discussion of the discrepancy in magnitude of scores in the activity/

inactivity subscale revealed contradictory rating instructions, which were subsequently modified; this supports the view that further work to improve a scale once it has been published should be undertaken (Snaith, 1981).

Correlations between scales were more variable. One general problem is in the different lengths of observation period for the three scales. Also, the different scales do not attempt to measure exactly the same dimension; for example, the MRSS symptom subscale aims at grading the effect of symptoms on the patient's lifestyle, and is not merely a straightforward mental state assessment. Another difference is that the Krawiecka Scale is aimed specifically at chronic patients, whereas the MRSS anticipates that some patients may have no symptoms at all. However, MRSS total scores correlated significantly with both the Krawiecka and SAS-SR. It was the symptom and dependence subscales that contributed most to the overall correlation with the Krawiecka; that the symptom subscale did so is not surprising, but the contribution of dependence was less expected. However, scrutiny of the information required to assess dependence (Appendix 1) shows that contact with staff is an important part of this assessment; patients with many abnormalities in their mental state will no doubt attract the attention of medical and other staff.

Correlations between the MRSS and SAS-SR, although statistically significant for both raters when total scores were compared, were in the main lower, and no subscale obviously contributed in both raters' assessment to the overall correlation. The assessment of social adjustment is obviously more hazardous. Patients' 'feelings and satisfactions' which are rated in the SAS-SR may not be tapped by the MRSS.

TABLE I
Inter-rater reliability

MRSS	*Pearson correlation (r)*	*P*
Total score	+0.83	<0.001
Dependence	+0.62	<0.01
Inactivity	+0.70	<0.001
Social integration	+0.77	<0.001
Symptoms	+0.81	<0.001
Krawiecka Scale[1]	*Kendell's coefficient of concordance (W) (between psychiatrists)*	*P*
Depression	+0.87	<0.001
Anxiety	+0.64	<0.001
Delusions	+0.83	<0.001
Hallucinations	+0.78	<0.001
Incoherence	+0.64	<0.001
Poverty of speech	+0.73	<0.001
Flattened, incongruous affect	+0.58	<0.01
Psychomotor retardation	+0.62	<0.001
SAS-SR[2]	*Pearson correlation (r) (between patient and observer)*	*P*
Overall adjustment	+0.72	<0.001
Work	+0.58	<0.001
Social and leisure	+0.71	<0.001
Extended family	+0.51	<0.001
Marital	+0.76	<0.001
Parental	+0.47	<0.001
Family unit	+0.40	<0.001

1. Taken from Krawiecka *et al* (1977).
2. Taken from Weissman & Bothwell (1976).

TABLE II
Pearson product-moment correlations between MRSS, Krawiecka and SAS-SR scales

MRSS	*Krawiecka (total score)*		*SAS-SR (total score)*	
	Psychiatrist 1 v. Psychiatrist 3, r	*Psychiatrist 2 v. Psychiatrist 3, r*	*Patient v. Psychiatrist 1, r*	*Patient v. Psychiatrist 2, r*
Total score	+0.67****	+0.51***	+0.46**	+0.50***
Dependence	+0.58***	+0.43*	−0.07	+0.28
Inactivity	+0.18	+0.36	+0.27	+0.29
Social integration	+0.50***	+0.36	+0.33	+0.54***
Symptoms	+0.68****	+0.58***	+0.56***	+0.20
Krawiecka			*SAS-SR (total score) Patient v. Psychiatrist 3*	
Total score	—	—	+0.16	

*$P<0.05$, **$P<0.02$, ***$P<0.01$, ****$P<0.001$.

There was no correlation between the SAS–SR and Krawiecka; these scales are obviously examining different aspects of the patient's functioning.

In conclusion, we agree with Hall (1979) that where possible more than one type of assessment should be used. The scales chosen will depend on clinical and research objectives, but it might be reasonable to consider the MRSS for overall rehabilitation status and a dimensional profile; the Krawiecka for symptoms; the SAS–SR for social adjustment from the patient's view; and perhaps (for research purposes only) the Social Behaviour Assessment Schedule (Platt *et al*, 1980) for the relative's view. This would enhance the probability of a standard approach to rehabilitation studies, but high levels of reliability and validity can only be maintained if the users of the scales know about the pitfalls and if reliabilities are assessed and discrepancies discussed.

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Appendix 1
MRSS information checklist

Dependency scale

How independent the patient is with regard to self-care, economics and medication

1. Living place
2. Other occupants and their role regarding patient's dependency
3. Domestic arrangements
4. How economic responsibilities are shared
5. Personal habits: hygiene, clothing, tidiness, rising routine
6. Visiting by staff
7. Medical arrangements
8. Other professional contacts if any

Inactivity (occupational and leisure) scale

How well the patient has initiated and sustained activity and performed effectively

1. Work
2. Training programme
3. Method of looking for work
4. Daily routine
5. Leisure time routine, indoor/outdoor (weekdays and weekends)
6. Reading habits and interests, TV, radio, etc.

Social integration/isolation scale

Frequency of contact and social competence

1. Companions in living place
2. Friendliness with other occupants
3. Neighbours known? how well?
4. Family members in current contact
5. Other *social* activities, games, sports
6. Social contacts at work
7. Close friends, intimate friends
8. Social difficulties or incompetence

Effects of current symptoms and deviant behaviour scale

Current problems only must be rated, i.e. only those which have occurred during the period being assessed, normally the last month.

1. Subjective symptoms
2. Other serious symptoms elicited by questions
3. Attitude to medication
4. Symptoms observed by others
5. Other unusual behaviour
6. Difficulties in organising daily routine

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Extent, Timing and Persistence of Emotional Disorders following Childbirth

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A three-stage community survey of a representative group of Southampton mothers was carried out. A semi-structured psychiatric interview was administered at 3, 9 and 15 months post-partum. The results of these interviews are presented and used to assess the extent and timing of psychiatric disorders arising in this period. In contrast to previous studies, it was found that the highest incidence of *new cases*, variously defined, occurred between the third and ninth month post-partum and not immediately following childbirth. These results are discussed in the context of previous studies using the same definition of caseness.

In recent years there has been a series of attempts to estimate the extent and nature of emotional disorders following childbirth. Pitt (1968) estimated that the incidence of 'atypical depression' at 6–8 weeks following childbirth was 10.8%. He was able to follow up 28 of his depressed mothers, and at 15 months 12 were still depressed.

In his paper Pitt emphasised the importance of this finding, even if most cases had been 'mild' from a hospital-based psychiatrist's point of view. Post-natal depression appeared an unexpectedly common condition and for 4% of his population it appeared to be persistent. In fact, re-examination of Pitt's data (Neugebauer, 1983) has indicated that because he only interviewed all his probable cases to estimate incidence, Pitt underestimated the total number of cases in his population. Neugebauer recalculated Pitt's figures, taking into account the cases lost by the screening method, and estimated the incidence to be nearly 20%.

Kumar (1982), in a critical review of the most important of the studies, including his own, pointed out some of the methodological difficulties which undermined the reliability of many of the estimates of the incidence of post-natal depression. These have varied from 3% (Tod, 1964) to 30% (Rees & Lutkins, 1971).

Kumar's own study (Kumar & Robson, 1984) had attempted to overcome some of these difficulties. He and his co-workers used a standardised semi-structured psychiatric interview (SPI) (Goldberg *et al*, 1970) to define their cases, and their 114 subjects were interviewed at least three times during pregnancy and five times after delivery. However, the SPI was administered to all the subjects only on three occasions. The investigators rated as 'new' cases only those cases who had not been cases at the previous interview. Upper- or middle-class subjects were over-represented and all subjects were dutifully attending regularly a London teaching hospital clinic. All the subjects were primips. It was found that 3 months after delivery, 17% of the mothers had significant disorder, and 11.4% of the women had developed their disorder since childbirth.

Although this study has remained the most intensive so far, Kumar himself has suggested that his subjects were not representative of a normal community sample. If indeed this was an unusually healthy and resilient sample, then two things follow. Firstly, Kumar & Robson's figures are probably an underestimate of the prevalence and perhaps the severity and persistence of post-partum disorders in other populations. Secondly, the evidence supports the idea that even in relatively favourable circumstances, pregnancy and even more the months post-partum are associated with an increased risk of depression.

One further important factor mentioned by Kumar is the possible supportive effect of their frequent interviews.

In a subsidiary sample of 38 primips and 39 multips who were studied only post-partum but otherwise in an identical manner, Kumar & Robson found that, overall, 28.6% were rated cases at 6 months. One of the 38 primips developed a psychosis, and of the rest 10% were depressed at one year. Of the multips, 23% were depressed at one year.

The study described in this paper used the same criterion of caseness as Kumar & Robson but the interviews were restricted to three interviews at 3, 9 and 15 months post-partum, and included a representative sample of Southampton mothers, primips and multips. The present paper describes the findings concerned with the prevalence, persistence and timing of disorders arising in this sample.

Method

The sample consisted of all 212 mothers listed as having had babies during a 3-month period. Their names were obtained from the birth books of five Southampton group practices. Each mother was contacted by her general practitioner (GP) and informed of the study and invited to take part.

Interviews

There were three interviews, all conducted in the subject's own home at 3, 9 and 15 months post-partum. At the first interview, each mother who agreed to take part first completed the 30-item General Health Questionnaire (GHQ-30). This was then placed in a folder without being scored, and the interviewer proceeded to fill in a data sheet. Finally, the semi-structured psychiatric interview (SPI) was administered. The second and third interviews followed the same procedure, using, however, different data sheets. After the third interview the subject also completed the Eysenck Personality Inventory (EPI). The present paper presents the findings related to the SPI. The GHQ-30 data and correlates of post-natal depression will be presented in separate papers.

Semi-structured psychiatric interview

The semi-structured psychiatric interview (SPI) (Goldberg *et al*, 1970) is acceptable to people who do not consider themselves mentally ill, and is shorter than other commonly used standardised interviews. It is suitable for measuring the nature and severity of neurotic disturbance and has been used previously in studies of post-partum depression (Cox *et al*, 1982; Kumar & Robson, 1984). It is designed to elicit the presence or absence of ten defined psychiatric symptoms. In addition, twelve manifest abnormalities are looked for by the interviewer during the interview. The SPI can be used in various ways to define 'caseness'. In this study, caseness was defined by the 'overall severity rating' (OSR). At the end of the interview the subject is classified on a five-point scale: normal, 0; minor symptoms short of clinical significance, 1; symptoms warranting GP referral, 2; symptoms requiring psychiatric out-patient referral, 3; and symptoms judged to need psychiatric in-patient treatment, 4.

Inter-rater reliability

Each of the three interviews was carried out by a separate interviewer. All three interviewers and the author were trained in administering the interview at the GP Research Unit at the Institute of Psychiatry. The author supervised each stage and discussed any difficulties in rating. A note was made of any subjects considered borderline. At each stage, 12 of the subjects were interviewed by two interviewers, and the results of this separate study (Nott, 1984) indicated a high inter-rater reliability.

Results

Characteristics of the sample

Of the 200 subjects completing the first interview, the age range was 16–39 years (mean 24.8). There were 188 married women, 6 single and 6 divorced or separated. Social class distribution defined by husband's occupation was similar in our sample to that of the social class distribution of Southampton heads of household by occupation determined in the 1982 Census. The incidence of obstetric intervention and complications was similar in our sample to the incidence in all Southampton deliveries.

Prevalence of cases

Table I shows the number completing each interview and the OSR rating. The percentage of cases (OSR 2 or more) at each stage is 18.5% at 3 months, 28% at 9 months, and 31% at 15 months. It can also be seen that although the majority of cases are less severe (OSR 2, warranting GP referral), the number thought to warrant psychiatric intervention is increased at 9 months and 15 months.

Symptoms experienced by cases

Each symptom rated during the structured psychiatric interview was rated on a scale of 0 to 4. A score of 2 or more was regarded as pathological. The percentage of *cases* ($n = 37$) with a pathological score for the various symptoms at 3 months was as follows: somatic symptoms, 43%; fatigue, 68%; sleep disturbance, 49%; instability, 84%; lack of concentration, 51%; depressed mood, 78%; anxiety, 62%; phobias, 11%; obsessions, 27%; depersonalisation, 22%.

At 3 months the commonest symptoms of note were irritability, depressed mood, and anxiety. The symptom profiles were similar at all three stages of the study and confirm that post-natal *depression* is probably an appropriate name for these disorders. None of our subjects developed a psychotic illness and none was admitted to a psychiatric unit.

Caseness with time

Having three consecutive interviews at 6-month intervals, we can follow the outcome of cases and non-cases. If a past history of emotional disorder is taken as caseness prior to the study, we can illustrate the probability of recovery or onset of illness during those time intervals. Table II shows that most change is taking place between the first and second interviews, and there is an overall tendency for both cases and non-cases to retain their category with time.

New cases arising post-partum

Using the data in this study, 'new' cases can be defined in two ways. New cases (1) are defined as cases who have no

TABLE I

Overall severity rating on SPI

	Rating									
	0		*1*		*2*		*3*		*4*	
	n	*%*	*n*	*%*	*n*	*%*	*n*	*%*	*n*	*%*
3 months ($n=200$)	93	46	70	35	34	17	3	1.5	0	0
9 months ($n=173$)	74	43.1	50	29	34	19.5	14	8	1	0.5
15 months ($n=164$)	46	28	67	41	41	25	9	5.5	1	0.5

TABLE II

Probability of becoming a case or recovering for each interval between interviews using past psychiatric history (PPH) as caseness prior to the study

	PPH to 1st interview (at 3 months)	*1st to 2nd interview (3 months to 9 months)*	*2nd to 3rd interview (9 months to 15 months)*
Chance of C–C	0.30	0.55	0.63
Chance of C–NC	0.70	0.45	0.36
Chance of NC–C	0.14	0.24	0.14
Chance of NC–NC	0.86	0.76	0.86

C – case, NC – non-case

history of contact with the psychiatric services and have not been rated cases at a previous stage in the study. New cases (2) are defined, even more exclusively, as cases who have no past psychiatric history at all – not even contact with a GP for emotional problems – and have not been rated cases at an earlier stage of the study. Table III gives the prevalence of 'new' cases at each interview for each of these two definitions, and the overall prevalence for comparison.

Defining new cases in this way excludes 'chronic cases' and the recruitment of cases with time as the study progressed. It can be seen that even by the strictest definition, the prevalence of new cases at each stage is considerable. Moreover, most new cases arise between 3 and 9 months, and not in the 3 months immediately following childbirth.

TABLE III

Ratios of cases to total number at each stage for all cases and 'new' cases defined in terms of past psychiatric history (see text for definitions)

Interview	*Cases/total number*		
	All cases	*New cases (1)*	*New cases (2)*
1 (3 months)	37/200 (18.5%)	32/188 (17%)	20/150 (13%)
2 (9 months)	50/173 (29%)	30/136 (22%)	20/112 (18%)
3 (15 months)	51/163 (31%)	10/94 (10.6%)	7/81 (9.5%)

TABLE IV

Persistence of 'new case' at interview 1 with time

Interview 1 (3 months)	*Interview 2 (9 months)*	*Interview 3 (15 months)*
32 (new case (1)[1]) →	15 (case) →	11 (case)
	12 (non-case)	3 (non-case)
20 (new case (2)[1]) →	9 (case) →	8 (case)
	8 (non-case)	1 (non-case)

1. See text for definition

Outcome of new cases

By following those subjects classified as new cases at the first interview, we can determine whether these cases recover quickly or not. Table IV gives the figures for those 'new cases' at interview 1 who completed the whole study. Some subjects were lost at both the later interviews, but not enough to affect the overall picture. The table clearly shows that, defining new cases in either way, of all the new cases at first interview, about half remain 6 months later, and the majority at that stage remain cases at 15 months.

Discussion

This study shows that post-natal depression as described by Pitt (1968) is a common finding in a representative group of Southampton mothers. We have found that it is characterised by irritability, depressed mood, fatigue and a considerable degree of anxiety. It is often associated with feelings of hopelessness and an inability to cope with daily tasks. However, none of our subjects developed a psychotic illness and none was admitted to a psychiatric unit.

The lack of psychiatric involvement in the management of post-natal depression in our subjects explains our previous psychiatric case-register findings (Nott, 1982). These had indicated that psychotic disorders increased sixfold following childbirth compared with other times, to a rate of 1.15 per 1000. Referral for less severe depressive and anxiety states decreased during pregnancy, and did not increase significantly following childbirth, compared with rates prior to and during pregnancy. This underlines the fact that community studies are necessary to obtain reliable evidence of the prevalence and incidence of less severe psychiatric disorders, which are mostly not referred to the psychiatric service and so missed by case-register studies.

Even in a community survey such as this, the meaning of specific prevalence figures may remain problematical. Barker & Rose, quoted by Williams *et al* (1980) in a paper which discusses the problems of case definition, state:

> apart from a few inherited disorders . . . in the general population, disease behaves as a continuously distributed variable. The question is not so much 'has she got it?' as 'how much has she got?'

We certainly found this was so in trying to measure disorder in our subjects. However, using a standardised interview and a defined cut-off point, we can compare the same subjects at different times, and allow our subjects to be compared with different populations who have been surveyed using the same instrument.

At 3 months post-partum, our prevalence of 18.5% for cases and 13% for 'new cases' is comparable with the result of Kumar & Robson (1984), who found 16% cases and 12.6% new cases arising by 3 months after delivery. Cox *et al* (1982) found a prevalence of 13% cases at 3 months, and Watson *et al* (1984) a prevalence of 16% (12% new cases) at 6 weeks post-partum. These studies used the same standardised psychiatric interview as the present study.

Our survey shows that the number of subjects rated as cases is greater at 9 months than at 3 months, and this increase persists at 15 months. We have previously shown that this is not due to the recruitment of chronic cases with time. The highest prevalence of new cases is at 9 months (18%): prevalence falls again to 9.5% at 15 months. The symptom profiles of the cases were similar at all three points in time.

Of our subjects who became cases for the first time post-partum (i.e. they had no previous history of emotional disorder prior to that time), half of them were still rated cases 12 months later. This tendency for cases to remain cases with time is not revealed by simple cross-sectional studies and is important. A persistent disorder is more likely to be related to adverse social and family consequences.

There is other evidence suggesting that these disorders are not shortlived. Pitt (1968), in a postal follow-up at one year post-partum, found that 12 of his 28 cases had not recovered. Feggeter *et al* (1981) carried out a survey of women one year after childbirth, using the General Health Questionnaire (Goldberg, 1972) as a screening instrument and the Present State Examination (Wing *et al*, 1974) to define caseness: they found that for those rated cases, the duration of the disorder assessed retrospectively from the time of interview was over one year in 13% and one year or slightly less in 35%. Kumar & Robson (1984) found that at 4 years post-partum, 14 of the 16 cases rated at 3 months post-partum were still available for interview: 6 of these, almost half, had continued to consult for emotional problems over the whole 4-year period.

Kumar & Robson (1984) found the highest prevalence of new cases to be at 3 months post-partum, thereafter dropping by about two-thirds to what they state is in line with their pre-pregnancy estimate. Watson *et al* (1984) found the highest prevalence at 6 weeks post-partum, but they do not appear to have carried out a formal SPI rating after that time. Kendell *et al* (1976), commenting on their case-register study, noted a secondary rise, "less dramatic but more sustained, from the 10th to the 24th month after delivery". This finding of a raised psychiatric consultation rate up to 2 years post-partum was supported by the finding of Kendell *et al* (1981) of a raised psychiatric admission rate for at least 2 years after childbirth in a separate case-register study. Our own findings of a peak prevalence for new cases at 9 months and a persistent high total prevalence may explain this discrepancy. In a relatively healthy population of primips it may be that those who are going to develop symptoms do so relatively soon after childbirth, but for multips and others, symptoms develop later. In either case, for a significant number the symptoms are liable to be persistent, and these 'chronic' cases are those most likely to be referred for psychiatric help at a relatively late stage, when primary health care measures have failed.

These studies do not answer the following question: are prevalence rates post-partum greater than those found in a general population sample of non-post-partum women? No study of puerperal women has yet included a suitable control group. Indeed, any suggested control group is likely to differ systematically from a post-partum group in numerous ways. Kumar & Robson (1984) point out that their period prevalence for the post-partum year (24%) was little different from the findings of surveys of the mental health of female attenders in general practice (Shepherd *et al*, 1966). They suggest that their figure would have been higher if they had

had a less healthy sample. In our own study, the 9-months period prevalence for new cases by our strictest criterion is 31%, a figure which supports the idea that the post-partum year is a particularly vulnerable period.

The only comparison we can make at present with a general population survey using the same standardised psychiatric interview is to compare our prevalence rates for all cases with those of Hobbs *et al* (1983). They carried out a survey of 1517 women aged 20–60 years using the General Health Questionnaire. They then arrived at estimated prevalence rates by interviewing subsamples of high scorers and low scorers. They used the same semi-structured psychiatric interview (SPI) as the present study. Using the overall severity rating as in this study, they found an estimated prevalence rate of 26%. This can be compared with our prevalence rates for all cases of 18.5%, 28% and 31% at our three interviews.

In conclusion, we have found evidence which supports the idea that the post-partum period is a period of increased vulnerability for women, but the suggestion of a strict temporal relationship of disorder to delivery was not corroborated. This has implications for attempts to understand the cause of post-natal disorder. The findings of this study suggest that the causes of post-natal depression are more likely to be in the psychosocial aspects of the puerperium, and perhaps the mother's own predisposition in terms of personality and background, rather than in the hormonal and other physical changes associated with childbirth itself. This is in agreement with the suggestions arising from the intensive study of Watson *et al* (1984). Indeed, it again raises the question of whether in fact 'post-natal depression' is qualitatively different from depression at other stages in a woman's life.

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Familial Aspects of Obsessive–Compulsive Neurosis

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The first-degree relatives of 50 obsessive–compulsive patients and those of matched controls completed the General Health Questionnaire (GHQ) and the Leyton Obsessional Inventory (LOI). Relatives who were identified as possible 'cases' by their high GHQ scores, or by their own or informant relatives' reports, were interviewed using the Schedule for Affective Disorders and Schizophrenia. Index relatives had a significantly higher lifetime prevalence of mental illness (36%) than had those of controls (17%), due mainly to an excess of depressive and neurotic disorders. However, only one relative from each group was diagnosed as having definite obsessive-compulsive neurosis. In addition, the LOI scores were similar for the index and control relatives.

Studies of the relative contributions of heredity and environment to the development of obsessive–compulsive neurosis have, in the absence of biological markers, centred on twin and family investigations. The present study was undertaken to determine the prevalence of obsessive–compulsive neurosis at a clinical and psychometric level in the first-degree relatives of probands with this disorder, and to examine the effect of the degree of kinship and birth order on obsessional symptoms.

Most twin studies point to a higher concordance rate for obsessive–compulsive neurosis for monozygotic (MZ) pairs than for same-sex dizygotic (DZ) pairs. However, such findings need to be interpreted cautiously, in view of (a) the bias in the selection and reporting of cases, (b) the use of unsatisfactory diagnostic criteria, (c) the diagnostic bias introduced when the researcher is aware of the twins' zygosity, and (d) the fact that zygosity is not always fully established. These points are emphasised by the conflicting conclusions of two reviewers of the same literature. Carey (1978) noted that there were 30 concordant and 13 discordant MZ twin pairs, compared with no concordant and 14 discordant DZ pairs. Black (1974), however, concluded that in only three published instances of identical twins with obsessive–compulsive neurosis was the zygosity and diagnosis reliably determined. More recent twin studies continue to report disparate findings (Carey & Gottesman, 1981; Torgensen, 1983), and although they indicate a higher concordance rate for MZ than for DZ twins for neurotic disorders, they question the idea that specific neurotic disorders breed true.

While the overall trend of family studies for obsessive–compulsive neurosis indicates familial aggregation, some investigations provide insufficient detail (Lewis, 1936), or lack control data (Rudin, 1953; Rosenberg, 1967) or agreed operational criteria (Brown, 1942; Kringlen, 1965), thus making the reliability of their findings less certain. However, the results of three studies bear comparison. Brown (1942) found that, compared with controls, the parents of obsessive–compulsive patients had a significantly increased incidence of obsessional neurosis (8%), as did the patients' siblings (7%). Rudin (1953) reported finding obsessive–compulsive features in 4.6% of parents and in 2.3% of siblings of obsessive–compulsive patients. Both these studies produced higher familial rates of obsessive–compulsive disorders than that of Rosenberg (1967), who found among 547 first-degree relatives only two cases of obsessional neurosis. The diverging familial rates probably reflect the different diagnostic criteria used.

Psychometric investigations of families of obsessive–compulsive patients have been limited to three studies (Coppen *et al*, 1965; Rosenberg, 1967; Insel *et al*, 1983). Coppen and his associates administered the Maudsley Personality Inventory (MPI) to relatives of patients with neurotic illness, of whom 19 had obsessional neurosis, and found that the familial level of neuroticism was lower than that quoted for normal subjects. Rosenberg (1967) reported that the mean neuroticism score on the MPI for the first-degree relatives of obsessional probands was slightly below the accepted norm.

The only psychometric study specifically of obsessionality is that of Insel *et al* (1983). They found that mean Leyton Obsessional Inventory symptom scores for the parents of ten obsessive–compulsive patients did not differ from normative data.

Abnormal pre-morbid personality traits have been noted in some two-thirds of those who develop obsessive–compulsive neurosis (Pollitt, 1960;

McKeon, 1983). If indeed obsessional neurosis has a familial tendency, the possibility of this being reflected in a latent form in the patients' pre-morbid personalities could be usefully explored by examining the relationship between the extent of familial clustering and degree of personality abnormality.

In this paper we report the prevalence and type of psychiatric morbidity among the first-degree relatives of probands with obsessive–compulsive neurosis and a matched control group, and examine the relationship between the extent of familial clustering of mental illness and the probands' pre-morbid personalities. Psychometric scores for obsessionality, phobias and psychiatric morbidity for index and control families are compared, and the effect of degree of kinship and ordinal birth position on obsessional symptoms is studied.

Method

Obsessive–compulsive probands

Index probands were consecutive attenders at the Maudsley and Bethlem Royal Hospitals' behaviour therapy unit who had received from their treating psychiatrist a primary diagnosis of obsessive–compulsive neurosis (300.3 in the International Classification of Diseases (ICD) (World Health Organization, 1975)), and who met the operational definition of the Research Diagnostic Criteria for obsessive–compulsive neurosis (Spitzer *et al*, 1978). Those agreeing to participate in the study had to have living first degree-relatives whom they consented to have interviewed, none of whom was already included as a proband. The first 50 patients meeting these criteria formed the obsessive–compulsive proband group. One subject who met the diagnostic criteria for inclusion in the study was excluded, as he declined to have his relatives interviewed.

Comparison-group probands

Controls were selected from consecutive attenders at two general-practice surgeries. Subjects agreeing to participate, who could be matched with a patient proband for sex, age (within 3 years), marital and socio-economic status (within one class on the Registrar-General's Classification), race, religion (parents' religion) and birth order, were interviewed using the Schedule for Affective Disorders and Schizophrenia – Life time version (SADS-L; Endicott & Spitzer, 1978). Those who did not meet 'case' criteria on the SADS-L were included in the study. Two matching systems were employed for birth order: one classified subjects as being either only children, first-born, middle-born or youngest, while the other was sex-related with eight categories – only boy, only girl, eldest boy, eldest girl, middle-born boy, middle-born girl, youngest boy and youngest girl. Three subjects who met the matching criteria declined to participate in the study.

Relatives

Patient and comparison-group probands were requested to distribute the questionnaires to all their first-degree relatives over the age of 16 years. The questionnaires were accompanied by an introductory letter requiring respondents to complete the forms independently and return them by post. Relatives who did not return the questionnaires within 4 weeks were written to and, where necessary, visited to encourage completion of the rating scales.

Questionnaires

General Health Questionnaire (GHQ) (Goldberg & Hillier, 1979). This 28-item version is a modified form of the original GHQ, and its sensitivity and specificity is at a maximum with a cut-off score between 4 and 5.

Leyton Obsessional Inventory (LOI). The pencil-and-paper questionnaire, which was modified by Snowdon (1980) from the original version of Cooper & McNeil (1968), was used. It enquires about obsessional traits and symptoms, and some symptom questions incorporate a measure of the degree of interference and resistance associated with certain obsessions and compulsions.

The Fear Questionnaire (FQ) (Marks & Mathews, 1979). This self-rating questionnaire yields four scores: main phobia, global phobia, total phobia and anxiety depression. The total phobia score is comprised of agoraphobia, social and blood injury subscores.

Health Report Sheet (HRS). This is an open-ended questionnaire which we constructed for this study, and it enquires about the mental health of the respondents, their history of psychiatric morbidity and details of any treatment received.

Interviews

SADS-L. Relatives who were identified by their HRS or by a family member's account as having had noteworthy psychiatric symptoms, or who had GHQ scores greater than 4, were interviewed using the SADS-L. To check the reliability of the GHQ and HRS as screening instruments, 15 randomly selected relatives from the index and control groups who were not already being given a SADS-L interview were interviewed using this schedule: only one was found to reach 'case' status; this was a comparison-group relative suffering from alcoholism.

Standard Assessment of Personality Schedule (SAP) (Mann et al, 1981). This is a standardised interview schedule conducted with an informant relative (parent, spouse or sibling) who has an adequate knowledge of the subject. It enquires about aspects of the subject's pre-morbid personality. Its intertemporal and interinformant reliability has been shown to be satisfactory for ratings carried out on obsessive–compulsive patients years after the onset of the illness (McKeon *et al*, 1984). It deals with six personality categories based on the ICD and two further categories not covered by the ICD, these being for 'self-conscious' and 'anxious' personalities. Each personality category is rated as follows: grade 0, no features; grade 1, features present; grade 2, features pronounced (or abnormal personality). Index and comparison-group probands had their personalities rated using this schedule.

All SAP and SADS-L interviews were conducted by one interviewer (P. McK.). The SADS-L ratings were not made blindly. SAP interviews were scored blindly by Dr A. Mann.

Characteristics of the relatives

There were 61 parents, 75 sibs and 21 children in the obsessive–compulsive group and 72 parents, 74 sibs and 15 children in the comparison group. There were no significant differences between the two groups in sex ratio, age–sex distribution or marital status. The only statistically significant difference was for socio-economic class (SEC), where 16% of the index relatives belonged to SEC IV compared with 4% of the comparison-group relatives. There were on average per family 0.42 and 0.30 children over the age of 16 years for the index and comparison-group probands respectively. Family size was comparable for both groups, with the patient probands having a mean sibship size of 2.40 and the comparison-group probands a mean of 2.48.

Response patterns

In all, 88% of the patients' relatives and 83% of the comparison-group relatives returned a completed GHQ questionnaire. There was no statistically significant difference in the response rates between proband and control relatives as a whole for the GHQ, LOI or HRS (McKeon, 1983). In addition, the response rates for corresponding relatives in both groups (e.g. fathers *v.* fathers) were comparable. As regards those who failed to return the GHQ and HRS, in the case of 9% of the probands' relatives and 14% of the controls' relatives adequate information on their mental health was gleaned from two or more informant relatives.

Results

GHQ scores and history of psychiatric morbidity and treatment

There were 20% of the index relatives identified as possible 'cases', in that they had a score in excess of 4 on the GHQ, compared with 13% of the comparison-group relatives (Table I). Information from HRS and informant relatives indicating a history of psychiatric morbidity is detailed in Table I. The table does not include information gathered at SADS-L interviews. The number who had attended a general practitioner and/or a psychiatrist with psychological symptoms was significantly greater among index probands' relatives than among comparison-group relatives.

When the case-notes of relatives attending psychiatrists both as in-patients and as out-patients were reviewed, a greater number of index relatives were diagnosed as having neurotic depression, phobic neurosis, and depression or manic–depressive illness. Perhaps surprisingly, one case of obsessive–compulsive neurosis was encountered in each group of relatives (Table II).

Prevalence of mental illness based on SADS-L interviews

The reasons why potential 'cases' were given a SADS-L interview are set out in Table III.

The prevalence of mental illness detected at SADS-L interview (Table IV) clearly indicates a statistically significant excess of psychiatric morbidity among the index relatives compared with the comparison-group relatives. This excess is predominantly a reflection of a higher prevalence of neurotic disorders of the anxious, phobic and depressive types. The prevalence of obsessive–compulsive neurosis was similar in both groups of relatives.

So far all the data refer to multiple diagnoses. Enumerating individuals with one or more diagnoses shows that there were 41 cases among the index relatives compared with 21 among the comparison-group relatives (χ^2 8.3, d.f. 1, $P<0.01$).

TABLE I
GHQ scores and history of attendance at treatment facility for psychiatric morbidity

	Index relatives (n = 149)			*Comparison-group relatives (n = 151)*		
	GHQ >4	*GP*	*GP + psychiatrist*	*GHQ >4*	*GP*	*GP + psychiatrist*
Father	2	1	2	3	2	0
Mother	10	4	7	6	3	3
Brother	3	1	5	2	0	1
Sister	11	8	8	4	2	1
Son	1	2	0	0	0	1
Daughter	1	2	1	2	0	4
Total	28**	18	23*	17**	7	10*

*χ^2 10.4, d.f. 2, $P<0.01$.
**χ^2 2.8, d.f. 2, $0.10>P>0.05$.

TABLE II
Primary hospital diagnosis (multiple) (ICD)

Diagnosis	*Index relatives (n = 149)*	*Comparison-group relatives (n = 151)*
Manic depression – depressed type (296.2)	9	3
Neurotic depression (300.4)	7	4
Anxiety neurosis (300.1)	4	4
Phobic neurosis (300.2)	3	1
Obsessive–compulsive neurosis (300.3)	1	1
Schizophrenia (295)	2	1
Non-specific psychotic (295)	1	1
Behavioural disturbance of childhood	1	—
Alcoholism (303)	1	—
Total	29*	15*
Records not available	2	1

*χ^2 7.2, d.f. 1, $P<0.01$.

SAP ratings

Forty-three of the obsessive–compulsive probands had their personalities rated with the SAP; the remaining seven either had no relative to act as informant (four) or had the onset of illness in childhood (three), and so could not have their personalities reliably rated with the SAP.

Personality assessments for the obsessive–compulsive probands showed a high frequency of abnormal personality traits in the obsessional, self-conscious and anxious categories, some two-thirds of these having a grade 1 or 2 score on the SAP obsessional category. The 'overall' personality grade (Fig. 1) refers to the rating irrespective of personality type: probands designated grade 2 personality category had at least one score of grade 2 on any of the three frequently occurring personality categories, those designated grade 1 had one or more grade 1 scores and those in grade 0 had no abnormal personality traits. When the overall personality grades for the index probands were plotted against the prevalence of psychiatric morbidity among their first-degree relatives, it was clear that the rate of family psychiatric morbidity was greater for those probands with grade 2 abnormal personality ratings (Fig. 1). However, neither χ^2 analysis of the data on which the figure is based (χ^2 4.8, d.f. 2, $0.10>P>0.05$) nor the correlation coefficients between the different personality categories and the familial rates of mental illness were statistically significant.

TABLE III
Relatives given SADS-L interview

Screening procedures	*Index relatives*	*Comparison-group relatives*
GHQ score >4	28	17
GHQ score >4 (only)	9	10
HRS report of psychiatric morbidity	41	19
Relative's report of psychiatric morbidity (only)	9	5
Number of 'possible cases' given SADS-L interview	59	34

TABLE IV
SADS-L interview diagnoses of neuroses and affective disorders

	Index relatives (n = 149)	*Comparison-group relatives (n = 151)*
Generalised anxiety state	12	6
Phobia	4	0
Agoraphobia	3	2
Obsessive–compulsive neurosis	1	1
Episodic minor depression	9	7
Chronic and intermittent minor depression	5	0
Major depression	12	5
Schizophrenia	1	1
Miscellaneous	7	3
Neurotic disorders	34	16
SADS-L prevalence of mental illness	54*	25*

*χ^2 10.86, d.f. 1, $P<0.001$.

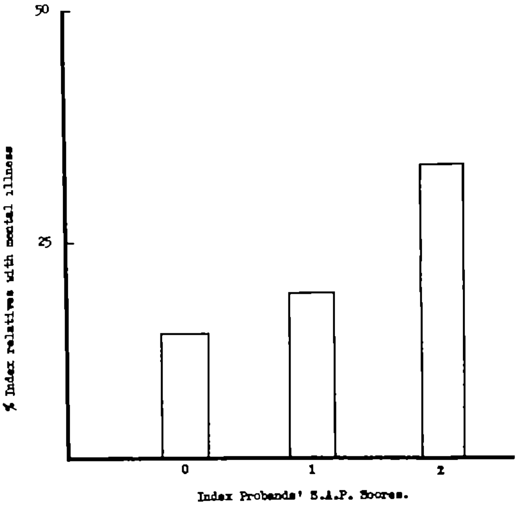

FIG. 1 The rate of family psychiatric morbidity for index probands with different SAP scores.

TABLE V
Mean Leyton Obsessional Inventory scores

	Trait	*Symptom*	*Resistance*	*Interference*
Probands				
Index ($n=50$)	13.0±0.4*	38.0±1.2*	24.3±2.1*	22.5±2.8*
Control ($n=50$)	8.3±0.4	11.5±1.1	3.7±0.6	1.3±0.5
Relatives				
Index ($n=130$)	8.1±0.5	12.8±1.5	3.2±1.0	1.4±0.9
Control ($n=127$)	7.8±0.5	12.7±1.4	3.8±1.7	1.3±1.1

*Unpaired t-test significant at $P<0.001$.

LOI scores

LOI trait, symptom, resistance and interference scores were, as expected, significantly higher for the index probands than for those in the comparison group (Table V). The differences between the scores of index and comparison-group relatives and between the respective relatives for all of the LOI scales did not differ significantly except for the LOI trait score between mothers, with the comparison-group mothers having a higher mean.

Examination of the correlation coefficients between probands and their relatives for mean trait and symptom scores produced few significant results. Within the index group the correlations between index probands and mothers, sisters and sons were positive and high, in contrast to the low positive or negative correlations between index probands and fathers and brothers (McKeon, 1983).

FQ scores

The index relatives' mean scores for agoraphobia, social and global phobia subsections, and their total fear score, were higher than those of the comparison group. However, the differences both between index and control relatives and between respective relatives were not statistically significant.

Birth-order effect

Sixty-six per cent of the males and 56% of the females were first-born or only children, and the later-born/first-born ratio was 0.75 for males and 1.4 for females, suggesting a birth-order difference between the sexes.

As the probands were matched for birth order, the only method of studying the effect of birth order on obsessional symptoms was to compare the LOI symptom scores for first-born (excluding only children) and later-born subjects. The LOI symptom scores for the index probands and their siblings in respect of birth order was as follows: for males, the first-born had a score of 13.7 and those later-born a mean of 10.2 (difference $0.05<P<0.10$) and for females the scores were 16.0 and 14.6 respectively (difference not statistically significant).

The relationship between birth order and rate of family psychiatric morbidity showed that later-borns, both males and females, tended to have more family morbidity. When only-children, first-born and later-born categories were used to examine the relationship between birth order and level of family morbidity, a correlation of +0.35 (NS) was obtained using Goodman's and Kruskal's G (Kendall & Stuart, 1966). G is similar to other non-parametric correlation coefficient formulae, but differs in that it can be used where there are a limited number of ranks, and its applicability in small-sample experiments has been verified.

Discussion

This study showed that the first-degree relatives of obsessive–compulsive patients had a significantly higher rate of lifetime psychiatric morbidity than the relatives of non-psychiatric controls, but that this excess morbidity did not extend to obsessive–compulsive neurosis itself. General Health Questionnaire scores, self- and informant reports of psychiatric morbidity and hospital diagnoses all concurred with the clinical findings of the SADS-L interview, on which the final case count was made. In addition, the similar obsessional trait and symptom scores for the index and control families were in keeping with the clinical results.

There seems to be little doubt that the relatives of index and control probands were comparable on all the important biographical variables. The proportion of relatives in each group completing the various screening instruments, having a SADS-L interview and returning completed questionnaires were comparable. Results from the SADS-L interviews, conducted to assess the reliability of the GHQ, and data on the false positives encountered indicate that as a screening instrument the GHQ was both sensitive and specific. A potential source of bias in this investigation was the absence of blind assessment for the SADS-L interviews, but the inclusion of objective measures such as GHQ, LOI and hospital diagnoses produced useful checks.

Our exclusion of controls who were shown as definite 'cases' by SADS-L requires comment. As the primary aim of this study was to measure the familial rate of clinical and psychometric obsessionality, we considered it essential to screen potential controls for obsessive–compulsive neurosis and affective disorders because of the complex relationship between obsessions and depression and the possibility of a low rate of familial aggregation of obsessional neurosis (Rosenberg, 1967). The fact that the index controls and their relatives had obsessionality scores similar to other normative data justifies this approach (Kelleher, 1972). While the difference in the rates of mental illness between the index and control relatives is possibly magnified by our strict selection of controls, it would seem preferable to have well defined criteria, rather than employ general medical or surgical controls with varying degrees of psychiatric morbidity.

Theoretically, the unexpected finding of obsessional symptoms scores being similar for both groups of relatives might be explained by a tendency of the index relatives to underscore on the LOI, and so present themselves in a socially desirable manner. This is an unlikely explanation, as index relatives scored higher than comparison-group relatives on the GHQ and several sections of the FQ, and reliably reported details of their current and previous mental health. Furthermore, the mean LOI symptom scores for index and comparison-group relatives of 12.8 and 12.7 respectively do not differ significantly from those reported by other investigators; an analysis of 202 normal English subjects produced a mean symptom score of 12.3 (Cooper & McNeil, 1968; Kelleher, 1972).

While the higher prevalence of mental illness detected among the index probands' relatives in this study concurs with other obsessional family studies (Brown, 1942; Rudin, 1953; Greer & Cawley, 1966; Rosenberg, 1967), the low rate for obsessive–compulsive neurosis (0.7%) detected among the parents and siblings contrasts markedly with the findings of Brown (7.3%) and Rudin (3.2%). However, Rosenberg reported a prevalence rate of 0.4%, which is in keeping with the finding in this study. How are these different rates to be explained? The most likely explanation is that the different diagnostic criteria used to define 'case' status account for the disparity: both Brown and Rudin employed less stringent criteria than those of the Research Diagnostic Criteria, while Rosenberg relied in the main on hospital case-notes, compiled presumably by less biased observers.

The correlation coefficients for obsessional symptoms between probands and their relatives did not reach statistical significance, but it is interesting to note that the correlations of +0.32 between index probands and their mothers is comparable with the correlation between a small group of monozygotic obsessional twins (McKeon, 1983). In a study of five monozygotic and five dizygotic twin pairs, where one of each pair had been diagnosed as having obsessive–compulsive neurosis, the correlation coefficient for obsessional symptoms on the LOI was 0.39 for the MZ twins and 0.07 for the DZ pairs. While the higher correlation between MZ pairs might suggest that genetic factors are of prime importance, it must be weighed against a similar correlation between the index probands and mothers in the present study.

In the same twin study, the mean difference between twins on the Eysenck Personality Inventory neuroticism scale was greater for the DZ than the MZ pairs, and the DZ/MZ ratio for these differences was 1.5, compared with a similarly calculated ratio of 1.4 for LOI symptoms scores. While the numbers involved are too small to allow firm conclusions to be drawn, these results suggest that neuroticism is as likely as obsessionality to be the inherited factor.

Snowdon's (1979) finding of a birth-order effect for male obsessive–compulsive patients was here confirmed, with a hint of a similar effect being present for females. The association between birth order of the index probands and the level of familial morbidity suggests that being first-born contributed to the development of obsessive–compulsive neurosis, even in the absence of significant family psychiatric morbidity.

In conclusion, this study indicates that the prevalence of mental illness in general, and neurotic disorders in particular, is increased in families of those with obsessive–compulsive neurosis, but questions the notion that this particular disorder breeds true. Furthermore, the positive correlation between the extent of familial aggregation and the index probands' pre-morbid personalities suggests that the familial neurotic tendency may contribute to the pre-morbid personality abnormalities noted in two-thirds of the obsessive–compulsive patients. Recent obsessional twin studies (Carey & Gottesman, 1981; Torgensen, 1983) indicate that the concordance rate for neurotic disorders between MZ twins is higher than that for DZ pairs but that there is little evidence that this higher concordance applies to obsessive–compulsive neurosis. A possible interpretation of these twin studies and the results of the present investigation would be that the familial neurotic tendency is inherited, that it is manifest in a subclinical form in the subject's pre-morbid personality, and that whether or not neurotic decompensation occurs will depend on the interaction between the stressful life events and personality traits (McKeon *et al*, 1984). The form the neurotic illness may take will be influenced by factors such as birth order and early life experiences.

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Anxiety Management for Persistent Generalised Anxiety

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A preliminary controlled investigation of the effectiveness of Anxiety Management as a treatment for generalised anxiety disorder (GAD) is described. Patients with a primary diagnosis of GAD, in which the current episode had lasted for at least 6 months but not more than 2 years, were included. Anxiety Management, a self-help treatment including procedures for managing somatic and cognitive symptoms, and for dealing with avoidance and low self-confidence, was given either immediately or after a 12-week waiting period. The average length of treatment was 8.7 sessions. Highly significant changes in anxiety, depression, and problem ratings were shown after treatment. These changes were replicated when the waiting list group had also received treatment, and gains were maintained by both groups for 6 months. Similar degrees of improvement and maintenance of change were shown in subgroups with and without minor depressive disorder or recurrent panic attacks.

It is now generally agreed that anxiety disorders should not be treated for long periods with anxiolytic drugs because of their doubtful efficacy and because tolerance to and dependency on the drugs may develop (e.g. Tyrer & Owen, 1984; Catalan & Gath, 1985). Although about two-thirds of general-practice patients with minor affective disorders, mostly anxiety states, recover within 6 months, the remaining third remain unwell for some time (Catalan *et al*, 1984). There is a need therefore for a simple effective psychological treatment for cases which do not recover rapidly. This treatment should be of a kind that can be provided within the resources of the National Health Service. Relaxation is widely taught, but although the procedure has been shown to have a significant short-term anxiolytic effect (Mathews & Gelder, 1969), it has not been shown to have substantial long-term effects in anxiety disorders; indeed the clinical impression is that long-term effects are not impressive. This may be because patients do not practise persistently (Hoelsher *et al*, 1984), or because relaxation training fails to deal with two components of the anxiety syndrome which appear to be of particular importance: cognitions and avoidance behaviour.

Beck *et al* (1974) showed that patients with anxiety disorders have characteristic cognitions concerning personal danger. In generalised anxiety neurosis these thoughts most frequently concern loss of self-control, inability to cope, illness and social embarassment (Hibbert, 1984). Such fears are usually recognised by the patient to be exaggerated or irrational (Rapee, 1985). Two kinds of treatment have been devised to deal with these cognitions: Anxiety Management Training (Suinn & Richardson, 1971) focuses on patients' fears of being unable to cope; Beck's cognitive therapy (Beck *et al*, 1985) makes a more ambitious attempt to alter patients' ways of thinking.

In Anxiety Management Training, fears of being unable to cope are allayed by teaching ways of coping with symptoms of anxiety. This method combines relaxation, reassuring 'self talk', and practice in the use of anxiety-provoking imagery to induce anxiety, which is then brought under control by the use of reassuring imagery ('image switching'). A clinical trial of this method for patients with persistent anxiety neurosis (Jannoun *et al*, 1982) demonstrated a moderately large therapeutic effect, which persisted during 3 months' follow-up. A small number of studies has been reported of other methods of changing cognitions, although all but one was uncontrolled (Barlow *et al*, 1984; Clark, 1986; Ost, 1985; Waddell *et al*, 1984; Woodward & Jones, 1980). The results were similar to those obtained with Anxiety Management Training.

Patients in the study of Jannoun *et al* (1982) reported that image switching was difficult to carry out. Moreover further (unpublished) work of our own showed that although patients with generalised anxiety disorders do not have the 'focused' pattern of avoidance seen in phobic states, they often show partial avoidance of a large number of different situations. We therefore removed image switching from the treatment and added exposure, to deal with the avoidance behaviour. At the same time we modified the cognitive component of the treatment to place more emphasis on the identification of and response to each person's specific thoughts, rather than drawing on a common set of thoughts, or single strategy, for all patients. We called the new treatment Anxiety Management. It was presented to patients with a rationale designed to mobilise their existing resources for coping with anxiety. Information was included about the nature of anxiety and about each of the component techniques, namely those for controlling symptoms, bringing about exposure and building confidence. An earlier version of this treatment has already been shown to be a useful addition to exposure in the treatment of social phobia (Butler *et al*, 1984). The main aim of the study reported here was to carry out a controlled evaluation of Anxiety Management.

Method

Patients

Forty-five patients with generalised anxiety disorder (GAD) as defined by the Research Diagnostic Criteria (Spitzer *et al*, 1978) entered the study. Patients meeting the criteria for panic disorder as well as GAD were included provided that GAD was the primary disorder. In addition, patients were included only if the anxiety reached 7 or more on the Leeds Anxiety Scale (Snaith *et al*, 1976), and if the current episode had persisted for at least 6 months.

Patients were excluded from the study if their symptoms met Research Diagnostic Criteria for phobic, obsessive–compulsive or major depressive disorder, or if the disorder was so chronic that there had been no period of one month in the last 2 years during which they were free from symptoms. Patients who reported recurrent but discrete episodes of GAD were included. The inter-rater reliability of the diagnostic and inclusion criteria was checked by a second psychiatrist, who rated a consecutive series of ten patients on the same occasion as the assessor; the two assessors agreed about the application of these criteria in all but one case. Successive referrals were accepted until 45 suitable patients had entered the study. They were randomly allocated to one of two groups and to one of three therapists. Three patients were replaced because they had attended only two treatment sessions after one month.

Therapists

Three clinical psychologists, each with extensive experience in the psychological treatment of anxiety disorders, treated equal numbers of patients. Before the trial the therapists completed a training period, using an instruction manual, during which they developed a consistent approach to therapy. During the trial each therapist tape-recorded a sample of therapy sessions. Regular meetings were held, to discuss issues arising during treatment, and to help maintain a consistent approach to therapy throughout the study.

Procedure

Patients were referred to the study either directly by their general practitioners or from psychiatric out-patient clinics. An independent assessor interviewed the patients, made the diagnosis and assessed the patients' progress. The assessor did not know which therapist was treating each patient or whether the patient was in active treatment or on the waiting list. Suitable patients were randomly allocated to immediate treatment with Anxiety Management (AM), or to a waiting list (WL). Patients in the WL group entered treatment after 3 months. Treatment was given individually, and consisted of between four and twelve sessions, according to the patient's need (see below), each session lasting not more than one hour. Booster sessions were given 2 weeks and 6 weeks after the end of treatment. A post-treatment assessment was carried out 3 months after the start of treatment, and follow-up assessments were conducted 3 months and 6 months later. The first and last of these assessments were carried out by the independent assessor, and the 3-month follow-up assessment was completed by the therapist. Some patients were seen at the hospital, some in health centres.

Treatment

Treatment was described in a booklet which was given to patients shortly before their first therapy session. The treatment was presented as a form of self-help designed to mobilise the patient's own resources for managing symptoms of anxiety. The booklet starts with information about the nature of anxiety and its common manifestations, and contains questions which require the patient to apply the ideas in the booklet to his or her own difficulties. The account emphasises that it is normal to feel anxious at times, and that anxiety becomes a problem only when it does not subside appropriately. The booklet goes on to suggest that patients should seek out maintaining factors and try to eliminate them. Maintaining factors include anticipatory anxiety, fear of fear, avoidance, and loss of self-confidence. Specific techniques are then described to counteract each of these maintaining factors. Anticipatory anxiety and fear of fear are counteracted by learning to control symptoms with relaxation, distraction, and the control of upsetting thoughts. The latter involves identifying thoughts and images that provoke, maintain, or exacerbate anxiety, examining them to find out how realistic they are and whether there are more helpful or more realistic ways of thinking, and devising practice tasks to test the thoughts. Avoidance is reduced by using graded practice. Self-confidence is increased by identifying the person's strong points, by engaging in rewarding and pleasurable activities, and by paying attention to aspects of life in which the person is functioning relatively well. Copies of the booklet are available from the first author.

Throughout treatment, patients were encouraged to play an active role by setting their own goals, deciding which techniques would be appropriate for them, planning homework assignments and monitoring anxiety. Tape-recorded instructions for progressive muscular relaxation were provided at the first session. The exercises were practised with the therapist as well as at home if either the patient or the therapist thought this would be helpful. Techniques for controlling symptoms were introduced one at a time, and practised both separately and while graded practice and activities to increase confidence were being carried out. All patients were encouraged to reduce their consumption of anxiolytic medication.

Patients could stop treatment after a minimum of four sessions provided that they were no longer distressed by symptoms of anxiety, their anxiety ratings had been stable for at least 2 weeks, and the therapist shared the patient's confidence that he or she could control symptoms. During the booster sessions, strategies for managing anxiety were reviewed and the patient was prepared for dealing with future fluctuations in anxiety, or other setbacks.

Measures

Mood ratings

The assessor rated both anxiety and depression with the Hamilton Scales (Hamilton, 1959, 1967) and with the 9-point rating scale developed by Watson & Marks (1971). Patients rated anxiety and depression with the Leeds Scales (Snaith *et al*, 1976) and the 9-point rating scale. They also completed the State–Trait Anxiety Inventory (STAI; Spielberger *et al*, 1970).

Expectations

Expectations about the outcome of treatment were rated by patients on a 0–8 scale after reading the booklet, and again 4 weeks later. Patients also rated the suitability of treatment on a 0–8 scale before and after treatment.

Background and descriptive data

The assessor completed the Present State Examination (PSE; Wing *et al*, 1974) at the first assessment and at 6-month follow-up. Patients completed the General Health Questionnaire (GHQ; Goldberg & Hillier, 1979) at each assessment, and also rated the severity and degree of interference with their lives of the three problems that they found most troubling. These were identified with the help of the assessor, and rated on a 0–3 scale.

At the first interview the assessor completed a structured interview designed to collect information about situational anxiety, avoidance, anticipatory anxiety, and the presence of anxiety-related thoughts. He also completed the Life Events Checklist (Paykel *et al*, 1969) and the Index of Social Support (Surtees, 1980). Findings related to these measures are presented in Butler & Gelder (1987).

Within-treatment measures

Patients rated anxiety (0–8) at every therapy session, and completed the Leeds Scales at the fourth session. At each session, therapists recorded the amount of medication taken since the last session, the number of reported panic attacks, the length of the session, techniques discussed and homework set.

Statistical analysis

Initial differences between the groups and change within-groups were analysed with *t* tests. Between-group differences were tested with analysis of covariance, taking the score on the first occasion of assessment as the covariate. Repeated-measures analysis of variance was used to analyse changes within the WL group over the first three assessments: assessment one, pre-treatment and post-treatment. Multivariate analysis of variance was used to evaluate the response to treatment of different subgroups.

Results

Patients

There were 144 patients referred to the study. Of these, 81 failed to meet the inclusion criteria: 28 had anxiety which was too mild, or too short a history; 12 had too long a history; 21 were phobics (14 of whom were agoraphobic); 10 met the criteria of major depressive disorder; and 10 had various other disorders. Of the remaining 63, 18 did not or could not attend, or preferred other treatment. Thus 45 patients (29 females and 16 males) entered the study. The average age was 37.4 years (range 20–57). The average school-leaving age was 16.3 years (range 15–19). Thirty-six patients were married, five single and four divorced or separated. Of all the patients, 82% were rated as cases according to the PSE at the time when they were accepted into the study. Using the PSE definition of panic attacks, 13 patients fulfilled the criteria for panic disorder.

There were 22 patients in the AM group, and 23 in the WL group. About half the patients were treated at health centres, and the rest at out-patient clinics. Three patients in the WL group did not receive treatment after the waiting period. Of these, two had to be withdrawn because they became significantly more depressed during the waiting period, and a third refused treatment because he had improved; a fourth patient finished treatment but declined to complete any further assessments. Therefore post-treatment ratings are available from only 19 patients in the WL group. At the end of the follow-up period one patient could not be traced and a further two, one in each group, had to be excluded because they had received antidepressant medication during the interval since treatment had ended. Thus at 6-month follow-up the total sample consisted of 38 patients, 20 in the AM group and 18 in the WL group.

There was no significant difference between the ages of the two groups (Table I). There were 14 women and 8 men in the AM group, and 15 women and 8 men in the WL group. Before treatment (Table I) there were no differences between the groups on any of the main measures, including the Leeds and Hamilton scales, the 0–8 ratings of both anxiety and depression, the STAI ratings of state and trait anxiety, and the GHQ ($P > 0.10$ in every case).

Expectations of improvement, rated by both patients and therapists, were moderately high before treatment started (mean = 5.49 on the 0–8 scale), and remained at this level when re-rated at the fourth session. Similarly, initial ratings of treatment suitability were moderately high (mean = 5.47 on the 0–8 scale), and did not change after treatment. Neither of these ratings differed significantly between groups or between occasions. There were no differences in response to treatment according to therapist or sex of patient, and these variables will not therefore be discussed further.

TABLE I
Main outcome measures: mean values

	Assessment 1		*After wait*	*After treatment*		*6-month follow-up*	
	AM[1] (*n*=22)	*WL*[2] (*n*=23)	*WL* (*n*=23)	*AM* (*n*=22)	*WL* (*n*=19)	*AM* (*n*=20)	*WL* (*n*=18)
Age	36.5	38.3	38.3	36.5	—	—	—
Anxiety							
0–8 (assessor)	5.5	5.6	5.2	2.6	2.7	1.9	2.4
0–8 (patient)	4.9	5.0	4.5	2.0	2.3	1.6	1.7
Leeds	11.5	12.7	12.4	6.7	6.8	5.0	6.4
Hamilton	16.0	17.4	15.4	6.6	7.6	5.0	8.0
STAI-S	58.0	54.6	57.1	43.9	41.8	38.9	41.5
STAI-T	55.8	54.8	—	—	—	43.7	38.9
Depression							
0–8 (assessor)	2.4	2.7	2.6	0.8	1.1	0.7	0.8
0–8 (patient)	2.7	3.0	2.7	0.9	1.7	0.9	1.2
Leeds	9.1	8.0	8.4	4.6	5.4	3.6	4.2
Hamilton	11.1	12.0	9.6	4.3	4.1	2.9	4.6
Problem ratings							
Severity	2.1	2.0	2.0	1.1	1.1	1.0	1.1
Interference	1.7	1.4	1.5	0.8	0.7	0.7	0.7

1. AM = Anxiety Management.
2. WL = waiting list.

Main between-group comparison

The pattern of changes over the whole period of the study is shown in Fig. 1, and the mean outcome measures for the main occasions of rating are shown in Table I. Data from the first two assessments were used for this analysis. In the interval between these assessments the AM group had completed treatment and the WL group had completed the waiting period.

For all the main measures, the scores of the two groups differed significantly at the second assessment ($P<0.001$ in every case). The AM group changed significantly on every measure ($P<0.001$). The WL group did not change significantly on eight of the nine measures of mood. Only the depression ratings of the Hamilton Rating Scale were significantly less after than before the waiting period, ($t=2.52$, d.f. 22, $P<0.05$).

Changes in the WL group after treatment

Data from the WL group alone were subjected to repeated-measures ANOVA for the first three occasions of assessment (occasion one, pre-treatment and post-treatment). This analysis showed a main effect of occasions on every measure ($P<0.001$). The differences between the mean scores on each occasion were then tested with Tukey tests. For eight of the nine measures, ratings did not differ between the first and second occasion (i.e. the waiting period) but they changed significantly during treatment ($P<0.01$ in every case). Despite a significant overall change in the patients' 0–8 rating of depression, neither the change during the waiting period nor that during treatment reached significance when tested separately.

Differences between groups after the WL group had received treatment

This analysis was designed to find out whether the effects of treatment in the AM group were replicated in the WL group when it subsequently received treatment. Between-group differences were tested with ANCOVA, using data from the first assessment as the covariate for both groups. On eight of the nine measures there were no differences between the groups after both had received treatment. On the ninth measure, the patients' 0–8 rating of depression, the WL group had significantly higher scores than the AM group ($F=5.57$, d.f. 1, 38, $P<0.05$). Patients in the AM group received a mean of 8.9 treatment sessions compared with 8.4 for those in the WL group; this difference was not statistically significant.

Analysis of the problem ratings was conducted in the same way as the analysis of mood ratings (mean scores are included in Table I). During treatment these ratings decreased significantly in both groups.

Maintenance of change

In the AM group, improvement was maintained during follow-up on eight of the nine measures of mood, and on the problem ratings. A significant further improvement was detected in ratings of the Leeds Anxiety Scale ($t=2.11$, d.f. 18, $P<0.05$). In the WL group, improvement was

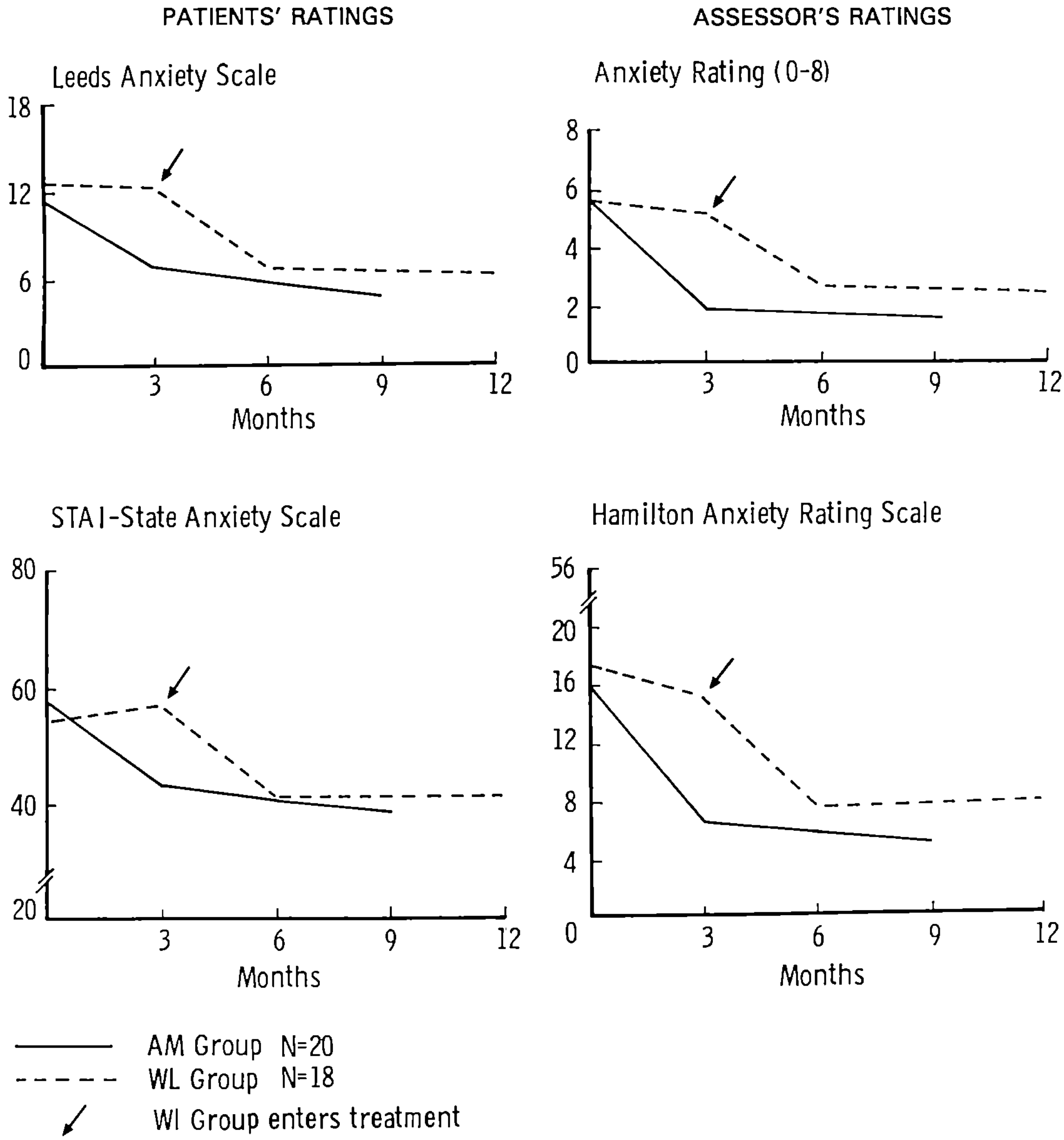

FIG. 1 The pattern of changes in measures of anxiety during the whole period of the study ($n = 38$).

maintained on every measure during follow-up. At the 6-month follow-up the groups did not differ significantly on any of the measures.

Summary of comparisons and changes

In summary, there were no differences between the groups either at the initial assessments or after the WL group had completed the waiting period. Both groups improved significantly with treatment. The groups did not differ significantly after treatment or at follow-up on any of the measures of anxiety. For this reason, in subsequent analyses data from both groups were combined wherever appropriate.

Other indices of improvement

By the end of treatment the scores of 21 patients (51%) fell below the cut-off between patient and non-patient populations on the Leeds Scales. By the end of follow-up 24 patients met this criterion, and at this time only 8 patients were still diagnosed as cases according to the PSE (mean total scores were 16.2 at the start of the study and 4.9 at the end). Mean scores on the GHQ for the whole group were 25.7 before treatment, 7.6 after treatment and 7.6 at 6-month follow-up. Initial scores on the STAI-trait anxiety scale at assessment one and 6-month follow-up are shown in Table I; on neither occasion was the difference between the groups significant.

Additional information was collected from general-practice records after the trial had ended. The 6-month follow-up period was thus extended by about a year (range 5–22 months). Records were available for 36 patients (95% of those included in the 6-month follow-up); the remaining two were untraceable. Just under two-thirds (22 patients) had had no further contact with their general practitioner because of anxiety. A further five patients had consulted their general practitioner occasionally because of anxiety (mean = 1.8 times). Two of these were reassured after discussion, and three were given anxiolytic medication to help with particular stresses; for example, examinations. The remaining nine patients, a quarter of the sample, had received more extensive treatment for anxiety, including taking regular anxiolytic medication for at least part of the follow-up period. Seven of these had also received antidepressant medication. Despite additional treatment, only two of these nine patients were reported as improved.

Changes in the use of medication

At the start of the study, 46% of the patients were taking regular medication. Of the prescriptions, 73% were for anxiolytics, 14% for hypnotics, and the remainder for antidepressants and combined preparations. During the waiting period three patients started to take regular medication and two ceased to do so. There was no difference between the groups in frequency of drug use. After treatment 16% of the patients were still taking regular medication but had decreased the dose. Six months later this had fallen to 13%. Two patients, 10% of those originally taking regular medication, failed to reduce their medication throughout the study.

Comparison of patients with and without minor depressive disorder

The group was then divided according to whether or not they had fulfilled the criteria for minor depressive disorder at the start of the study. The 18 patients (44%) with minor depressive disorder as well as GAD were compared with the 23 who had GAD alone. These two subgroups did not differ in age, or in the mean number of treatment sessions (depressed 9.0, non-depressed 8.5). Although 81% of the depressed and only 50% of the non-depressed patients were women, this difference was not significant. Multivariate analysis of variance, using data from the main measures at the first assessment showed that the depressed patients had more severe symptoms than the non-depressed patients at the start of the study ($F=5.79$, d.f. 9, 31, $P<0.001$). Multivariate analysis with repeated measures showed a highly significant main effect of time ($F=78.9$, d.f. 2, 35, $P<0.001$). Both subgroups improved during treatment and maintained this improvement during the follow-up period. There was no main effect of depression, but a significant time × depression interaction ($P<0.02$) indicated that the depressed patients tended to improve more during treatment, and hence no longer differed from those who were not depressed either immediately after treatment or at 6-month follow-up.

Comparison of patients with and without panic attacks

At the time of the first assessment, 21 (47%) of the 45 patients had recurrent panic attacks (i.e. at least one every 3 months), and 19 (42%) had never had a panic attack. (Panic attacks were defined as in the PSE.) Five patients (11%) had had rare, isolated attacks. In the WL group, 9 patients (39%) reported having recurrent panic attacks at both the first and the second assessment, and there was no significant difference between the estimated weekly frequency of attacks on the two occasions. Twenty of the 41 patients treated had recurrent panics. Their mean attack frequency was 1.76 per week before treatment, 0.23 after treatment and 0.10 at 6-month follow-up; by this time attacks were generally occurring so seldom that it was difficult to estimate their frequency accurately. However, of the 20 patients who had recurrent attacks at the start, 14 (70%) reported that they had had no attacks for a period of at least 3 months. The frequency of panic attacks before treatment and at 6-month follow-up is shown in Table II.

TABLE II
Panic attack frequency before treatment and at 6-month follow-up

	Pre-treatment (%)	*6-month follow-up (%)*
⩾1 a week	65	0
⩾1 a month	25	20
<1 a month	10	10
0 in 3 months	0	70

In order to compare the response to treatment of those with and those without recurrent panic attacks, the AM and WL groups were combined, omitting the five patients who had rare panic attacks. As noted above there were 20 patients in the panic attack group and 16 in the panic-free group when treatment began. At 6-month follow-up the two groups comprised 18 and 15 patients respectively. Comparison of these two groups using multivariate analyses shows that the patients with panic attacks tended to have marginally ($P=0.07$) more severe symptoms at the start of the study. Treatment effects were highly significant and similar in both groups, and the improvement during treatment was maintained during follow-up. There was no difference between the groups on any subsequent occasion.

Comparison of patients receiving shorter and longer treatment

In order to find out whether the patients who required the shortest period of treatment could be identified at the start of the study, the whole sample was also grouped according to length of treatment. When the group was divided at the mean number of sessions (8.7), there were 20 patients in the short treatment group (mean 6.3 sessions) and 21 in the long treatment group (mean 11.0 sessions). At the 6-month follow-up there were 18 and 20 patients in the two subgroups. The multivariate analyses showed that those who had longer treatment started with more severe symptoms. There were main effects of both time and treatment length, without any interaction between these factors, showing that both groups improved to the same extent, and maintained this improvement during follow-up.

Discussion

The main aim of this study was to find out whether Anxiety Management is an effective treatment for generalised anxiety. The findings presented here show that patients with GAD treated with Anxiety Management changed more than a matched group waiting for treatment. The changes during treatment were highly significant, and were replicated almost exactly when patients in the waiting list group completed treatment. In both cases the effects of treatment persisted throughout the 6-months follow-up. By the end of the study the anxiety ratings of half the patients had fallen below the cut-off point that distinguishes a normal from a clinical population on the Leeds Anxiety Scale. The improvement was shown in measures of depression and of patients' problems, as well as in measures of anxiety. We cannot say how far our population after treatment differs from the normal population because the relevant norms are not available. Nevertheless, it seems reasonable to conclude that Anxiety Management has a substantial effect. An appropriately designed comparative treatment trial will have to be carried out in order to ascertain the specificity of the treatment, and to identify its most effective parts.

Particular care was taken to ensure that the diagnosis of generalised anxiety disorder was reliable, and to exclude phobic patients with generalised anxiety for whom exposure might have been the treatment of choice. The group was homogenous, in so far as only those with anxiety that had persisted for at least 6 months were included. Patients with more chronic disorders were excluded because they are difficult to distinguish from those with personality disorders, and because many complain of depression as well as anxiety. We excluded those chronic patients whose symptoms had been continuously present for 2 years. However, some of those included had had previous discrete episodes of anxiety and had high trait anxiety scores; they might therefore fulfil less stringent criteria for chronicity. Our results cannot be taken to apply to patients with more chronic anxiety, and this group requires further study.

It might be supposed that patients who are depressed and anxious would respond less well than those with anxiety alone. In these cases depression might be the primary disorder, despite the presenting complaint of anxiety; Anxiety Management might not help overcome coexisting depression or depression might reduce the vigour with which patients would carry out Anxiety Management procedures. In keeping with this idea, there is some evidence from this study that the initial level of depression is important. Thus, two patients had to be withdrawn from the study because they became significantly more depressed during the waiting period and urgently required other treatment. Two others continued to ask for help for depression during the follow-up period, and received antidepressant medication. The remaining patients were about equally divided between those who were only anxious and those who were anxious and fulfilled the criteria for minor depressive disorder (i.e. depressed mood persistent for a minimum of 2 weeks, and at least two symptoms from a checklist of 16). These two subgroups were not significantly different after treatment or at follow-up, suggesting that Anxiety Management is effective in both kinds of case.

About half of our patients had recurrent (though not very frequent) panic attacks. The frequency of attacks declined substantially with treatment, and there was no difference between subgroups with and without panic attacks either in their immediate response to treatment, or in the maintenance of change. This may be because lowering the level of general anxiety makes panic attacks less likely. This would be the case if panic attacks are merely an index of severity of anxiety, rather than an indication of a distinct form of anxiety disorder. It may also be because Anxiety Management, which contained instructions on how to manage panic attacks, helped patients to re-evaluate the significance of the symptoms they experienced during attacks, and helped them to control these symptoms, perhaps by reducing their fear of possible catastrophic consequences.

Even though it is not yet known which components of Anxiety Management are effective, this brief and simple treatment is clearly suitable for patients with persistent generalised anxiety disorder, who are commonly seen in general practice and for whom prolonged drug treatment is unsatisfactory. Cognitive therapy (Beck *et al*, 1985) and combinations of relaxation and cognitive treatment (e.g. Barlow *et al*, 1984) have also been proposed for anxiety disorders, but they have not yet been evaluated in controlled trials. These treatments may be more complex and in most cases last longer than Anxiety Management, which requires about half as much therapist time as the treatment described by Barlow *et al.* Anxiety Management is easily taught to therapists, and readily understood by patients. It is also a rational method of treating generalised anxiety disorders because, as well as including procedures for controlling symptoms, it deals with anxious cognitions and avoidance behaviour, both of which appear to contribute to the maintenance of anxiety disorders.

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Suicide Following Parasuicide in Singapore

W. F. TSOI and E. H. KUA

Suicide was attempted by 1641 people (502 males and 1139 females) in Singapore during 1980. Eleven of these committed suicide during the following three years, giving a suicide rate of 0.67%. Of the eleven suicides, five had schizophrenia and three depression. The risk of suicide in the year following attempted suicide is lower in Singapore (0.5%) than in Britain (1.6%). High risk factors for the Singapore patients were being Chinese, being male, and having a mental illness.

Singapore is an island republic of 610 km^2. In 1980 it had a population of 2.4 million, comprising Chinese (77%), Malays (15%), Indians (6%), and other ethnic groups (2%). The aim of this study was to find out how many patients who attempted suicide in 1980 died of suicide during the following three years. This may throw some light on those patients who are a high suicide risk.

A follow-up study on the fate of attempted suicides has not previously been carried out in Singapore. A study of the fate of all the attempted suicides in one year for a whole country is not easy to carry out; it is possible in Singapore because (a) it is an island with a non-mobile population, (b) it has a unique registration system, in which every Singapore resident carries only one identification number which is used both for hospital registration and death registration, and (c) almost all its attempted suicides are admitted to government general hospitals for treatment.

Suicide statistics in Singapore

The first comprehensive study of suicide in Singapore was by Murphy (1954). He analysed data from the period 1925–1952 and concluded that there had been a steady increase in the suicide rate since 1948. A subsequent study by Chia (1978) showed that over the previous 80 years the suicide rate had been about 10 per 100 000, with a peak of 15.5 per 100 000 during the period 1936–1940. The commonest method of suicide was caustic soda poisoning (43%) in 1950 (Murphy, 1954), hanging (49%) in 1961 (Chia & Tsoi, 1972), and jumping from heights in 1980 (Tsoi & Kok, 1982). The commonest predisposing causes were believed in all three studies to be mental and physical illness.

Murphy (1954) also studied attempted suicide in Singapore, and analysed 34 cases from police records. The commonest method was caustic soda ingestion (34%). Tsoi (1970) investigated 192 cases in a general hospital, and showed that household poisons such as bleaching liquids, washing detergents, and insecticides accounted for 57% of the cases. The two most recent surveys (Chia & Tsoi, 1974; Tsoi & Kok, 1981) showed that the commonest method was drug overdose (57% and 61% respectively). This shows a trend towards the use of less dangerous substances.

Method

All cases of attempted suicide admitted to the four major government general hospitals in Singapore between 1 January 1980 and 31 December 1980 were recorded. Attempted suicide, in this survey, was defined as all non-accidental self-poisoning and self-injury. This did not include a small number admitted to private hospitals or those who were treated at home or in out-patient clinics. Details of all cases of suicide between 1 January 1980 and 31 December 1982 were obtained from the records in the Coroner's Court. The Court in Singapore is based on the English legal system, and a verdict of suicide by the Coroner reflects a legal decision in which an important element is that the deceased intended to take his own life – evidence of such intent is necessary before classifying a death as suicide. If information obtained is insufficient to enable the Coroner to arrive at a decision, an open verdict is returned. During the period of study there was one Coroner. The names and identification numbers of the suicide and attempted suicide patients were matched.

Results

During 1980 1641 persons attempted suicide. Of these, 46 (2.8%) made second attempts and 12 (0.7%) made three

TABLE I
Attempted suicides in 1980, categorised according to ethnic group

Ethnic group	*Males*		*Females*		*Total*	
	n	*Rate per 100 000*	*n*	*Rate per 100 000*	*n*	*Rate per 100 000*
Chinese	365	39	842	91	1207	65
Malay	27	14	53	31	80	23
Indian	94	107	192	288	286	185
Others	16	59	52	209	68	132
Total	502	41	1139	96	1641	68

TABLE II
Attempted suicides in 1980, categorised according to age group

Age group	*Males*		*Females*		*Total*	
	n	*Rate per 100 000*	*n*	*Rate per 100 000*	*n*	*Rate per 100 000*
10–19	72	27	214	84	286	55
20–29	231	82	581	217	812	148
30–39	96	55	205	120	301	87
40–49	44	37	62	53	106	45
50–59	11	13	26	33	37	22
60–69	21	39	13	23	34	31
70+	12	44	13	35	25	39
Unknown	15	—	25	—	40	—
Total	502	41	1139	96	1641	68

or more attempts. There were 502 males and 1139 females (ratio = 1:2.3). Their distribution by ethnic group is given in Table I, and by age in Table II.

Of the 1641 attempted suicide patients, 11 (8 males and 3 females) died of suicide during the next three years (8 in the first year, 2 in the second year and 1 in the third year). The methods of suicide were jumping (6), hanging (4) and poisoning (1). They were all Chinese, and their mean age was 32.1 years (s.d. = 12.3 years), which was slightly higher than that for the attempted suicides (mean = 30.6 years, s.d. = 17.4 years). Seven (64%) suffered from mental illness (five from schizophrenia and two from depression). The data therefore showed that in Singapore only a small proportion of attempted suicides (0.67%) died of suicide during the following three years.

Discussion

The attempted suicide rate in Singapore was highest among the Indians and lowest in the Malays. The main reason for attempted suicide was relationship problems, such as the threat of separation from boyfriends or girlfriends (Tsoi & Kok, 1982). Such problems affect the Indians more because they are a minority group and the choice of marital partners within the same ethnic group is limited. The main reason for the low rate among the Malays was their Islamic religion – for Moslems, suicidal behaviour is a sin.

Follow-up studies of attempted suicides indicate that only a small percentage will subsequently commit suicide; Stengel (1971) showed that rates varied from 0.05% to 13.2%. The wide variation was due mainly to the nature of the original parasuicide samples. If the samples consisted of high risk cases such as those admitted to mental hospitals, the subsequent suicide rate would also be high. Three studies (Kessel & McCulloch, 1966; Buglass & McCulloch, 1970; Pierce, 1981) followed up unselected general hospital referrals; they found rates of 1.6% for one year, 3.3% for 3 years, and 2.9% for 5 years respectively. These studies show that the risk of subsequent suicide after attempted suicide is 3–5 times higher in Britain than in Singapore, where the rates are 0.5% after one year and 0.67% after 3 years. For the Singapore patients, high suicide risk was found to be associated with being Chinese, being male, and having a mental illness.

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Can Psychiatric Nurses 'Catch' Schizophrenia?

The incidence of schizophrenia among psychiatric and general trained nurses was investigated for the period 1955–1979 in Northern Ireland. No difference was found. These results do not support the hypothesis of schizophrenia as a horizontally transmitted infectious disease.

The hypothesis that viral infection might either be the cause of or one of a number of aetiologies of schizophrenia has received greater attention in recent years. Crow (1984) and King *et al* (1985*a*) have reviewed the history of the hypothesis, which was initially suggested by the model of post-influenzal psychosis (Menninger, 1926). There are four principal suggestions as to how viruses might be involved. The most widely investigated is the possibility of a latent infection with a common virus, initiated perhaps in the perinatal period or as a result of impaired immunity (Torrey *et al*, 1978; De Lisi *et al*, 1981; King *et al*, 1985*a*,*b*). Another hypothesis, as yet only barely investigated, is of infection by uncommon viruses, such as arborviruses. A third hypothesis suggests the possibility of transmission of an unknown infective agent from schizophrenic patients to susceptible individuals (Crow, 1983*a*); and a fourth hypothesis, also proposed by Crow (1984), suggests the possibility of retroviral integration into the genome.

This study was designed to investigate the third of these hypotheses, the so-called 'contagion hypothesis'. On this hypothesis we would predict that people in close contact with schizophrenic patients would be more likely to develop the illness. Apart from family members, where the complication of genetic factors makes investigation of the hypothesis difficult, a useful group to study are psychiatric nurses. We have examined the rates of schizophrenia in psychiatric hospital nurses compared to general hospital nurses from 1955 to 1979.

Method

In Northern Ireland a computerised Central Mental Health Records Scheme (CMHRS) has been in operation since 1962. Psychiatric hospitals send details of all their hospital admissions and out-patient clinic referrals to a central office. The information provided includes simple demographic data, such as age, sex and occupation, as well as date of admission or referral, length of illness and diagnosis. The patient's name is not included. Each patient receives a Central Record (CR) number, and information relating to subsequent readmissions or re-referrals to the same hospital will be relayed to the CMHRS under the same CR number. If a patient moves to a different hospital, they should retain the same CR number because of cross-checks between hospitals when a patient declares a previous history. Some patients, however, inevitably obtain more than one CR number. Diagnostic information held on the CMHRS computer file seems fairly reliable, in our experience, for in-patients with diagnoses of psychotic illness, but is much less so for out-patients and for diagnoses of neuroses and personality disorders. We obtained the CR numbers for all those cases entered on the CMHRS whose diagnosis was, at some stage of their illness, 'schizophrenia', and whose occupation was recorded as 'nurse'.

TABLE I
Cases discarded

Nursing assistant	10
Veterinary nurse	1
Other occupation	4
Wrong diagnosis	4
Ill before nursing	2
Onset of illness after age 60 years	3
Total	24

This gave us 106 CR numbers and 96 individual 'cases'. With the consent of the individual hospital Medical Staff Committees we then traced the casenotes of all these 'cases' through each hospital's Medical Records Department. Some of the 'cases' had been referred to more than one hospital. The casenotes were examined in detail for diagnostic information (to accord with ICD-9 classification) and occupational history. We discarded 24 cases for the reasons noted in Table I. We retained only 'cases' who appeared to have been either SRN or RMN trained. Some of our so-called 'nurses' had been nursing assistants and we felt that these would not reliably be recorded as having an occupation as a 'nurse', so that quite possibly many other nursing assistants might have been diagnosed as having schizophrenia but not have appeared in our list of 'cases'.

Though the CMHRS was commenced only in 1962, all current in-patients at that time were entered. These patients, as well as many others entered on their next referral, of course, had ages of onset of illness before 1962. In order to relate the data to the respective numbers of nurses at risk, we obtained figures from the local Department of Health and Social Services for the total numbers of nurses employed in general hospitals and in psychiatric hospitals. These were available only from 1955.

TABLE II
Numbers employed, and numbers developing schizophrenia

Period	*General nurses*		*Psychiatric nurses*	
	Employed	*Falling ill*	*Employed*	*Falling ill*
1930–1939	—	2	—	0
1940–1949	—	1	—	1
1950–1954	—	3	—	1
1955–1959	3854	4	1312	0
1960–1964	5247	5	1841	1
1965–1969	7165	16	2346	4
1970–1974	8978	16	2453	6
1975–1979	13020	8	2900	4

Results

Results are shown for the final 72 'cases', of whom 52 had nursed exclusively in general hospitals and 20 had nursed in psychiatric hospitals. However, nine of the latter had apparently worked for periods in both. Of these, three had worked largely in general hospitals and appeared to have become ill before doing psychiatric nursing. These three cases' were therefore included with the 'general' nurses. The other six had worked primarily as psychiatric nurses, with only brief periods in general nursing. For the 'general' nurses (54 female, 1 male) the mean age of onset of illness was 29.8 years, and for the 'psychiatric' nurses (11 female, 6 male) it was 28.1 years. There was no statistically significant difference between these ages of onset.

Table II shows the average numbers of nurses employed in each category for quinquennia from 1955 to 1979, along with the numbers developing schizophrenia for the first time during the same periods, as well as in the period 1930–1954. These figures were then expressed as an *annual* rate per 1000 of the population of each group of nurses. Using the same formula as for comparing death rates, the two groups were compared for the period 1955–1979, but because the numbers were small the figures for the whole 25-year period were averaged. This gave an annual rate in general nurses of 0.256 per 1000 and in psychiatric nurses of 0.276 per 1000. These are not statistically significantly different. Clearly, no statistical comparison is possible for the period before 1955 as we do not have nurse employment statistics prior to this date.

Discussion

The idea of schizophrenia as a contagious, infectious disease would imply that those in close contact with schizophrenic patients would be more liable to develop the illness, and thus that psychiatric nurses should be at greater risk than general hospital nurses. The hypothesis was suggested from a synthesis of certain genetic and epidemiological data (Crow, 1983*a*), the interpretation of which was the subject of some debate (Murray & Reveley, 1983; Crow, 1983*b*). Our results clearly do not support it.

The data used can, of course, be criticised. It is possible that there might be under-reporting to the CMHRS of the occupation 'nurse' as there might be a feeling that this would prejudice a colleague in some way. However, if anything, the annual rate of new cases among both groups of nursing staff would appear to be higher than the 14 per 100 000 population per year often suggested (Gottesman & Shields, 1982). We may also have missed some of the cases of schizophrenia because they had not been diagnosed as such and therefore not recorded on the CMHRS. This error should only result in our numbers being smaller than expected (which does not seem to be the case) and in any case should affect both groups equally.

One advantage of studying nurses is that they will usually be working fairly consistently during the period of greatest risk for the onset of schizophrenia. A comparison of psychiatrists with other doctors would be of little value as they would often not begin to specialise until after a substantial part of the period of greatest risk had been lived through. What this approach does not take into account is the possibility that the schizophrenic patient might be 'infectious' before the onset of the illness, but this seems unlikely. We were unable to find any comparable data, as most previous research on psychiatric illness in health care workers has focused on anxiety, depression, stress and suicide (Denning, 1986). Study of psychiatric illness in doctors in general has not identified schizophrenia as a particular problem (Murray, 1983).

Thus, the idea of schizophrenia as an infectious disease does not really appear to have support. Indeed, in a re-analysis of the original argument, Crow (1984) also accepts this. The hypothesis also fails to explain the phenomenon of birth seasonality (Hare & Price, 1968). Furthermore, attempts to transmit an infective agent from brain tissue of schizophrenic patients to animals have so far been unsuccessful (Baker *et al*, 1983; Asher *et al*, 1984).

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Paranoid Psychoses after Abuse of Proprietary Cold Remedies

It is well established that psychosis may arise in the context of the abuse of certain prescribed or illicit drugs. Two cases of paranoid psychosis are described in which proprietary preparations containing amphetamine-like drugs were abused. The role of these drugs is discussed with reference to previous case reports, and it is emphasised that initial clinical assessment and urinary screening for drugs may fail to reveal the relevant history with respect to proprietary medicines.

Abuse of proprietary cold remedies may lead to psychosis. Two cases are reported in which psychosis occurred and in which the connection with drug ingestion was not immediately evident.

Case reports

Case 1

A 55-year old man was admitted to hospital with a 6-week history of withdrawal, abnormal behaviour and paranoid delusions involving a plot to kill him. He described delusional ideas of reference and misinterpretation of commonplace events (identifying passers-by as undercover "hit men"). He had attempted to blow himself up to escape his persecutors and had tried to buy a sawn-off shotgun to protect himself. When admitted to hospital, he demonstrated widespread and systematised delusions. His speech was difficult to follow and at times was incoherent, closely resembling the speech pattern characteristic of schizophrenic thought disorder. Although at admission there was some evidence of clouding of consciousness in that he gave clearly erroneous dates of birth for his family members, this was not immediately apparent or striking and when formally examined in the psychiatric unit 7 days after admission he was fully oriented, demonstrated no cognitive abnormality, but remained floridly deluded. At this time he also described an episode of visual hallucinosis which had occurred some weeks previously. Further questioning revealed he had for 2 years taken various proprietary medicines such as kaolin and morphine, analgesics, and Collis Brown's mixture to settle his nerves, but in recent months he had had only Contac 400 capsules, in doses of ten per day, and paracetamol. A drug screen for amphetamines, barbiturates and opiates was negative. He was treated with neuroleptic medication and recovered completely over a period of 2 months. Neuroleptic medication was discontinued after 8 months, and at follow-up over 20 months he remains drug-free and free of symptoms.

Case 2

A 48-year-old policeman was admitted to a general hospital with a 12-day history of headaches and cold symptoms. Two days later he experienced complex and frightening visual hallucinations. These were experienced as real events occurring on the ward. The following day he was experiencing auditory hallucinations (including his own thoughts recorded and played over the public address system) and developed paranoid delusions about the hospital and staff. He was transferred to the psychiatric unit at this hospital where he described a plot to discredit himself and the police force, and to overthrow the government. He was hallucinating voices in the second and third persons and believed himself to be under a hypnotic influence from the first hospital. Although alert, he was disoriented in time, giving the date as that on which he had been admitted originally to hospital 4 days previously. There were no other cognitive abnormalities. He was treated with chlorpromazine and his symptoms receded over the next 3 days. At this time he revealed that he had been taking Actifed linctus, Benylin Forte and phenylephrine nasal drops for some weeks before admission. His Actifed consumption approached one bottle a day. He had taken small doses of Librium and glutethamide for many years to calm himself, but had had no glutethamide for more than a month before his admission. A urinary drug screen for cannabis, amphetamines, barbiturates and opiates was negative. He recovered completely from his psychosis, into which he retained full insight, and was discharged 5 days after admission. He has subsequently been treated with tricyclic antidepressant for anxiety and depressive symptoms and remains well at 2-year follow-up.

Discussion

The main constituent of Contac 400 is phenylpropanolamine (50 mg) which is structurally similar to amphetamine. Contac 400 also contains small quantities of belladonna alkaloids (0.25 mg). This is less than one-tenth of the maximum equivalent recommended dose of hyoscyamine. Actifed linctus contains pseudoephedrine, structurally similar to methylamphetamine, and an antihistamine triponidine hydrochloride. Benylin contains diphenhydramine hydrochloride and dextromethorphan. There have been several reports linking cold remedies with psychosis.

Sankey *et al* (1984) described visual hallucinations occurring in three children receiving Actifed preparation in the recommended doses.

Leighton (1982) reported a case of paranoid psychosis occurring in a 27-year-old man with stable bipolar affective illness following abuse of Actifed. The psychosis occurred in clear consciousness and in the absence of mood change and lasted 12 days, settling the day after his admission. His symptoms had cast doubt on his diagnosis, and the history of Actifed was not revealed until follow-up 10 days after discharge.

Norvenius *et al* (1979) cited five cases of psychosis, three in children, following phenylpropanolamine ingestion. Two of the children were confused and the third experienced visual hallucinations and a *grand mal* fit. A 25-year-old woman experienced sleep disturbance, "vivid paranoid misconceptions" and muscular twitchings in the legs. The symptoms disappeared 24 hours after the drug was stopped. A 17-year-old demonstrated a "mania-like psychosis" with aggressive behaviour on three occasions before the use of phenylpropanolamine was revealed.

Cornelius *et al* (1984) described "paranoia, homicidal behaviour and seizures" after phenylpropanolamine abuse in a woman with borderline personality and a history of polysubstance abuse. Chaplin (1984) has noted reports of the use of phenylpropanolamine as a "street drug" or "pseudo-speed". Diaz *et al* (1979) described the chronic abuse of pseudoephedrine in a chronically depressed patient with habituation to the drug but no psychotic symptoms.

The history initially taken from patients may not reveal the fact or the extent of drug usage, and routine screening for drugs may be negative. The clinical presentation may show striking psychotic symptoms without obvious clouding of consciousness. Confusional elements suggesting a toxic cause may be absent or brief. In the first case described here, these features were minimal and cleared soon after admission, the psychotic symptoms persisting for many weeks. In the second case, the confusional elements were more apparent, but the psychotic features persisted for 7 days.

It may be that patients with borderline personalities or affective disorders are particularly at risk.

Patients presenting with paranoid psychoses should be questioned about proprietary medications as well as prescribed and illicit drugs.

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Encephalitis Lethargica, a Contemporary Cause of Catatonic Stupor
A Report of Two Cases

Two cases of catatonic stupor are described due to presumed encephalitis lethargica. Catatonic and obsessional phenomena in the setting of a depressive psychosis associated with a movement disorder may constitute a post-encephalitic syndrome.

Fifty years after the original description of encephalitis lethargica by Von Economo (1931), the cause remains enigmatic. Its natural history has the characteristics of an encephalitic disorder due to a neurotropic virus: recent epidemiological studies confirm Von Economo's original suspicion that it is related to influenza (Ravenholdt & Foege, 1982). The illness, however, was established as an entity by its neuropsychiatric sequelae, particularly Parkinsonism, oculogyric crises, movement disorders and a wide range of chronic psychiatric illnesses, especially psychopathic and psychotic states. Catatonic states occur in both the acute and post-encephalitic states of the illness and may develop quite independent of any psychotic symptoms (Davison & Bagley, 1969). The amyostatic–akinetic form of encephalitis lethargica was the third most common form of presentation of the illness in the 1918 epidemic, and as a result it came to be known as 'epidemic stupor'. Two recent cases of acute catatonic stupor are reported for which no adequate physical cause could be defined. That these cases were probably due to encephalitis lethargica is suggested by their resemblance to the post-encephalitic states of stupor described by Von Economo (1931).

Case reports

Case 1

A 23-year-old single male mechanic was admitted mute, akinetic, incontinent of urine and with severe blepharospasm. He adopted and maintained imposed postures for 10–15 minutes and would stand for hours in a bowed posture with flexed elbows and extended fingers. He had many compulsive rituals which involved sniffing and grunting and which occurred prior to eating food. He would communicate by answering 'yes' by moving his eyes horizontally or 'no' by a vertical movement. When he could be induced to stand and walk he performed a complex series of steps in the form of a square before entering doors.

His early development and parental relationships were normal, and in adolescence he had been a jovial and well adjusted youth. A depressive illness began three years previously and was thought by his parents to coincide with the suicide of a friend and the breakdown of a relationship with a girlfriend. He developed sleep inversion and according to his mother "turned night into day". His face became swollen in the region of the parotid glands and he lost weight. He was referred to Newcastle General Hospital where a full neurological investigation including CAT scan, LP and EEG were all normal. He lapsed into a catatonic stupor and was treated by abreaction, sodium pentothal interviews, and finally ECT, which produced some improvement.

The following year he again became depressed and would spend many hours in the bathroom counting and taking his clothes off in a precise sequence. This would involve sniffing and grunting and he would become angry if interrupted. He would 'freeze' during some movements such as eating or standing. He had two further hospital admissions but no firm diagnosis was ever arrived at. He was given further courses of ECT, behaviour therapy and lithium, and eventually improved.

He was then referred to Withington. All investigations were repeated and found to be normal. There were again no features of schizophrenia: he was severely dehydrated, weighed 84 lb and was hypotensive (BP 90/60). He was given intravenous fluid and ECT to a total of 14 treatments. He improved, and began to swallow spontaneously and move around his bed. At this point the possibility was considered that his catatonic stupor was due to encephalitis lethargica, and a trial of l-dopa with benserazide (125 mg t.d.s.) was given. Further improvement occurred and he began to speak spontaneously after 12 weeks of mutism. He remained, however, inert, lacking in spontaneity, and it was decided to give him flupenthixol 40 mg fortnightly intramuscularly. At this stage he was able to dress, walk and converse freely and over the next two weeks his rituals subsided completely. He was finally discharged home after three months. His mood was still morose, but he did interact with other patients, and he weighed 112 lbs. When seen at follow-up six months later his progress had been maintained and he was living a reasonably normal life at home.

Case 2

A 17-year-old male had a three-weeks history of an acute depressive illness in which he developed secondary delusions that he had contacted typhoid and was infecting his family. He took a small overdose of an antihistaminic (Piriton) and was admitted to hospital. On transfer to the psychiatric unit three days later he was mute and akinetic, and had marked negativism, hypertonia of his limbs and flexibilitas cerea. When he could be induced to move he would 'freeze' in fixed postures. He was incontinent of urine and had a low grade pyrexia. Physical examination was normal apart from hypotension (BP 90/70), attributed to dehydration. An EEG was the only abnormal investigation and showed diffuse, asynchronous delta and theta activity compatible with an encephalopathy. After rehydration with intravenous fluids and a course of ECT × 12 he improved and his catatonic phenomena disappeared. He remained amnesic for the period of his illness. His EEG prior to discharge had improved despite the course of ECT. He was seen again two years later when a report was requested for him while he was on remand in prison. Apart from poverty of expression and a lack of sponteneity, there were no other psychiatric or neurological symptoms.

Discussion

Catatonic phenomena which occur during the course of a depressive illness are uncommon, but they were a prominent feature in both these cases and remitted with ECT. Kraepelin included catatonic states within his concept of dementia praecox and considered they had a poor prognosis. This view was subsequently challenged and some forms of catatonic stupor were thought to have a good prognosis and to be more closely related to manic-depressive disorders than to dementia praecox (Hoch, 1921). Both these cases exhibited severe catatonic phenomena in the setting of depressive psychosis and resembled the cases described as 'Benign Stupor' by Hoch. He raised the possibility that his cases may have been due to encephalitis lethargica, which had just been described.

The triad of catatonic, obsessional and depressive phenomena, as in case 1, occurring in the absence of schizophrenic symptoms, is suggestive of encephalitis lethargica, while the movement disorder, particularly blepharospasm with a history of sleep inversion, reinforces this possibility. Lewis (1957), in his review of obsessional states, acknowledges the relationship between encephalitis lethargica and obsessive-compulsive behaviour.

Case 2, by contrast, presented as an acute depressive psychosis with catatonia and a recurrent pyrexia. No other schizophrenic phenomena were ever present and no alternative organic aetiology was ever advanced. This acute catatonic state resolved with ECT and closely resembled the amyostatic-akinetic syndrome that occurred in over a third of acute cases of encephalitis lethargica described by Von Economo during the 1918 epidemic.

A wealth of clinical evidence has accrued over the past 50 years to confirm Von Economo's original belief that encephalitis lethargica is a pathological and clinical entity; it still occurs in sporadic form today and is the antecedent of much chronic psychiatric illness (Hunter, 1973). It is now generally accepted that catatonic states are symptomatic of a neurological illness, and the possibility of encephalitis lethargica should be considered in the differential diagnosis of cases of catatonic stupor.

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A Case of Affective Psychosis following Bereavement in a Mentally Handicapped Woman

Mentally handicapped people are increasingly being subjected to the stresses of everyday life, and bereavement represents a particularly potent stress. This report considers the case of a mentally handicapped woman whose difficulty in mourning and expressing and sharing grief had a profound effect on her recovery from bereavement and led to her developing an affective psychosis.

Bereavement represents a highly significant life event which may precipitate major depressive or other psychotic illness; our knowledge of bereavement in mentally handicapped people and how they cope with death, however, is limited (Oswin, 1981). Increasing numbers of mentally handicapped people are now living to middle age (Tait, 1983); they are often cared for in the community by ageing relatives or carers and consequently more handicapped people are experiencing bereavement than was formally the case. Parkes (1964) gives evidence to suggest that a correlation exists between bereavement and mental illness in people of normal intelligence, and it is likely that the same is true for mentally handicapped people.

Case report

A 47-year-old moderately mentally handicapped woman (IQ Stanford Binet 50) was admitted to the acute treatment ward of a mental handicap hospital after she had become increasingly unmanageable at her hostel and training centre.

There is no known cause for her mental handicap and there is no known past history of mental illness in her family. She has had no previous psychiatric illness, although she has been described as having an anxious personality; she has a long history of chewing her nails. The patient has good self-care skills, being able to feed, dress and toilet herself; she is unable to read or write but enjoys simple craft activities; she is capable of helping with simple domestic tasks.

The patient lived with her parents uneventfully until four years ago, when her mother suddenly died. She was described as having a very close relationship with her mother. Following her mother's death the patient was noted to have become disturbed and overactive. She was found wandering about in the local town at all hours of the day and night in search of her mother; because of this she earned herself the nickname "The Wanderer". Shortly after her mother's death she went to live in a local authority hostel, as her elderly father could no longer manage to look after her at home. Although she attended her mother's funeral, she did not talk about her mother's death. Her wandering behaviour continued for a few years but eventually stopped. She remained in regular contact with her father.

A year ago the patient's seventy-six-year-old father was diagnosed as suffering from carcinoma of the prostate gland; the patient was informed of this and appeared to understand that the condition would limit his life. Shortly after she was told this, the wandering started again. She became very restless and agitated and was difficult to manage in the hostel and the adult training centre. About this time she was referred to the psychiatric services and it was felt that she was undergoing a symbolic anticipatory mourning reaction. No clear evidence of clinical depression could be found at this time.

Several months later her father died; she attended his funeral but again she became extremely agitated, wandering away from the hostel and pestering the neighbours in the vicinity of her old home. She was sleeping very little, was overactive and had little interest in food; she also admitted to hearing her father talking to her during the night. She was started on thioridazine 25 mg t.d.s., 100 mg at night; her condition quickly improved.

After two months, however, she started to become increasingly withdrawn; she lost interest in food and her weight decreased by one stone. She had no spontaneous conversation and avoided contact with other hostel residents and staff. Her sleep was disturbed, she was weepy and very restless. Her self-care had deteriorated markedly and she began to smear faeces. She was diagnosed as suffering from severe depression; at the time of admission her score on the Hamilton 17-Item Rating Scale for depression was 29, which supported the diagnosis of severe depression. Antidepressant medication was gradually introduced to a final dose of trimipramine (150 mg at night), and over the next six weeks her condition began to improve. She started to attend occupational therapy, her sleep and appetite improved and she was now able to talk about the death of her parents, which was being encouraged. Her current score on the Hamilton Rating Scale is 8.

It became apparent from discussions with the nursing staff that her relationship with her father just before his death had become very disturbed. Previously they had had a close relationship, but following his illness the father became unable to cope with his daughter's constant pestering, and on several occasions he had beaten her off with his walking stick in his attempt to make her leave him alone. Her feelings towards him were clearly ambivalent and this may have made it more difficult for her to come to terms with his death.

Discussion

Reactions following bereavement have been well described (Lindemann, 1944). Feelings of shock, denial and alarm are common initially, followed by a mourning phase in which searching for the dead person may occur. Transient hallucinatory episodes are common in the normal bereavement process, as are symptoms of depression. After about 6–8 weeks, however, these symptoms usually fade, and in successful mourning, acceptance of the loss usually occurs after 6–18 months. Parkes (1964) describes a typical mourning in which there may be a serious prolonged impairment of function or a precipitation of serious psychotic illness.

Following the death of her mother, the patient displayed wandering or searching which could be seen as being part of a normal grieving process, but its prolongation was abnormal. At this time she not only suffered the death of her mother, but was also moved out of the familiar surroundings of the family home, she lost the daily contact with her father and also had to begin making relationships with a new group of people previously unknown to her. These changes, which occurred suddenly and for which she was completely unprepared, would have constituted an overwhelming degree of distress, which she was probably never able to come to terms with satisfactorily.

The mentally handicapped have a limited ability to conceptualise (Bihm & Elliott, 1982); this may have prevented the patient from coming to terms with grief after her mother's death, which was probably still unresolved at the time of her father's illness. The anticipated loss of her father several years later caused a recurrence of her initial symptoms, but with greater severity. After her father's death she became acutely agitated; she was treated with thirodazine, but she later went on to develop symptoms of a severe clinical depression.

The diagnosis of psychiatric illness in the mentally handicapped in notoriously difficult and some of the typical symptoms may not be displayed or expressed (Reid, 1972). Some writers have queried in the past the capacity for the mentally handicapped to become depressed (Herskovitz, 1941). It would be unreasonable to suppose that mentally handicapped people do not develop affective psychosis in the same way as people of normal intelligence, and stress may be an important precipitant.

Penrose (1972) indicates that the mentally handicapped react more severely to changes in the environment than do people of normal intelligence. Day (1985) believes that mentally handicapped people living in the community are living a more stressful life than people who are not mentally handicapped, and that death of a carer is a particularly potent stress.

Mentally handicapped people are often shielded from death and they may be prevented from attending funerals. Their lives may be profoundly altered following the death of a care-giver (Bicknell, 1983); this, coupled with their reduced intellectual capacity to come to terms with the loss, may result in a considerable degree of distress which may be unnoticed. It seems likely, therefore, that the mentally handicapped are going to be a group subject to significant morbidity following bereavement. It is hoped that research will be done to assess the full extent of the problem and to explore methods which can be employed to minimise distress (McLoughlin, 1986).

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Short-Cycle Manic–Depressive Psychosis in a Mentally Handicapped Child without Family History

A Case Report

Diagnosis of classic psychiatric illness in mentally handicapped individuals remains difficult. Manic–depressive illness has previously been reported in both pre-pubertal and pubertal children with a mental handicap and with a family history. This paper reports a case of manic–depressive psychosis in childhood, with no family history, short-cycle mood swings and good response to lithium therapy.

John is now 15 years old. He was referred three years ago for psychiatric assessment, with a five-year history of mood swings and associated behavioural disturbances.

History

John is the elder of two boys. He was a full term normal delivery, to a healthy female in her mid-20s, and weighed 8 lb 14 oz. Early development appeared normal except for a persistent tendency to vomit small amounts. At the age of 18 months his development slowed down, speech ceased to progress and his parents sought the advice of a paediatrician. Between the ages of two and three his behaviour became clearly overactive; he suffered several minor head injuries at this age, but with no loss of consciousness or obvious repercussions. He has had no serious physical illnesses. John's early education was in a specialist unit for speech-retarded children. At the age of seven he underwent extensive psychological assessment which revealed that he was functioning cognitively at the 5 year level on the Merrill–Palmer Scale (Stutsman, 1931), but that language skills were at 3–3½ year stages on the Reynell Department Language Scale (Reynell, 1969). He was diagnosed as having a developmental language disorder.

At the age of nine he was accepted for education at a specialist school for autistic children, where he is a weekly boarder. While John does exhibit various autistic traits, including obsessional behaviour and reversal of pronouns, throughout his childhood he has been described as affectionate. The primary diagnosis is one of moderate mental retardation of unknown cause. He has made little academic progress in the last four years, but his self-care skills have improved. Further recent testing on the British Ability Scales has confirmed his intellectual limitations.

His mood swings are substantiated by school records: associated periods of pallor have been investigated by paediatricians, without any significant abnormal findings.

Prior to treatment, behaviour was monitored for 18 months, and a clear cyclical pattern to the mood swings emerged, substantiated by daily school and parental reports.

When well, John presents as a clean, sociable boy, responsive to and seeking affection. This phase is followed by a gradual period of withdrawal. John looks pale and ill, he is reluctant to eat or speak. He wakes early in the morning; he soils and wets himself. He sometimes cries for his "mummy", he headbangs and he has been heard to say "I can't stand it any more". He gradually returns to normal. This is followed by a phase when John becomes increasingly restless and distractible. His mood is incongruously jolly; he is overcome with waves of noisy hilarity. His speech becomes more rapid, its content fantastic and there is flight of ideas. His behaviour becomes "uncontrollable", with bouts of unexplainable aggression: he breaks things, throws his clothes out of the window and smears faeces. He gradually settles and returns to normal. Each phase lasts approximately 14 days.

In spite of extensive questioning, no family history of psychiatric illness has been elicited. A maternal great uncle had a developmental speech disorder, but was of normal intelligence.

Treatment

John was commenced on lithium therapy. Therapeutic blood levels were achieved. His behaviour continued to be monitored via daily reports from school and home: there was marked attenuation of mood swings, with an associated marked improvement in school progress.

After 12 months of treatment, the parents requested discontinuation of lithium therapy, believing much of his improvement to be attributable to "growing up". Within two weeks of cessation of lithium, John's behaviour deteriorated dramatically into a restless phase. Therapy was recommenced and he gradually settled.

Discussion

The reported case raises several interesting points. Manic-depressive illness in childhood is still a relative rarity, and its occurrence in a mentally handicapped child is even rarer and more likely to be missed (Hucker, 1975). Hucker's case had a strong family history, as well as evidence of precipitating events, neither of which are found in this case. Short-cycle manic-depressive illness has previously been reported in mentally handicapped adults (Reid & Naylor, 1976), but not children.

Diagnosis of manic-depressive psychosis in the mentally handicapped is fraught with difficulty, behaviour due to illness often being attributed to the handicap, or even misdiagnosed.

Rivinus & Harmatz (1979) have commented on the eminent treatability of manic-depressive psychoses. They reported a 100% success rate treating mentally handicapped adults with the disorder with lithium. Treatment of all childhood psychiatric disorders tends to be more conservative than in adulthood.

ECT has been used in childhood bipolar illness, and Hassanyeh & Davison (1980) report on the use of ECT and the use of lithium in ten manic-depressive, intellectually normal children.

Accurate diagnosis and effective treatment are never more vital than in childhood. The mentally handicapped already have their intellectual impairment as handicap; therefore identification and treatment of psychiatric illness are essential to enable them to achieve their maximum potential.

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Neurosyphilis – a Treatable Psychosis

A patient presenting with mixed affective symptoms was found to have active neurosyphilis after intermittent cognitive impairment suggested an organic cause. Most of these symptoms resolved after treatment with penicillin.

Neurosyphilis has long been associated with psychiatric syndromes, but it now rarely presents to the psychiatrist at an acute, treatable stage. With the introduction of penicillin the number of cases has diminished and in many psychiatric centres serological tests for syphilis are not performed routinely. The widespread use of antimicrobials may have also led to the masking of the early stages of the disease because of coincidental therapy for other conditions. We report here a case of neurosyphilis presenting as an organic psychosis, which resolved with penicillin.

Case report

A 43-year-old, unemployed, single, homosexual man was admitted to a psychiatric hospital (Mapperley Hospital) informally from police custody following several incidents of bizarre and destructive behaviour during the preceding fortnight. Mental state examination revealed a mixed affective state with intermittent clouding of consciousness and loss of memory. Initial biochemical and haematological profiles were normal, but because of his intermittent cognitive impairment, additional laboratory investigations were performed. Syphilis serology showed the following titres: VDRL 64, TPHA 128 and FTA (Abs) positive. He was Hepatitis B surface antigen negative, and HIV III antibody negative, and chest X-ray was normal. Lumbar puncture was carried out and this yielded results characteristic of active neurosyphilis, the cerebrospinal fluid (CSF) being Igm FTA positive and having a lymphocyte count of 14/μl. Psychological assessment demonstrated an acquired cognitive deficit and NMR scanning revealed no structural abnormality.

He was treated with penicillin and his mental state improved rapidly; he was discharged from hospital on no drug therapy. Five months later he became paranoid and suspicious, necessitating admission to a day hospital. No cells were seen on repeat examination of the CSF so he did not receive a second course of penicillin.

One year after his relapse he had remained free of paranoid symptoms with a mild degree of cognitive impairment.

Discussion

Recent surveys (Nordenbo & Soresen, 1981; Alani & Millac, 1982) suggest the incidence of neurosyphilis to be two per million population per year. Of these, approximately 25% present with a psychiatric disorder. The remainder present with cerebrovascular accidents, visual problems, learning deficits and on incidental testing.

Abnormalities of the CSF may occur at any stage of the disease and specific serological tests on CSF are positive in every untreated active case. They may or may not revert following treatment. Residual neurological damage following treated neurosyphilis is a frequent finding. Although there is evidence that unnecessary laboratory investigations are often carried out in psychiatric patients (Ferguson & Dudleston, 1987), in those patients whose mental state suggests an organic component, serological screening tests for syphilis are still essential. This case serves as a good illustration.

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Contents: A search for sub-clinical arteriosclerotic dementia/Review of *Behavioural Psychotherapy*/ Range of mental illness among the elderly in the community/Mania following head injury/Schizophreniform episode following measles infection/Electrodermal response as a monitor in ECT/Neuroleptic malignant syndrome or lithium neurotoxicity?/ Tuberous sclerosis and the autistic syndrome/Delusional parasitosis/Lucid intervals in catatonia: a neuropsychiatric snare for the unwary/Sub-cortical dementia and the EEG/'Barking mad'/Ofloxacin-induced psychosis/Oxymethalone and aggression

A Search for Sub-Clinical Arteriosclerotic Dementia

SIR: McDonald & Sudhaker (*Journal*, July 1987, **151**, 134–135) state that "the high discordance between the pathological findings and the ante-mortem diagnosis of arteriosclerotic dementia is well known". Such discordance was not evident in my own cases from an American mental hospital (Birkett, 1972) in the days before CT scans. The autopsy findings were predicted with considerable accuracy.

Proving or disproving the existence of arteriosclerotic dementia without infarcts is difficult. The closest we have so far come is probably in Corsellis's (1962) study. He found cerebral arteriosclerosis without infarcts to be significantly commoner in demented patients than in matched controls from the same hospital with functional psychosis.

Until such studies can be replicated it is probably best to follow Alzheimer (1894) and call the condition mental disorder based on arteriosclerosis ("Seelenstoerungen auf arteriosclerotischer Grundlage"), specifying the presence of dementia and the presence of proven infarcts.

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Review of *Behavioural Psychotherapy*

SIR: Mackay (*Journal*, July 1987, **151**, 136) implies that the behavioural interventions described in this book are applicable only to "simple phobias and obsessional disorders". He seems to have missed most of the book's content. Of its eleven detailed case histories with clinical management protocols, only two concern simple problems. The remaining nine case illustrations are of complex disabling difficulties, including severely handicapping agoraphobia, social skills deficits, sexual dysfunction, sexual deviation, eating disorder, irritable bowel syndrome, and stammering. Many of the patients whose behavioural treatments are detailed are among the most complex and difficult to be found in psychiatric practice. Mackay's suggestion that adding a cognitive approach enhances behavioural treatment flies in the face of most controlled evidence in the field of anxiety disorders (reviewed by Marks (1987), chapter 14).

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Range of Mental Illness Among the Elderly in the Community

SIR: I am perplexed as to why Copeland *et al* (*Journal*, June 1987, **150**, 815–823) consider their elderly mental ill as divided into Caucasian, Mongoloid and Negroid "races". These are now abandoned terms in biological anthropology, and have no obvious relevance, either, to current (socio-political) use of the term 'race'. What did the interviewers mean by describing an individual as 'Negroid'? Most

Afro-Caribbeans have, for instance, White ancestry. Using the Victorian definitions of 'Caucasian' (predicated upon 'Indo-European' linguistic patterns), South Asian immigrants and their children born in Britain are 'Caucasian'. Or do the authors mean 'White' by 'Caucasian'?

The ambiguous and tendentious use of an idiom which connotes a simple causal relationship between phenotype, genotype, behaviour, and social experience is disturbing. What is wrong with the terms (a) *ethnic group* (as currently perceived), (b) *race* as a sociological term, or (c) quantitative measures of population genetics, as variously needed?

Estimates of various ethnic groups in the general population *can* be derived from the 1981 Census head of household figures using the Labour Force Survey correction factors.

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SIR: We thank Littlewood for giving us an update on the latest anthropological view for describing the demography of samples. To clarify the use of terms in our study, 'Negroid' refers to those individuals who have the appearance of some African ancestry, and 'Caucasian' refers to Indo-Europeans, white and non-white.

Clearly, associations in themselves cannot be used to imply a causal relationship, simple or otherwise.

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Mania Following Head Injury

SIR: Clark & Davison (*Journal,* June 1987, **150,** 841–844) argue that the infarct their second patient was noted to have in the left frontal region during a manic episode was unlikely to be causative because Cummings & Mendez, in their review on the association between mania and the site and nature of intercerebral lesions, did not find any reports of mania associated with cerebrovascular lesions in the left frontal region. There are at least two case reports of mania associated with lesions in the left frontotemporal region: Jampala & Abrams (1983) reported a 52-year-old man with recurrent manic episodes with onset at the age of 24 following a rupture of a left middle cerebral artery aneurysm. Herlihy & Herlihy (1979) have described a 58-year-old lady with manic episodes following a haemorrhage in the left middle cerebral artery. Both these patients were noted to have lesions in the left frontal and frontotemporal region.

Clark & Davison also note that the two previously-reported cases of mania associated with head injury occurred in younger patients and that this is contrary to the commonly held view that secondary mania is more common in the elderly, as suggested by Krauthammer & Klermann (1978). Shukla *et al* (1987) studied 20 patients who developed mania following closed head injury, and the age at first psychiatric episode in their patients ranged from 17 to 42 (mean = 27.5 years). Brackens (1987) reported a lady who developed mania at the age of 48 following head trauma. So, from the above reports, it appears that mania can occur at any age following head injury.

Carbamazepine is very effective in the treatment of secondary mania (as suggested by Jampala & Abrams (1983)) and this should be considered as one of the first line agents in addition to lithium.

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Schizophreniform Episode Following Measles Infection

SIR: The description by Stoler *et al* (*Journal,* June 1987, **150,** 861–862) may be the first adult case reported in the literature, but Nunn *et al* (1986) have previously reported four cases where there was an association between a viral illness (measles, rubella, varicella, and herpes) and adult-type psychosis in children. All the children had some neurodevelopmental disorder rendering the children more vulnerable to psychosis once infected by a virus.

Nunn *et al* drew attention to the need to clarify the role of the virus in the aetiology and suggested a hierarchy of questions exploring the strength of relationship between viral presence and psychosis: (a) does the patient have a systemic viral illness? (b) is the virus present in the cerebrospinal fluid (CSF) or is there evidence of an immunological response even if the virus is not present? (c) is the virus actually infecting the central nervous system (CNS)? and (d) is the viral infection of the CNS responsible for the observed psychosis?

Stoler *et al* would not appear to have sufficient evidence to answer the second question as, in the case they describe, the presence of monocytes in the CSF only indicates involvement on the meninges and is not adequate proof of brain tissue involvement. This highlights the need for rigorous use of more sophisticated techniques to demonstrate CNS involvement by viral agents (e.g. virus isolation, CSF banding) before moving from temporal associations to causal relationships.

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Electrodermal Response as a Monitor in ECT

SIR: Simpson & Hyde (*Journal*, April 1987, **150**, 549–551) give a description of the "cuff" technique (Adderley & Hamilton, 1953) for monitoring ECT.

I regret to have to point out that the description is incorrect. Before giving ECT, a sphygmomanometer cuff is applied to one arm and the pressure raised to above that of the systolic blood pressure. The suxamethonium is then injected through another vein, and after all the muscular twitching has stopped the sphygmomanometic cuff is released and the electric shock administered.

One should be wary of forcing a muscle to contract vigorously when its blood supply has been cut off.

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SIR: We thank Hamilton for clarifying details of his technique. It does not invalidate the result of our brief study, as the convulsion was observable as by the original method. Total systolic occlusion time was very brief and no harmful sequelae have been observed.

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Neuroleptic Malignant Syndrome or Lithium Neurotoxicity?

SIR: Jee (*Journal*, April 1987, **150**, 568–569) argues that Abbott & Loizou (*Journal*, January 1986, **148**, 47–51) in their review of the neuroleptic malignant syndrome (NMS) appropriately excluded the data from Cohen & Cohen (1974) as an example of this syndrome on the basis that their evidence was inconclusive.

However, Abbott & Loizou had cited the observations of Baastrup *et al* (1976), whose study "was carried out in order to determine whether the syndrome described by Cohen & Cohen is, in fact, seen frequently in patients given both lithium and haloperidol", without reference to Cohen & Cohen's original findings. We were concerned by this omission and our own correspondence (*Journal*, September 1986, **149**, 385) simply drew attention to the descriptive superficial resemblance of the Cohen & Cohen cases to NMS.

We think that the evidence for lithium neurotoxicity in these cases as proposed by Jee is no stronger than our own postulations. He quotes Schou (1984) who reviewed case reports on 40 patients with persistent neurological sequelae after lithium intoxication, but fails to mention Schou's special comments on the Cohen & Cohen cases whom he regarded as atypical. Schou noted that none of them had particularly high serum lithium concentrations compared with the group as a whole; also, there was a high fever of unknown origin in all four cases, whereas in the rest of the group fever, where it was documented, was identified with a somatic illness in all but one case. Finally, on follow-up 2–10 months later none of these patients had the clear-cut cerebellar syndrome characteristically attributed to lithium toxicity.

More recently, Goldney & Spence (1986) in a retrospective study of 60 manic patients treated with neuroleptic drugs alone and 69 manic patients treated with neuroleptic drugs and lithium could demonstrate no significant differences in side-effects between the two groups, including comparisons made between patients on haloperidol only and those treated with haloperidol and lithium. Their comments thus add further weight to the original observations of Baastrup *et al.*

Doubts regarding the true cause of the clinical events in Cohen & Cohen's patients will never be completely resolved, although the validity of their own postulate of a lithium/haloperidol interaction seems to be diminishing as the full explanation.

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Tuberous Sclerosis and the Autistic Syndrome

Sir: Lawlor & Maurer (*Journal*, March 1987, **150**, 396–397) described tuberous sclerosis presenting with autistic behaviour. Reported cases of this association are rare; however, I believe that it is not that uncommon. I can recall seeing three such cases. The following is an example.

Case Report: A male was born following a delayed labour and assisted delivery but recovered normally. Within 5 days he was found to be a restless baby with much screaming and difficulty in feeding. He walked at 12 months, but at 15 months he started to have temper tantrums. Subsequently, it became apparent that he had delayed speech. Later he developed overactive and disruptive behaviour, which eventually lead to his admission to a child psychiatry unit at the age of 3 years. He was diagnosed as having a profound language disorder with severe autistic traits, but was of potentially normal intelligence. Physical examination at this time showed no neurological abnormalities, but roughened skin on the cheeks and between the shoulder blades was noticed. Over the next few years there were many reports of poor social interaction, delayed speech with reversal of pronouns, mannerisms, inappropriate emotional reactions, resistance to change, and unusually good reading ability. At the age of 6 he developed epilepsy, which remained well controlled with anticonvulsants. At various times during his childhood he attended an ESN (M) day school, several private residential schools, and a school for autistic children.

At the age of 9 he was referred to me by the social worker because of difficult and aggressive behaviour. Physical examination revealed clear facial adenoma sebaceum, several areas of depigmented skin on the trunk and legs, and the previously recorded roughened areas between the shoulder blades. The family was advised of the possible diagnosis of tuberous sclerosis. Both parents were very intelligent and they had a normal daughter. The only significant feature in the family history was that the mother had had a mentally handicapped half brother who died at the age of 22 years with a tumour of the liver. Further physical examination of the family by a consultant geneticist revealed that the mother had a slight skin lesion in keeping with the diagnosis. The father and the sister appeared to be unaffected. The parents were advised of the significance of the findings, but had already decided to have no more children.

The patient has continued to present problems of management, especially aggressive behaviour, and still has many autistic features. He attends an Adult Training Centre and lives in a Social Services hostel.

The above illustrates not only the association of the two conditions but also supports the authors' conclusion that a suspicion of tuberous sclerosis should be aroused if autistic features and epilepsy coexist. It also suggests that history taking and physical examination in child psychiatry should always involve a genuine appreciation of genetic factors.

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Delusional Parasitosis

Sir: Macaskill (*Journal*, February 1987, **150**, 261–263) reports the first case of a non-pharmacologically induced remission of delusional parasitosis. Hunt & Blacker (*Journal*, May 1987, **150**, 713–714) suggest that this was a mild form of the disorder, associated with clear precipitants. I report two further cases of spontaneous remission.

Case reports: (i) A 51-year-old housewife with no previous history of medical or psychiatric illness presented to a dermatology clinic with a one-month history of parasites in her hair. She described hearing them moving, and could feel them burying into her scalp. She produced a matchbox containing skin scrapings as proof. There were no obvious precipitants, although she was under chronic strain through coping with her demented mother. The dermatologist found no abnormality, and prescribed no treatment. However, he concurred with her suggestion of a brief holiday in Spain. On her return two weeks later she was seen by the psychiatrist. She said the parasites had now gone, but was certain they had been present. There was no evidence of psychiatric disorder (CIS score = 7; HAD score = 4). She remained well one month later.

(ii) A 58-year-old shop assistant attended the dermatology clinic with a three-month history of delusional parasitosis. She claimed she had been infected by her sexual partner, and that she could now feel insects crawling under her skin, stinging her, and living in her anus. She also felt worms moving in her stomach. She felt dirty, and thought her body odour was offensive. She had attended her GP, a private dermatologist, and several alternative practitioners, but without benefit. She had had a genuine hook worm infestation as a teenager. She had also received antidepressants from her GP when she was 32 and 42, during times of marital stress.

When seen by the psychiatrist she had been ill for four months. Her beliefs were delusional in nature, and she recorded maximal conviction on visual analogue scales of intensity of belief. Other than fatigue there was little to suggest depression (CIS score = 21). She refused treatment. One month later she reported that she had passed a "worm" in her stools, and described a three inch long creature with horns. Since then the rest of the parasites had died, and she no longer felt infested. She still felt she had a dirty tongue and skin, and still felt self-conscious of her body odour (CIS score = 12).

Previous reports in the psychiatric literature have emphasised the intractable nature of untreated delusional parasitosis (Monro, 1980; Sheppard *et al*, 1986), unless as part of a depressive illness. In a questionnaire survey of dermatologists, Lyell (1983) identified 282 cases, and confirmed the generally poor prognosis, but claimed 13 had remitted. He also described one case history of a spontaneous remission after three months. Batchelor & Reilly (1986) used a similar method to obtain details of 55 patients. Although again confirming the poor prognosis, "a third had a duration less than a year" while one had "improved slowly on dermatological treatment only". The gloomy natural history reported by psychiatrists may partly reflect patterns of referral.

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Lucid Intervals in Catatonia: A Neuropsychiatric Snare for the Unwary

SIR:

Case report: A 42-year-old woman with a recent history of treatment of a major depressive disorder with dothiepin (150 mg/day) suddenly became agitated, complaining of being unable to walk and that her mind was going blank. She was admitted in a state of retarded depression, but despite an increase in dothiepin (225 mg/day) continued to deteriorate. Her mental state fluctuated between mute immobility and terror-stricken agitation, for which she was given chlorpromazine (100 mg i.m.) on three occasions over a one-week period. She resisted all nursing care and had to be fed and toileted, at times exhibiting verbigeration, bizarre and stereotypical behaviour, and catalepsy. Diazepam (20 mg i.m.) prior to CT scanning produced a dramatic improvement, lasting several hours. She spoke coherently and started eating, before relapsing. Investigations, including CT scan and EEG, were normal. Full recovery followed after three bilateral ECTs, and she remained well at six-month follow-up.

Amylobarbitone sodium interview continues to be advocated as a diagnostic aid in distinguishing catatonia secondary to idiopathic psychiatric ('functional') disorders from those that are toxic-metabolic or neurological ('organic') in origin (Altshuler *et al*, 1986). Our patient's response would be a case in point were it not for the fact that the catatonic signs became apparent only after the introduction of neuroleptic treatment. Ainsworth (*Journal*, January 1987, **150**, 110–112) and Chick *et al* (*Journal*, July 1987, **151**, 130–131) describe similar results with benzodiazepine and barbiturate infusions in catatonia, due to viral encephalitis and structural brain damage respectively. Further doubt is thrown on the diagnostic value of these techniques by reports of temporary or permanent relief obtained in a case associated with pituitary adenoma (Sheline & Miller, 1986), neuroleptic-induced catatonia (Fricchione *et al*, 1983) and the neuroleptic malignant syndrome (NMS) (Lew & Tollefson, 1983), which clinically resembles an iatrogenic variety of Stauder's "lethal catatonia". In addition, Lim *et al* (1986) have reported dramatic responses to i.v. phenytoin in catatonia as a manifestation of non-convulsive status epilepticus.

Several lines of evidence suggest that catatonia is the product of disturbed central neuroregulatory-vegetative functioning. The similarity in symptomatology between the neuroleptic-induced and other varieties of catatonia may reflect a common pathophysiology, involving dopamine and GABA neurons in the mesostriatal and mesolimbic systems and hypothalamus (Fricchione, 1985). Dopamine agonists, e.g. dantrolene and bromocriptine, appear to relieve NMS by direct alteration of dopaminergic transmission, while barbiturates, hydantoins, and benzodiazepines, which interact with receptors closely related to the GABA/chloride-ionophore complex, do so indirectly, mediated by GABA feedback loops in the mesostriatal and mesolimbic systems. The relatively weak GABA-ergic properties of barbiturates may also account for the shorter duration of lucid intervals following amylobarbitone sodium infusion compared with those produced by benzodiazepines.

In view of the wide variety of conditions associated with catatonic states it seems doubtful whether a response to barbiturate or benzodiazepine infusion has any *diagnostic* validity, although the latter may be of therapeutic benefit, depending on the extent and localisation of underlying cerebral pathology. A safeguard against the pitfalls of the traditional 'functional/organic' dichotomy would therefore be to conceptualise catatonia as a non-specific neuropsychiatric syndrome, a final common pathway of response to an overwhelming psychiatric, neurological, or medical insult.

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Sub-Cortical Dementia and the EEG

SIR: The review of the concept of sub-cortical dementia by Cummings (*Journal*, December 1987, **149**, 682–697) was timely, comprehensive and persuasive. A further body of evidence that can be cited in favour of the nosological distinction between cortical and sub-cortical dementia comes from the electro-encephalogram (Fenton, 1974). The EEG in the cortical dementias of the Alzheimer-senile type is invariably abnormal, characterised by diffuse asynchronous delta and theta dominant records. By contrast, the EEG of the sub-cortical dementias of Huntington's chorea, Parkinson's disease, post-traumatic encephalopathy, and post-encephalitic states is either normal or 'flat' low voltage in type. The vascular encephalopathies occupy a variable and often intermediate position between the two.

These differing EEG patterns in the cortical and the sub-cortical dementias must reflect the progressive disintegration of different neural systems, and is further evidence in support of employing this nosological classification of the dementias. We are at present attempting to carry out a double-blind assessment of the EEG in these two categories of dementia.

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'Barking Mad'

SIR: "She had something that girl. She's mad, that's the worst of it. Bonkers, barking, round the bend". 'Barking mad' is a term in colloquial use which has started to appear in English literature as the line above, from John Welcome's 1968 play *Hell Is Where You Find It*, illustrates. In David Hare's play, *Plenty*, Sir Leonard Darwin observes that "in the diplomatic service it isn't as if a mad wife is any kind of professional disadvantage... Some of our senior men, their wives are absolutely barking". Despite such current use, barking is not described in psychiatric texts as a sign of mental illness. I have recently seen a patient in whom barking was part of the clinical picture.

Case Report: A sixty-year-old Irish divorcee had lived alone in a council flat since separation from her husband four years previously. Two months prior to the onset of symptoms she had retired from her work as a caterer. At presentation she gave a four-month history of feeling depressed and anxious with loss of appetite and energy. Two months previously, while on a shopping trip with her sister, she had started to grimace and make growling noises in a supermarket. She continued to make barking and growling noises, frequently on buses and in shops, until, finally, she was admitted for assessment.

While on the ward it became evident that her barking was partly under voluntary control: she was able to reduce its frequency, but not abolish it altogether, upon being asked to do so. She was agitated, but showed no clear evidence that she was suffering from a depressive illness. She was treated with diazepam (2 mg t.i.d.), and over the course of a month her agitation progressively resolved. She stopped grimacing and making growling or barking sounds and was discharged.

I wonder if others have observed this symptom which, even in the diplomatic service, would seem likely to confer some disadvantage.

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Ofloxacin-induced Psychosis

SIR: The new quinolone derivatives, also known as gyrase inhibitors and widely used as potent chemotherapeutic agents, have frequently been implicated in causing central nervous system side-effects (Blomer *et al*, 1986; Cohen *et al*, 1984; Gleckman *et al*, 1979). Until now, only a few patients who have developed central nervous system side-effects have been seen by psychiatrists. We present two such cases, both induced by the latest gyrase inhibitor, ofloxacin (presently under clinical evaluation in the UK and US).

Case Reports: (i) A 28-year-old woman was given ofloxacin (200 mg po b.i.d.) over a course of 5 days for recurrent scalp infections. On the night of day one, she experienced a decreased need for sleep, and on the following day progressed to a hypomanic state with an overly cheerful mood, increased social and physical activity, restlessness, racing thoughts, and loss of appetite. Her condition did not change until day 4, when she appeared to enter a kind of intermediate stage with predominant depressive features, lasting until day 10 (ofloxacin was discontinued on day 5): she was dysphoric and irritable , with labile affect, complained of fatigue, loss of energy, indecisiveness, and lack of concentration. At the same time, she still experienced racing of thoughts, insomnia, loss of appetite, and a 10% loss of weight. During this time she also had four panic attacks. From day 10 to day 18 she progressed to a marked depressive syndrome with loss of energy, depressive mood, loss of interest in all usual activities, continued lack of appetite, and insomnia. After day 18, she improved rapidly; 25 days after the onset of symptoms she was back to normal. Our patient had no relevant psychiatric or family history and had not received any medication in the four weeks preceding her illness. Medical examination, EEG, CT-scan, and laboratory studies were normal, except for an elevated TSH (21.1 U/ml).

(ii) A 64-year-old woman had been treated with ofloxacin (200 mg po b.i.d.) for a complicated urinary tract infection. The drug was discontinued on day three, when she progressed acutely to a catatonic state with rapidly alternating episodes of stupor and excitement: short periods of mutism, catatonic posturing, and waxy flexibility changed within minutes to states of excitement with perplexity, screaming, verbigeration, and stereotypies. Her thinking became incoherent, with marked loosening of associations. At times she was perplexed, and partly disoriented in time and situation. She had religious delusions, auditory hallucinations, and recurrent optical illusions; all symptoms were present on days one and two after ofloxacin was discontinued. She was given chlormethiazol on days three and four, and improved within hours: she was oriented, and her thinking was coherent. She still complained of pressure of thoughts and distractibility. During the following 10 days she improved steadily and was almost back to normal on day 14, with partial amnesia for the duration of the psychosis.

This patient had had a psychiatric history with a poorly defined depressive episode in full remission, 15 years previously. Prior to admission, she had been given several spasmolytic and analgesic medications for urolithiasis, and had a history of compensated hyperthyroidism (TSH 0.98 U/ml, T3 75 g%, T4 8.37 g%). Medical examinations and other laboratory investigations were normal, and CT scan and EEG were negative.

The essential feature of case (i) was a bipolar mood disturbance, and our diagnosis according to DSM–III was organic affective syndrome. Using these criteria in case (ii), we diagnosed both an atypical organic brain syndrome and an organic delusional syndrome. According to ICD-9, our diagnosis in both cases was subacute organic psychosis.

Ofloxacin seems to be potent enough to produce different yet well-defined psychiatric syndromes. We have received an increasing number of reports on ofloxacin-induced psychoses. Further research should focus on ofloxacin's ability to generate model psychoses.

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Oxymethalone and Aggression

Sir: An 18 year-old male, treated with oxymethalone for idiopathic aplastic anaemia, developed a temporary increase in aggression. I believe this to be the first report of such an association with anabolic steroids prescribed for therapeutic reasons and there have been no reports to the Committee on Safety of Medicines of similar cases. However, hypomania (Freinhar & Alvarez, 1985) and depression (Sheffer *et al*, 1979) have been described as a result of this class of drug. The use of anabolic steroids in sport has led to reports of increased aggression (Strauss *et al*, 1984). Doses, however, were idiosyncratic, excessive, not medically indicated, and often involved illicit drugs which purported to contain mixtures of somatotropin, a substance called "rhesus growth factor", and various anabolic steroids. They were usually taken in large quantities, far exceeding manufacturers' recommended therapeutic ranges. Overall, 35–80% of users reported an increase in aggression; of these, 75% found this to be a desirable effect.

Case Report: Within three weeks of commencing treatment with oxymethalone (150 mg daily), the patient had broken the nose of an acquaintance, smashed two expensive guitars and considerably strained the patience of his girlfriend. He was an intelligent, insightful young man, weighing 70 kg, who was studying for his 'A' levels, and who had every chance of gaining a place at university. He stated that his temper just 'snapped' following little or no provocation. Previously he had been a placid individual, and reports from his college tutor bore this out.

He was referred to psychiatric services after he had visited his GP, as he was worried either that his parents would eject him from the family home, or that he would end up in serious legal trouble, as a consequence of his actions. He was a modest drinker of alcohol (one pint of beer per week), and denied any other drug usage. His hobbies were unremarkable, and included listening to "heavy metal" music, of a variety which explored a rich seam of mysticism. He espoused philosophical ideals of peace and harmony. Exploration of his attitudes to his physical condition (which was responding well to treatment) revealed no major difficulties. There was similarly no evidence to suggest prior familial or other areas of interpersonal conflict. Previous medical history was unremarkable; there was no history of head injury. There was no family history of aggression or epilepsy. Mental state examination revealed no major abnormality other than the presenting complaint as described. Overall, he was an extremely pleasant young man.

The patient's haematologist confirmed that increased aggression had been reported within weeks of commencement of treatment. It was also felt that it was essential to continue the therapy for the next few months, depending on the haematological response.

In view of this, a cognitive/behavioural treatment plan was commenced. Within a fortnight, aggressive thoughts had diminished considerably, with only three being recorded, and no violent action had been taken on any of them. Pre-treatment levels were considerably higher than this, with daily aggressive thoughts, and perhaps weekly aggressive, remorse-inducing deeds having been reported. One month after starting this management the dose of oxymethalone was reduced by one-third.

This case indicates that in therapeutic doses anabolic steroids may induce aggression. It also shows that cognitive/behavioural techniques can serve to modify such anger and aggression. Finally, given the widely-reported illicit, uncontrolled usage of such substances by athletes and body-builders, the forensic implications of these observations may be considerable.

I am grateful to Dr Alan King for permission to report this case.

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CORRIGENDA

Journal, June 1987, **150**, 737–751 (C. V. R. Blacker & A. W. Clare). The penultimate sentence in the first column of page 747 should read as follows: (In addition, the reported gain over the drugs-only group was lost at three months, suggesting that the benefits. . . .

Journal, June 1987, **150**, 880. Letter by Jee. In paragraph 2, ". . . remitted after a 17-day course of amantidine" should read ". . . relapsed after . . .".

British Journal of Psychiatry (1987), **151**, 565–577

Book Reviews

Editor: Sydney Crown

Ethnic Psychiatry. Edited by CHARLES B. WILKINSON. New York: Plenum. 1986. 199 pp. $25.00.

This is an impressive book arising out of several years of collaboration between psychiatrists in North America; yet its relevance for the UK is particularly poignant. It is well written, and the layout allows a ready scanning of its ideas. The book provides working definitions of race, ethnicity, and minorities, and their relevance to mental health. It is concerned with American Indians, Black Americans, Spanish-speaking peoples, orientals, and Pacific islanders within North America. It is about minorities as defined as a group of people who are differentiated from others in the same society by ethnicity, race, nationality, or religion, and who occupy subordinate positions in the communities in which they reside. The minority group may be numerically smaller, but could constitute a majority as far as populations and size is concerned, e.g. the black population in some Southern states of the US. The exclusion of minorities is preserved by the retention of power of the dominant group; power that is used for economic, political, and sexual exploitation.

The impressive aspect of this book is the way it combines straightforward accounts of the history of the minority groups in the US, their present day psychiatric and mental health needs, and the political framework within which they exist. There is a link between the impact of racial discrimination and adverse mental health. Discrimination and racism are not avoided as major issues, and the United States has not become a melting pot; the authors state that this is unlikely to occur, and mental health professionals, as well as the majority and minority cultures, need to consider the rich diversity of minorities in pluralistic society.

There is a chapter on mental health of Black Americans which is a reminder of the history of discrimination in the US, with the effect it had on psychiatric treatment and more especially on psychotherapy. Hispanic psychiatric issues are the topic of chapter 3; there are over 14 million Spanish-speaking or Hispanic Americans in the US, and over 3 million Puerto Ricans. The tight family relationships in which members rely on each other for support and assistance are discussed, and their effects, both good and bad, on mental health described. The preservation of their language and the links between Latin America and North America are preserving these ethnic characteristics. The need for therapists to understand the explanatory models of the Hispanic group, and to take into account traditional beliefs and the extent of stigma associated with mental illness by Asian Americans and Pacific Island communities, is emphasised by Yamamoto.

The final chapter is an interesting review of American Indian mental health and the lack of a culturally relevant therapeutic approach. There were 300 distinct tribal groups in North America. Alcoholism, violence, suicide, and accidental death have increased in Indian people in the past 25 years. The reasons for this include cultural ambivalence, loss of traditional culture language and beliefs, loss of control over one's life, and a learned pattern of survival behaviour.

To this reviewer it is readily apparent that some of these minority groups, together with several others, exist in an equally cohesive form in the UK. The important contribution of this book to thinking in cultural and ethnic psychiatry is that it raises the question of why similar texts have not been produced in the UK. Maybe psychiatrists with practical clinical experience and a political sensitivity should begin to pool their creative resources more efficiently.

JOHN L. COX, *Professor of Psychiatry, Department of Postgraduate Medicine, University of Keele*

The Quality of Life of Cancer Patients (Monograph Series of European Organisation for Research and Treatment of Cancer (EORTC), Vol. 17). Edited by NEIL K. AARONSON and JORN BECKMANN. New York: Raven Press. 1987. 304 pp. $68.50.

To be able to recommend a book unreservedly is a rare pleasure. This monograph is a result of the work of the EORTC study group on quality of life. Recent years have seen notable advances in the treatment of cancer as judged by such criteria as reduction in tumour volume and increased duration of survival. Unfortunately, these improvements have not always been mirrored by improvement in patients' sense of well-being. This is largely due to the distressing side-effects of treatment, particularly certain chemotherapy regimens. Growing dissatisfaction among patients with what they perceive as dehumanised high-technology medicine has focused the attention of clinicians on the quality of life of cancer patients under their care. In basing their choice of treatment on a careful consideration of the balance between the beneficial and harmful effects of various treatments, clinicians need accurate data not only about duration of treatment but also about the quality of life of the survivors.

What is "quality of life" and how can it be measured? These are the salient questions addressed in the monograph. Topics considered here range from philosophical concepts of quality of life to practical problems encountered in psychosocial cancer research. There are sections on quality of life measurements in children with cancer, in adult patients, and in those who are terminally ill. Other chapters deal with such disparate topics as the weird but by no means wonderful quack remedies currently on offer to cancer patients, differences between physicians and their patients in perception of disease and treatment, and a study of self-help groups. The contributions are lucid, concise, and informative. It would have been helpful if abstracts had been provided at the beginning of each chapter and if the various authors had used the same reference system, but these are minor quibbles. This handsomely produced monograph makes a useful and interesting contribution to the young, rapidly developing discipline of psychosocial oncology, a discipline of increasing importance for psychiatrists in general hospitals.

STEVEN GREER, *Director, Cancer Research Campaign Psychological Medicine Research Group, The Royal Marsden Hospital, Surrey*

Mental Health and Primary Care: Dutch and Israeli Experiences. Edited by G. J. VISSER, J. M. BENSING and B. P. R. GERSONS. Utrecht, The Netherlands: NIVEL. 1986. 217 pp. fl 31,50.

This book consists of the papers and subsequent discussions from a symposium sponsored by the Dutch Israeli Cultural Treaty on the interaction between mental health services and the primary care sector. The aims of the symposium were for the participants from both countries and both disciplines to meet, share their experiences, and plan for future co-operation and collaboration.

The book, like the symposium, is divided into four sections. The first presents an over-view of the organisation of primary care and mental health services in Israel and the Netherlands; the second provides the family physician's concept of mental health and mental health services; the third deals with the training that primary care physicians receive in psychiatry, both at undergraduate and postgraduate level; and the fourth section takes a look at some of the research that has already been done in this field.

Although this symposium brought together nationals of two countries with markedly different health care systems, the first two sections of the book describe more vividly the differences and sometimes difficult relations between the two disciplines, rather than the national differences, which take second place. The boundary disputes between primary care physicians and mental health specialists, both of whom now regard 'psychosocial' as well as 'psychiatric' problems to be within their domain, are an underlying theme. The gatekeeping function of family doctors who determine which patients are referred to mental health services is criticised on the one hand as protectionist and on the other as denying patients (some of whom, in the family physician's opinion, do not require medical treatment at all) the right to a specialist opinion. It is concluded in several papers that there is a need to improve communication between the primary care team and mental health services, and suggestions as to how each might work with the other in their different locations are offered.

The largest section of the book is devoted to training issues, and several papers are devoted to describing education programmes which aim to improve communication skills – both between patient and doctor, and between the professionals themselves. There is also agreement on the need for much better training in psychiatry for family doctors.

The final section on research is the weak link in the book. Three dreary, only partially completed, descriptive studies are presented and leave one feeling rather dispirited as to how all the useful and valid comments and criticism from the earlier part of the symposium can actually be put into practice.

DEBORAH J. SHARP, *Lecturer in General Practice, United Medical & Dental School of Guy's & St Thomas' Hospitals, London*

Understanding and Treating the Psychopath. By DENNIS M. DOREN. Chichester: John Wiley & Sons. 1987. 246 pp. £30.95.

This is a sound and interesting book, clearly written by an author with first hand experience of a treatment situation which he calls "not hopeless, just difficult". Perhaps many psychiatrists are not sufficiently involved with psychopaths to find the book of great value, but forensic psychiatrists, and particularly probation officers, or prison officers in a treatment facility, would find it invaluable.

The first half of the book is a comprehensive literature review of the current major theories of psychopathy – role-playing deficiency (Gough), deficient conditioning (Eysenck), stimulation seeking (Quay), low critical arousal (Hare) – written in a style which often makes them easier to assimilate than the originals. The author gives his critical comments on each, but also attempts an eclectic integration. The second half of the book, in contrast, is then virtually devoid of references and reviews of other work and gives straightforward examples of the games psychopaths play in the attempt to control their environment and how the therapist might respond, rather after the style of a behaviourist or transactional analytic method.

It is very much a personal account of individual interactions with psychopaths, and one recognises only too well all the ploys, the projections, the traps and the torment. Just reading another's account of such gives the practitioner encouragement to persist. My only disappointment is that, in contrast to the widest review of the theoretical aspects, the treatment description is restricted to one style of client-therapist interaction. There is no mention of group, peer pressures, or therapeutic community, all of which can also help to share the load, arouse self-awareness, stimulate responsibility taking, and increase self-esteem.

J. STUART WHITELEY, *Consultant-Psychotherapist, Henderson Hospital, Surrey*

Reassessing Community Care. Edited by NIGEL MALIN. Beckenham: Croom Helm. 1986. 354 pp. £22.50.

As Community Physician at Hammersmith Hospital I used to pass Consultants, in the corridors, hurrying to their next ward round. "How is the community?" they would ask cheerily, but not await my reply. For psychiatrists who are hospital-bound but have time to open a book, Malin offers an answer, in this timely collection of essays on community mental health care. Much of the experience described is drawn from work with mentally handicapped people, but the ideas and proposals have contemporary relevance in a decade that may see the closure of several large mental illness hospitals. An authoritative essay by Val Reed on community nursing indicates the transition of community psychiatric nurses from 'medical handmaids' towards a greater autonomy in assessment and therapy. David Race gives a concise outline of normalisation theory, and Alan Tyne describes programmes for training in PASS, the detailed evaluation instrument derived from normalisation. Elsewhere there are useful descriptions of experience with group homes and rehabilitation projects. Nevertheless, in his opening chapter, Malin draws attention to the underlying structural problem of mental health care in Britain. Social services departments often have progressive ideas and would like to provide more community services, but the Government's policy of rate-capping Local Authorities directly inhibits these initiatives. Health Districts, on the other hand, often have considerable resources tied up in the large institutions, but there is professional resistance to change. Only a realignment of care philosophies between the two major services will overcome the impasse and refute the prophecy that community care is destined to fail.

MARK MCCARTHY, *Senior Lecturer in Community Medicine, University College Hospital, London*

Philosophical Medical Ethics. By RAANON GILLON. Chichester: John Wiley. 1985. 189 pp. £8.50.

In the past 30 years psychiatry has gone through something of a scientific revolution. It is no longer sufficient that we base our beliefs and management on intuition or experience. We are expected to defend our decisions, giving reasons and evidence for what we do.

Clinical practice is not simply a scientific enterprise; it is also a moral one. However, in general we are not expected to bring the same sophistication and rigour in our thinking to our moral decisions as we are to our scientific ones. Gillon, a general practitioner and philosopher, challenges this. In his opinion we need to be equally critical in thinking about both aspects of clinical practice.

This book is made up of 26 essays which originally appeared in the *British Medical Journal*. Because together they form a coherent whole they are, I think, more successful as a book than they were in weekly instalments spread over half a year.

Gillon has successfully achieved his two aims. The first is to argue that doctors should take moral philosophy seriously. The second is to give an introduction to medical ethics. In less than 200 pages, a clear outline is given of moral philosophy. The reader is introduced not only to the central topics such as utilitarianism, rights, and justice, but also to many of the detailed issues, for example to the principle of double effect, to the distinction between acts and omissions, and to the doctrine of ordinary and extraordinary means. These various principles and ideas are brought to bear on several problems of medical ethics, including abortion, paternalism, confidentiality, and (of particular interest to psychiatrists) consent.

This is an exceptionally good introduction to medical ethics, which can be read with pleasure, a chapter a night for a month (with weekend off), or it will serve well as a basic text for a seminar course, with several weeks devoted to each essay.

R. A. HOPE, *Clinical Lecturer in Psychiatry, Oxford University*

Handbook of Psychiatric Drug Therapy. By STEVEN E. HYMAN and GEORGE W. ARANA. Edinburgh: Churchill Livingstone. 1987. 173 pp. £12.95.

This is a well laid out paperback that aims to be a handbook of psychiatric drug treatment. It succeeds in its aims of being a useful introduction to psychopharmacology and of providing help in careful and effective drug prescription. At the end of each chapter a few recent references are provided, but these are not comprehensive reading.

The two-page introduction to psychopharmacology contains sensible principles to guide drug therapy in psychiatry (e.g. be sure to allow for a full trial of an adequate dose before changing treatment, keep the regimen simple). Chapters 2, 3, and 4 cover antipsychotics, antidepressants, and lithium respectively. Chapter 5 describes carbamazepine and other anticonvulsants used in psychiatry, while chapter 6 tackles the benzodiazepines. The final chapter is entitled "Other agents: psychostimulants, beta adrenergic blockers and clonidine". All the chapters use a similar format and cover chemistry, details on metabolism, blood levels, absorption and mechanisms of action of the drug, side-effects (including mechanisms), indications for prescription of the drug, therapeutic usage, and consideration of populations to whom special attention needs to be given. There is also a consideration of the treatment of overdosage with the drug.

It needs to be borne in mind that this is an American book aimed at the United States market, and therefore only considers drugs available for prescription there. Thus, no consideration is given, for example, to the use of chlormethiazole, mianserine, or lofepramine. In addition, there are drugs mentioned which are not on prescription in this country, such as thiothixine, molindone and loxapine. Where diagnoses are considered these are based on DSM–III, but this should not nowadays prove a great problem to the British readership.

The authors also sensibly exclude consideration of drugs with only historic interest, such as some of the older sedatives and hypnotics. One particularly useful feature of the book is a table which provides dose equivalences for the anti-psychotics and expected side-effect profiles.

I found the book quite readable and a fairly useful handbook, but the fact that it is not fully comprehensive for a British readership and relatively expensive for such a small book might reduce its popularity in this country.

ANNA HIGGITT, *Locum Consultant Psychiatrist, The Maudsley Hospital, London*

Sexuality and Medicine. Volume 1: Conceptual Roots. Edited by EARL E. SHELP. Dordrecht, The Netherlands: Kluwer. 1986. 271 pp. £38.95

In a foreword to this volume Masters & Johnson state that this book "is an excellent example of how to present material of sexual content to the health care field". They further comment that the strength of the volume resolves around the material of ethical consideration and that the chapters on philosophical approaches to human sexual behaviour provide an additional dimension to ethical considerations. I find those opinions to be balanced and an accurate assessment of a thought-provoking volume.

The volume consists of two sections. The first largely relates to the more clinical aspects of human sexuality, and in particular the changing concepts regarding the treatment of sexual problems. Suppe's essay ("Medical and psychiatric perspectives on human sexual behaviour") concludes, "If medicine and psychiatry are to regain their lost authority over sexuality they had best recognise the truth of Kinsey's insight, put to a final rest the Victorian "theory of normal love", and commit themselves to practice rooted in and constrained by scientific research meeting the highest contemporary standards. Such a medicine and psychiatry of sexuality will be quite unlike what we are accustomed to. But what authority they enjoy will then become as legitimate as their prior authority has been unscientific and fraudulent".

The second edition, mainly of a philosophical nature, concentrates the reader's attention on matters not necessarily considered of primary importance by those individuals involved in the treatment or counselling of those with sexual difficulties. It is impossible to comment in detail on the many essays in this section, but I draw attention to Solomon's essay "Heterosex", written with clarity and a sense of humour. He concludes: "If medicine seeks to understand heterosex, physicians should study poetry, not just physiology".

Breggin, in the final essay ("Sex and love"), states: "A sexual dysfunction is a life dysfunction and a successful sexual life is inseparable from a good life".

I found this volume informative, thought-provoking, and a valuable contribution to the literature on human sexual function and dysfunction.

H. B. MILNE, *Emeritus Consultant Forensic Psychiatrist, Bradford*

Humor and Psychotherapy. By THOMAS L. KUHLMAN. Illinois: Dow Jones-Irwin. 1984. 129 pp. $22.50.

"I don't believe in growth through pain" said Peter Cook, "I would rather be stunted by pleasure". Both psychotherapists and their patients are familiar with the proposition that through suffering we can arrive at maturation. This book is part of a growing literature which proposes not only that humour and the pleasurable experiences associated with it may be an acceptable part of the therapeutic process but that humour may even promote that process. Or, in Kuhlman's words, humour may serve "as an insight vehicle" or as a valuable "intervention tactic during the psychotherapy hour". If you can take this linguistic style then this book is a good read. It provides a matter-of-fact account of the increasing acceptance of varieties of humour as a useful part of the communication between therapist and patient as well as tracing the history of this development. There are chapters relating to the acquisition of insight and the development of functional detachment to the individual's capacity for humour. Far from stunting growth, it seems humour may enhance it. Nonetheless, the risks and dangers involved are also discussed and the author is to be commended for his honesty in recounting the history of a patient who terminated therapy after "a quip" which the author made in the seventh session.

Humour does not always travel well. The experience of humour often seems peculiarly subjective, and definitions of humour can be idiosyncratic. There are some good jokes in this book, but the verbal transcripts of therapeutic sessions incorporating humour seem singularly unfunny. Humour is a tricky subject to pin down.

The task-orientated tone of the book may derive from the author's training as a clinical psychologist and his current position as Unit Chief of the Maximum Security Aggression Unit at Mendota Mental Health Institute in Madison, Wisconsin. This book will not convert the sceptics, but for those therapists who wish to feel respectable when they take humour seriously it will provide much-needed encouragement.

JANE CAMPBELL, *Associate Therapist, Warneford Hospital, Oxford*

Clinical Empathy. By DAVID M. BERGER. Northvale, NJ: Jason Aronson. 1987. 297 pp. £27.50.

This book raises many interesting issues which warrant close attention. Berger explores the concept of empathy from the two psychoanalytic schools of self-psychology and classical psychoanalysis. In terms of these two perspectives, the therapeutic value of empathy respectively resides in promoting growth and self-understanding in the patient. Empathy is defined as the process by which the therapist knows what the patient is feeling emotionally, particularly as this is related to past relationships.

The book falls into three parts. The first provides a theoretical discussion of the concept. The chapters that follow may be the most rewarding for the practising clinician, in that they illustrate with clinical examples the ways in which empathy may be enhanced. The concluding section discusses the role of empathy in other aspects of therapy, such as supervision and termination.

The author shows how insight may be fostered by the openmindedness of the therapist in oscillating between putting himself or herself in the position of the patient and stepping back and reflecting on how the patient's behaviour may be related to the past. This book deserves the careful attention of psychotherapists.

DUNCAN CRAMER, *Lecturer in Social Psychology, Loughborough University of Technology*

Dream Life, Wake Life: The Human Condition through Dreams. By GORDON GLOBUS. Albany, NY: State University of New York Press. 1987. 203 pp. $34.50 (hb), $10.95 (pb).

I found this a most enjoyable book, and recommend it to anyone with an interest in the philosophy of mind. Globus attempts an analysis of the human condition through the study of dreaming. His arguments lead, step by step, to unusual and frightening conclusions.

First, he argues against the notion that dreams are composed from memory traces of concrete waking experiences. He points out that many events in dreams have not and could not have actually happened. Dreams, therefore, cannot be a collage of memories but must be newly created experiences. At this point I was drawn to his conclusion that we have "the capacity for infinite creativity".

The next step proved more difficult to accept. He suggests that dream life and wake life are indiscernible. He says there is no difference in our unreflective experience of the life that we lead in our dreams and the life that we lead while awake. As we experience them, both seem to be really happening – so much so that philosophers such as Descartes have wondered how they could prove that they were awake and not in a dream.

Globus goes on to argue that if dreams and waking consciousness are indiscernible, then they must be determined by the same mechanisms. That is, they are both formatively creative. Our awake life is dreamed up. We don't look out at the world and perceive it as it is. Our consciousness is created from within. "In each case, dreaming and waking, the monadic organism generates a life-word, *de novo*, by abstract means". Is this disturbing conclusion fanciful? Much rests on whether or not dreams and waking consciousness are discernible in the way that Globus describes. I was not convinced.

He goes on to propose a model of the brain capable of such formative creativity and to suggest how its images may be linked to input from the outside world. The model is based on holography and the mechanisms of the immune system – a refreshing change from the idea that the brain is a sophisticated computer.

Globus has produced a challenging book based on ideas from psychoanalysis, linguistics, philosophy, and cognitive psychology. There is much to disagree with, but it does make one think again. He argues for human creativity. His book shows that he is endowed with a large dose of it.

C. R. WHYTE, *Consultant Psychotherapist, Humberstone Grange Clinic, Leicester*

Variant Sexuality: Research and Theory. Edited by GLENN D. WILSON. Beckenham: Croom Helm. 1986. 268 pp. £25.00.

I looked forward to reviewing this book, which attempts to explain why some men, seemingly well-adjusted, indulge in sexual activities judged by society to be variant. A book which updates and edits the few empirical studies and scarce systematic investigations in this area must be welcome. The book is, however, disappointing. You have to be a dedicated reader to finish it, especially when it gets into technical, mathematical, or anthropological argument. At the end the reader remains no wiser as to why people behave in this way. I had hoped that the authors would mention the clinical or therapeutic implications of their findings, but they encourage a sense of therapeutic nihilism by implying that these people cannot be helped therapeutically. This sad state of affairs reflects our deficient knowledge of the subject. Extensive research is needed.

WAGUIH R. GUIRGUIS, *Consultant Psychiatrist, St Clement's Hospital, Suffolk*

The Borderline: Current Empirical Research. Edited by THOMAS H. MCGLASHAN. Cambridge: Cambridge University Press. 1986. 116 pp. $15.00.

This little book is part of a new American Psychiatric Series, designed to bring selected material from the APA Annual Meetings to the attention of a wider audience (topics covered in similar publications include premenstrual syndrome and affective disorders in the elderly). It contains five chapters covering such areas as diagnosis, biological markers, outcome, transitional objects, and the relationship between psychodynamics and symptoms.

The individual papers are of interest, and should be read by researchers in the field. Together they remind one yet again of the persistent confusion in this area between 'borderline' as a specific psychological constellation, and 'borderline' as a phenomenological diagnosis. The case for the latter may be waning. Unlike many conference proceedings this book is attractively packaged and well printed, but nevertheless suffers from the common problems of appearing very late (three years!) and being very expensive. Psychiatrists with access to the major journals may not need to buy this book.

MARK BERELOWITZ, *Senior Registrar in Child Psychiatry, The Maudsley Hospital, London*

Psychiatry in Medical Practice. By DAVID GOLDBERG, SIDNEY BENJAMIN and FRANCIS CREED. London: Tavistock Publications. 1987. 322 pp. £12.95.

This handsomely produced paperback might be called the 'Manchester text'. It was written by three senior academic psychiatrists, with contributions from six other academic and NHS colleagues. Part 1, the clinical approach to the patient, includes history and mental state, concepts of diagnosis and aetiology, a well-written long chapter on treatment, and a particularly good guide to the formulation. Part 2 describes the syndromes of psychiatric disorder and their treatment. Part 3, concerned with disorders peculiar to stages of the human life cycle, covers mental retardation, child and adolescent psychiatry, sexual and reproductive disorders, and psychogeriatrics. Part 4 consists of two chapters: an interesting one on psychiatry for the houseman, and another on legal aspects. Finally, there is the card, a glossy five part *aide-mémoire* that includes the CAGE questionnaire, a short mood scale, the Newcastle dementia scale, and the headings of the history and mental state.

The book is aimed at the newly qualified doctor as well as the medical student, and claims to offer more guidance on treatment than the usual undergraduate text. It also claims to offer an up-to-date account of the sciences basic to psychiatry, neurochemistry (mostly pharmacological), epidemiology (well done, as you would expect from the senior author), and psychology (not very extensively dealt with). These topics are not given chapters to themselves, being discussed where appropriate in the text.

The style is generally terse but always clear, with illuminating clinical illustrations. The order in which topics are considered is unusual and interesting. The book is stimulating, and students will like it.

J. L. GIBBONS, *Professor of Psychiatry, University of Southampton*

Forty Two Lives in Treatment: A Study of Psychoanalysis and Psychotherapy. By ROBERT S. WALLERSTEIN. New York: The Guilford Press. 1986. 784 pp.

This large tome is the 69th publication which has come out of the Meninger Foundation's Psychotherapy Research Project. In the preface to the book the project is described as probably the most comprehensive as well as the most ambitious psychotherapy research programme ever conceived and carried out. From the outset it must be stressed that the Meninger Foundation is a psychoanalytic institution, that the project is concerned exclusively with psychoanalysis, and that to fully appreciate the book the reader needs to be well versed in, and sympathetic towards, the theory and methods of psychoanalysis.

The 42 analysands, upon whom the project is based, were divided, in a non-random allocation, into 22 who were to receive classical psychoanalysis, involving the establishment and ultimate resolution of a regressive transference neurosis, and 20 who were to receive expressive or supportive psychoanalytically-based psychotherapy which did not evoke a full transference regression. In the event, six of the original psychoanalysis patients were subsequently transferred to one of the two psychotherapy categories and a further six received only modified psychoanalysis. The outcomes of the different treatment modalities were broadly similar, approximately 40% of each being reported as having made "really good improvement".

The author of the book was one of the originators of the project, and he has been associated with it throughout. He has an overall grasp of the project and a familiarity with each of the 42 patients, to whom he makes recurrent reference. Here the reader is at a disadvantage, and can only gradually accumulate a picture of each patient by working through the issue-related chapters.

The study was intended to be 'uncompromisingly naturalistic' in that neither the analysands nor the analysts knew at any time throughout the course of therapy that it was in progress. The patients, who were amassed between 1954 and 1958, were considered to be difficult and challenging, requiring long-term therapy. Duration of treatment ranged from 7 months to over 10 years; at the time of publication, 23 years after the inception of the project, four of the patients were still involved in therapy. By this time eight patients were known to have died, six from "mental illness related causes".

The author considers this to be "the most intensively studied and systematically documented large collection of psychotherapy cases extant". Each patient is reported to have a 'case study' of 50–70 pages of typing summarising history, diagnosis, treatment, and outcome, and the principal objective of the project would appear to be to examine carefully why the treatments that worked worked and why those that failed failed. It would be reasonable to conclude that this meticulous teasing out of the strategies employed in the treatment of this small, albeit heterogenous, sample of patients has contributed to an understanding of what works best with whom. This would indeed appear to be one kind of science.

JOHN BIRTCHNELL, *Scientific Officer and Senior Lecturer, Institute of Psychiatry, London*

Altruism and Aggression: Biological and Social Origins. Edited by CAROLYN ZAHN-WAXTER, MARK E. CUMMINGS and RONALD LANNOTTI. Cambridge: Cambridge University Press. 1987. 337 pp. £30.00, $39.50.

Of the 12 chapters in this multi-author book, four deal with the "biological, sociobiological and ethological approaches to the study of altruism and aggression" and seven with the "development, socialisation, and mediators of altruism and aggression in children". The concluding chapter helpfully summarises the problems of research in these fields. The fact that this book, first published in 1987, is stated to be "the outcome of a conference held in 1982" (at Bethesda, Maryland) should not embarrass the reader, for some ten percent of the references are to publications in 1983 or later.

It might perhaps be a criticism that there is no formal or agreed definition of the two terms which form the title of this book. The meaning of 'aggression' is taken as obvious – but it is not made clear whether selfishness or competition are forms of aggression. *'Altruism'* "is commonly defined as regard for, or devotion to, the interests or welfare of others"; and the term is consistently used in the sense of co-operation, helping, or sharing. But this usage is quite distinct from that of the sociobiologists, for whom altruism typically means 'self-destructive behaviour performed for the benefit of others'. The difference in meaning seems to diminish the revelance of sociobiology to the problems discussed here.

No one would deny that the aim of such studies in child psychology is a worthy one, for the greater our knowledge of the development of competitive and co-operative behaviour, the better will the forces that make for hate or love, crime or service, war or peace, be understood. But the present book is evidence of the difficulties of such study. That most of the field work has been limited to white, middle-class American families is a minor criticism. The real problems lie in the elusive nature of the concepts and in the complexity of longitudinal studies. Thus, the expression of altruism and aggression may take different forms at different ages; the distinction between selfishness and self-interest may be hard; and the tendency of some children to internalise their emotions may make facial expression an unreliable guide to the detection of empathy.

No doubt because of such difficulties, there has been little consensus of finding. One finding is that the basic pattern of a child's aggressivity is present at an early age and tends to remain stable through childhood, especially in boys. Genetic factors are not discountenanced here; but, as one contributor observes with regard to the expression of emotion in boys and girls, sexual stereotyping probably begins at the moment of birth. The emphasis of the book is on the social causes of behaviour and on the prospects of special training to guide behaviour into acceptable channels.

EDWARD HARE, *Honorary Consultant Psychiatrist, The Maudsley Hospital, London*

Measuring Emotions in Infants and Children (Vol. 2). Edited by CARROLL E. IZARD and PETER B. READ. Cambridge: Cambridge University Press. 1986. 277 pp. £27.50, $39.50.

Many clinical decisions in child psychiatry require that a judgement be made on the emotional reactions being shown by an infant or young child. This can be a crucial issue when an opinion is being given on, for example, whether a child should remain with foster parents or return to its family of origin. However, our knowledge of the expressions of emotions by the under fives is limited, and any judgements must be cautious. This book is to be welcomed, as is any work in this field. Volume I, published in 1982, presented research completed by 1979. This book brings the field up to date. There are four sections: physiological approaches, facial and vocal expressions, mother/infant interactions, and the conceptualisations of emotion. Much of the work is methodological and is intended for future researchers, but throughout the clinician will find much of interest and importance. In the physiological field I was excited by the findings of differential right/left frontal lobe responses to positive and negative emotions in ten-month-old infants.

In the cognitive field, the ability of children as young as three to accurately differentiate and undertake a wide range of emotions is of importance. Child development research is progressing rapidly and becoming highly sophisticated. It is essential that these findings be fed into the clinical field, and equally essential that relevant clinical questions be fed back to the research teams.

This book would be a useful addition to a child psychiatric library.

STEPHEN N. WOLKIND, *The Maudsley Hospital, London*

Models for Mental Disorder: Conceptual Models in Psychiatry. By PETER TYRER and DEREK STEINBERG. Chichester: John Wiley. 1987. 118 pp. £6.95.

The authors have tried to draw together the various ways of conceptualising mental disorder. They identify a disease model, psychodynamic model, behavioural model, and social (environmental) model, and give an account of the advantages of each. They then describe their solution to the limitations of using only one of these models by means of their 'correlative model'. This 'eclectic's charter', as they put it, allows each of the four models to be used sequentially, or perhaps together, so as to obtain the fullest understanding of the person's difficulties and then the best therapeutic methods to employ.

The descriptions of the models, illustrated by Steinberg's engaging drawings, are clinically based and can help the student, confused by abstract and sometimes acrimonious talk of rival theories, to understand what these different approaches have to offer. The book is written in plain language, yet is detailed enough to be useful to students and postgraduates in medicine and the social sciences.

It is a valuable book in that it indicates that there is no essential incompatibility between the models, merely that they have their uses at different times and in different clinical situations. What matters is that clinicians *know* in which conceptual framework they are working at any given time, and that they do not allow themselves to believe that any one model contains the whole truth.

GLIN BENNET, *Consultant Senior Lecturer, University of Bristol*

Brain Systems Disorders and Psychotropic Drugs. By HEATHER ASHTON. Oxford: Oxford University Press. 1987. 547 pp. £40.00.

In the preface the author rightly chides those who prescribe psychotropic drugs and yet fail to appreciate the underlying brain systems on which they act. What follows is a valuable account of just those systems. There are five main divisions: the first three concern arousal and sleep, reward and punishment, and learning and memory, and then follow two sections on psychiatric disorders, one concerning mania and depression, the other schizophrenia. For each the anatomical, physiological, and biochemical/transmitter substrate is elucidated, followed by a discussion of the drugs that act on the structures in question. In the case of the 'reward and punishment', for example, alcohol and illicit compounds that have an effect are also discussed. It is pointed out that some compounds act on only one transmitter system and others have multiple effects. The author appreciates that here, as elsewhere, there is overlap and interaction, but as the book was first conceived as a teaching vehicle for undergraduates this is acceptable. What emerges, however, is a well-written book of appropriate length with 86 pages of bibliography, useful for a much wider audience. All practising psychiatrists can read it with profit, and it is a good reference book. Sadly, it may be too expensive for all those who would benefit. Arm-twisting of librarians is suggested!

D. F. SCOTT, *Consultant Neurophysiologist, The London Hospital*

Child Psychiatry: A Developmental Approach. By PHILIP GRAHAM. Oxford: Oxford University Press. 1986. 463 pp. £25.00 (hb), £12.50 (pb).

When I received this book, I must admit my initial reaction was: "Do we really need another introductory text in child psychiatry?" (thinking of the contributions made by Michael Rutter, Sula Wolff, Helen Connell, and most of all Philip Barker, whose *"Basic Child Psychiatry"* is now in its fourth edition). Graham states in his preface that he intends the book to be of use to all doctors dealing with children and their families, but especially to paediatricians and psychiatrists in training, and it seems to me that this goal is admirably realised. It is laid out in a comprehensive and systematic way, and the style is clear, pragmatic, and wholly readable.

In keeping with the subtitle (*'A developmental approach'*), the largest section of the book deals chronologically with various aspects of development: pregnancy, delivery and the neonatal period, feeding and growth, the development of social relationships, etc. There are sixteen different sections, each beginning with a review of the normal and moving on to separately headed topics or problems. Thus, the section on emotional development includes school refusal, depressive disorders, and suicide and attempted suicide, and that on sexual development includes anomalies of gender role and sexual behaviour, and pregnancy in schoolgirls. The first three chapters of the book are concerned with an overview of the subject, including classification (both the ICD–9 and DSM–III multiaxial approaches) and prevalence of psychological problems, the principles of assessment, and a review of family influences and parenting disturbances. The long chapter on development is followed by a fairly short one on specific psychiatric syndromes, picking out psychotic, hysterical, and obsessional disorders. In some respects, this seems the least satisfactory section of the book. It is oddly placed, and these particular diagnostic categories may seem to have been chosen rather arbitrarily to non-psychiatrist readers. It might have been illuminating to introduce the chapter by emphasising that these are often disabling conditions and more narrowly the province of psychiatrists than of similar other professionals.

There follow two chapters on psychosocial aspects of physical disorders, which should be particularly helpful to trainees without paediatric experience. The first and shorter of these is on general issues, such as hospitalisation and the care of the dying child; the second goes systematically through the range of specific physical conditions. The eighth chapter covers treatment approaches briefly but clearly, and it is here that the only illustrative case descriptions in the book are to be found. (It is of some comfort too that not all the cases described were unqualified therapeutic successes!).

Next comes a helpful chapter on children's services in general, clarifying the inter-relationships between different professionals and their services and pointing up the similarities and differences and the areas of overlap. The last, all too brief, chapter is on prevention. Each chapter ends with a short list of suggestions for further reading and there is a comprehensive (over thirty pages) reference list at the end of the book.

This is a splendid but very down-to-earth introductory text which, in spite of my initial reservations, I must agree fulfills the specific need identified by its author. I have already taken the opportunity of recommending it wholeheartedly to different groups of community physicians and GP trainees to whom I have lectured recently.

PATRICIA AINSWORTH, *Lecturer in Child and Adolescent Psychiatry, University of Manchester*

Object Relations, the Self, and the Group: A Conceptual Paradigm. By CHARLES ASHBACH and VICTOR L. SCHERMER. London: Routledge & Kegan Paul. 1987. 313 pp. £25.00.

The authors of this book have tried to create a synthesis between individual and group therapy. Psychoanalysis was originally only concerned with the structure of the psyche and its disturbances, and many psychoanalysts have remained sceptical of the attempts of group therapy to treat people within and as a group. On the other hand, Foulkes, the originator of group analysis in this country, after recommending a combination of individual and group therapy in the beginning, emphasised in his later writings the capacity of group analysis to deal with most kinds of disturbance. In this way, the gap between two approaches grew.

More recent developments within psychoanalysis focused on the inter-psychic elements in human growth. Object relations theory and self psychology see the interaction between mother and child as crucial for the child's development. One could say that already mother and child form a sort of mini-group with its own kind of dynamics. At this point the barrier between the intra-psychic and the inter-psychic breaks down, and the way is open for an attempt to relate individual and group therapy to each other.

Within group therapy the authors distinguish between three 'systems': the intra-psychic, the interactive (the relationship between the individual group members) and the group-qua-group. All three systems interact with each other in a great many different ways, and these are plotted in great detail in a 'group analytic grid'. It is impossible to do justice in a short review to this fundamental and scholarly contribution to an important area of theory and practice.

HANS W. COHN, *Group-Analytic Society, London*

Criminal Law and Psychiatry. By D. POWER and D. H. D. SELWOOD. London: Kluwer Law Publishers. 1987. 265 pp. £50.00.

Power is a former senior prison medical officer, and Selwood a member of the army legal service and a crown court recorder. The main part of the book comprises a number of chapters on the association between crime and sexual deviation, psychosis, psychoneurosis, mental impairment, and psychopathy. It opens with a clear account of the elements of crime, a section on the law of homicide, and a description of exemptions from criminal responsibility. The first chapter concludes with a discussion on chromosomes, which is both misplaced and has paragraphs of the text transposed. The references to the chapter on young offenders have been omitted altogether.

Other chapters which discuss social factors in crime, young offenders, and illicit drug-taking are notable for the authors' anecdotal style and dubious moral judgements. The references are almost wholly out-dated: for example, the major studies on psychosis and crime, and on delinquency, are completely ignored.

The book is overly concerned with the bureaucracy which prison medical officers suffer, and the final chapter on military psychiatry involves such unlikely bedfellows as indoctrination, brainwashing, battle fatigue, exhaustion, assassination, and urban guerilla warfare.

Pages 21–26 contain numerous errors. The prosecution cannot accept "strong evidence" of unfitness to plead, since the matter is always decided by a jury. The authors state that restriction orders are always added to hospital orders in homicide cases and that these Section 37/41 Mental Health Act 1983 cases go to special hospitals. Both assertions are incorrect.

Page 23 refers to "mental responsibilities" where it should read "responsibility". On the same page it states that three persons were tried for murder in 1963, wheras the actual figure is nearer 300. The reference to "statistics for England and Wales" should read "Criminal

Statistics". On page 24 'mental responsibility' is applied only to the exercise of will-power, but the judgement in the case of R. *v* Byrne [1960] 3 All ER 1 states: "The expression 'mental responsibility for his acts' points to a consideration of the extent to which the accused's mind is answerable for his physical acts which must include a consideration of the extent of his ability to exercise will-power to control his physical acts".

There are two major errors on page 25: that homicide committed during the course of acute psychotic illness would come within the McNaughton rules; and that psychopathy coming within the Mental Health Act 1983 definition would amount to diminished responsibility.

The publishers' blurb states that the book is the first serious attempt to counteract misunderstandings by endeavouring to bridge the gap between the criminal law and psychiatry. For those who use this text, misunderstandings will be multiplied and, following the allegory, the bridge is used at the peril of those attempting to cross.

PAUL BOWDEN, *Consultant Psychiatrist, Maudsley Hospital, London*

Issues in Diagnostic Research. Edited by CYNTHIA LAST and MICHEL HERSEN. New York: Plenum. 1987. 349 pp. $45.00.

This book would not have been published if DSM–III had not been invented. One of the main results of the introduction of DSM–III has been much greater interest in the diagnostic process. This volume is therefore a timely look at the methodology of diagnostic assessment and broader issues about its aims, achievements, and pitfalls.

The first chapter, by Theodore Millon, is 84 pages long but represents a distillate of the principles of classification that could easily have been expanded to a book in itself. It is a masterly review of different approaches to classification and emphasises the modesty of the true taxonomist. However successful our diagnoses appear to be, we must "bear in mind that these labels and transformations are not 'realities' ". The rest of this section is concerned with general issues in classification, and include a particularly useful discussion by Grove of methods of recording reliability of psychiatric diagnosis. The second section is concerned with methodological issues, the particular problems of making psychiatric diagnoses during development and over a lifetime, and biological and genetic approaches. The latter are particularly valuable because they point to other ways of achieving diagnosis, now or in the future; their advantage is that they do not depend on the vagaries of clinical symptomatology. The third part of the book is less well integrated, and discusses diagnostic problems with special subjects. These include child psychiatry, where diagnosis still appears to be much less important to its practitioners than in other parts of psychiatry, and an interesting chapter on computer-generated diagnosis by Headlund & Biewig.

Although this book is primarily for the taxonomist and research worker, it would be of value for others to read to see the directions in which psychiatry is going. The time has not yet come whereby a clinician's diagnosis is likely to be independently validated by other measures, but there are glimmerings of this on the horizon. We are all diagnosticians now.

PETER TYRER, *Consultant Psychiatrist, Mapperley Hospital, Nottingham*

Self and Identity: Psychosocial Perspectives. Edited by KRYSIA YARDLEY and TERRY HONESS. Chichester: John Wiley & Sons. 1987. 332 pp. £28.00.

This book is a curious mixture of psychology, psychiatry, and sociology with sociology as the dominant perspective. It takes the problem of the self, one of the key issues in modern philosophy, and explores the theoretical and practical implications of the concept for sociology and psychiatry.

Like any collection of essays, some of them, such as the analysis of Eastern concepts of the self, are excellent, while others are at best pedestrian. Psychoanalytic concepts of identity and object relations are well reviewed, but the contribution of the existentialists and existential analysts such as Victor Frankl and Rollo May are, for unexplained reasons, ignored.

This is primarily an Academic work with a capital A, rich in references but rather short on insights and practical strategies for clinical practice. The academic, the research worker, and the armchair theoretician will find the depth and width of scholarship in this book a rich source. The practicing clinician, however, will probably find it rather arcane and alien to the world of patient care.

L. RATNA, *Consultant Psychiatrist, Barnet General Hospital, Herts*

The Psychological Experience of Surgery. (Wiley Series in General and Clinical Psychiatry). Edited by Richard S. Blacher. Chichester: John Wiley & Sons. 1987. 236 pp. £30.45.

This is a valuable collection of essays, which I would recommend highly to liaison psychiatrists and, more particularly, surgeons and other professionals concerned with the impact of surgery on patients. Written by twelve authors, it has the advantage of avoiding monotony of style; it varies from the beautifully written chapter on denial, with nine general references, to the comprehensive chapter on superobesity with 98. There is a tendency, however, for the theme of surgical 'trauma' to be repetitious without acknowledging the fact that many patients look forward to surgery as an end to pain, discomfort, or the invasion of malignancy.

The book includes many short case histories which make the text vivid, although the emphasis on individual psychotherapy tends to be a little dismissive of the value of the diagnostic psychiatric interview and physical treatments. The last chapter redresses the balance somewhat by underlining the need for rapid goal-directed psychotherapeutic intervention. The book should contain more emphasis on the strategies used to improve the ward milieu, communication between staff and patients, and the morale of staff. Equally, perhaps, the importance of understanding the premorbid personality traits should be stressed, because these so often predict the patient's attitude to the operation and contribute to his post-operative satisfaction.

These are minor strictures on an otherwise very readable and useful book.

BERNARD ADAMS, *Consultant Psychiatrist, University College Hospital, London*

Studies in Neuropsychology: Selected Papers of Arthur Benton. Edited by L. COSTA and O. SPREEN. Oxford: Oxford University Press. 1986. 351 pp. £36.00.

Professor Benton has not published a general comprehensive work on neuropsychology; his vast contribution to the field remains scattered throughout neurological and neuropsychological journals. This welcome volume contains seminal contributions that Benton and his collaborators have made to neuropsychological investigation. It represents, however, only a fragment of his output.

The book has been divided into nine sections, each dealing with a specific neuropsychological problem. Aphasia is the topic of the first section; the two papers in this section present a historical review touching on early descriptions of aphasia and the contribution of Freud to the interpretation of aphasic disorders. Sections II and III contain articles dealing with cerebral localisation and the appraisal of the method of double stimulation in which the problem of sensory extinction is discussed. Constructional apraxia and spatial abilities is the focus of section IV, and section V is devoted to the topic of reaction time and brain disease. The so-called 'Gerstman syndrome' is the subject of three articles in section VI, and the problems of hemispheric dominance and vision are discussed in section VII. The last two sections have a more direct relevance to the psychiatrist. One deals with the topic of developmental neuropsychology, which includes Benton's seminal paper 'The concept of pseudofeeblemindedness' (1956) in which a then-popular term is carefully analysed and criticised. The final section contains two papers useful in the psychiatric clinic: "Problems of conceptual issues in neuropsychological research in ageing and dementia" (1984), and "Normal observations on neuropsychological test performance in old age" (1981).

The collection shows many facets of neuropsychology, and offers useful guidance in testing and test design. The historical emphasis of most of Benton's articles provides a useful perspective of the history of neuropsychology.

MARIA A. WYKE, *Senior Lecturer in Neuropsychology, Institute of Psychiatry, London*

The Clinical Roots of the Schizophrenia Concept: Translations of Seminal European Contributions to Schizophrenia. Edited by J. CUTTING and M. SHEPHERD. Cambridge: Cambridge University Press. 1987. 238 pp. £27.50.

Whoever writes the first history of psychiatric anthologies in English will have to observe that, since the earlier centos by White, Goshen, Hunter & McAlpine and Hirsch & Shephard, the compass of these useful (albeit non-historical) publications has gone from the general to the particular. The same historian will also have to say that this change has made things harder for would-be anthologists.

The old anthologies assumed little historical knowledge; to produce a good one it was sufficient to rummage one's library and choose the right morsels. 'Committed' anthologies, on other hand, demand such knowledge, as inclusion criteria entail a historical hypothesis. Old anthologies were difficult to review (omissions could always result from whims or quaint preferences); the new ones can be judged historically.

The book under review offers an *aperçu* of the history of schizophrenia. The material is heavily edited, but it is possible to gain an idea of the views involved. The translations are competent and clear. Because of its guiding historical hypothesis, work by Kraepelin, Bleuler, and their clientele is mostly considered. The inclusion of Kleist is more imaginative, but Wernicke is not mentioned nor indeed Meynert, particularly his work on amentia. One can understand why Kahlbaum's *Catatonia* has been left out (it is available in English translation), but not why his superb 1890 paper on 'Heboïdophrenie' has been omitted, nor why Daraszkiewicz's classic monograph on 'Hebephrenie' and Jung's work do not get a mention.

The most glaring omission concerns French works. It feels often as if inclusion has been guided more by availability than by historical intelligence. For an analysis of the earlier vicissitudes of dementia praecox in France (which, incidentally, the Germans followed with worried amusement) would have shown that during the first decade of the current century the protagonists were Marandon de Montyel, Mongeri, Parant, Monod, Deny, and Roy, and during the second Mairet and

Margarot, whose superb monograph on 'démence précoce' appeared in 1920.

But it would be unreasonable to demand that this modest work, edited by clinicians, should meet high historical standards. As it is, it offers enough pabulum for younger colleagues to feed on for many a winter night. It should therefore be included in all psychiatric libraries.

G. E. Berrios, *University Lecturer in Psychiatry and Consultant Psychiatrist, University of Cambridge*

MCQ Examinations in Psychiatry. By John M. Eagles and David A. Alexander. Aberdeen: The Aberdeen University Press. 1986. 263 pp. £10.95.

Few trainees approaching the MRCPsych examinations will be able to resist this book, which contains 300 multiple choice questions. The authors have compiled five MCQ papers based on the format of the membership examination (pre-1987) and supplied answers, referenced to support their accuracy. Apart from having a good factual knowledge, knowing when to guess is the most crucial component of good MCQ technique. Rather than offer advice on this issue, the authors invite readers to make answers with various levels of confidence (certain, doubtful or guess), and from the results to draw up their own individualised codes of guessing practice.

MCQ examinations test the breadth of the candidates' knowledge; this book aims to stimulate 'revision' as well as sharpen technique. It should continue to serve this purpose despite the recent changes in the format of the membership examination.

Paul Lelliott, *Honorary Lecturer, Institute of Psychiatry, London*

The Biochemical Basis of Neuropharmacology (5th ed.). By Jack R. Cooper, Floyd E. Bloom and Robert H. Roth. Oxford: Oxford University Press. 1986. 400 pp. £25.00 (hb), £12.50 (pb).

This is the fifth and much revised edition of the most popular introductory text on the biochemical basis of neuropharmacology, by three eminent neuroscientists. It distils the enormous literature on the biochemistry and physiology of the central nervous system with emphasis on neurotransmitters in relation to the drugs which specifically affect these functions. Current research trends in the neurosciences, such as the use of molecular genetics, are well synthesised with a critical appraisal of patterns of research design and the procedures used to obtain data. Two chapters on molecular neuropharmacology and on the modulation of synaptic transmission have been added to the classical chapters on cellular foundations of neuropharmacology, metabolism in the central nervous system, and receptors and the major chapters on specific neurotransmitters including acetylcholine, catecholamines, and neuroactive peptides. The chapter on modulation of synaptic transmission exposes the simplistic conception of point-to-point transmission between pre-synaptic and post-synaptic neurones, and offers a framework for understanding the complexity of neurotransmitter/neuromodulator/neurohormone interactions and the flexibility necessary for intergrating neurochemical with behavioural mechanisms. The authors have adhered to their original aim of presenting authoritative overviews with a few selective references rather than large bibliographies. They have also maintained the lively and entertaining style in which previous editions had been written: an earlier edition engaged the writer in what appeared to be the hitch-hikers guide to the galaxy of neurotransmitters. It is an essential reading to students of neurosciences, and it should provide a working knowledge of the subject for psychiatrists in training.

M. T. Abou-Saleh, *Senior Lecturer in Psychiatry, Royal Liverpool Hospital*

Understanding Mental Retardation. By Edward Zigler and Robert M. Hodapp. Cambridge: Cambridge University Press. 1986. 292 pp. Price £27.50 (hb), £9.95 (pb).

This book unashamedly sets out to provide a summary of the work of the Yale School of the psychology of mental retardation. For those who have valued the frequent critical reviews of Zigler and his colleagues it provides an immensely readable summary which will be valued for its scientific rigour and breadth of interest. Although nearly a third of the references are to the authors' own work, it remains an authoritative text which may be used with value by postgraduate students of all disciplines.

At a time when the concepts of social adaptation and IQ in the identification of people with mental retardation are coming under critical scrutiny, the opening chapters provide a useful account of the issues involved in the debate and argue strongly for the Yale group's insistence on a developmental perspective and aetiological approach to classification. It is persuasively concluded that overzealous avoidance of labelling may be counter-productive in depriving the person with mental handicap of the help he or she needs. For the psychiatrist, the limited discussion of personality and motivational development in a single chapter, while of theoretical interest, will seem too detached from the problems of severe maladaptive behaviour and the difficult issues of their everyday management.

Of more interest are the reviews of specific interventions, particularly in young infants, and the critical account of the search for 'miracle cures' which will prove of particular value in helping to answer the questions of concerned parents.

All through the book there is an emphasis on a humane and caring outlook towards retarded individuals, which tempers the scientific and the critical discussion and search for increased knowledge which provides the main strength of the text.

J. A. CORBETT, *Professor of Mental Handicap, The University of Birmingham*

The Royal College of Psychiatrists. By HENRY R. ROLLIN. London: The Royal College of Psychiatrists. 1987. 15 pp. £2.00.

History becomes alive when it brings insights into present day practice, as admirably demonstrated by this booklet.

A graceful view of Belgrave Square invites us to the Post-Regency mansion that houses the College for a meeting with the author, one scholar Librarian who dedicates his work to another. In tracing the history of the oldest psychiatric professional body in the world, Rollin presents a valuable brief background account of psychiatry in Britain since the 18th century. His history demonstrates that the main professional issues have not changed: each succeeding challenge was met by the foundation of Parliamentary Committee, Council, Divisions, Court of Electors, Journal, Library, etc.

However, two major pieces of unresolved business are exposed by his account. The first, from the last century, left the profession's examination, and thus its standards, in the hands of others. It was rectified by the foundation of the Royal College in 1971. This was what the halting, tortured quest was about. The second remains an issue. As the author points out, from the First World War emerged the psychopathologists. Arising from this, some day an uncomfortable decision will have to be made: should the psychiatrist find fulfillment in a biological approach in his field, or does his true vocation lie in psychopathology, as his title implies – 'healer of the psyche'?

The appeal of this elegant work to Fellow and Member is evident, but the most harassed Inceptor should not fail to put a few minutes aside to enjoy this compelling account of the antecedants and functions of his professional body.

JOHN HOWELLS, *Formerly Director, Institute of Family Psychiatry, Suffolk*

Brain Injury Rehabilitation: A Neurobehavioural Approach. By RODGER Ll. WOOD. Beckenham: Croom Helm. 1987. 196 pp. £17.95.

The author was until recently the senior psychologist at the Brain Injury Rehabilitation Unit at St Andrew's Hospital, Northampton. The main substance of the book is a series of vignettes and single-case studies illustrating the management of aggression and other inappropriate activities with behavioural techniques in a token economy setting. Flanking this core of material is a discussion of the nature and value of a 'neurobehavioural' approach. These chapters are disappointing – particularly the final one, where it is difficult to follow the author's arguments.

It is clear that the unit is managing a highly-selected group of severely brain-damaged individuals. This point is not emphasised by Wood, who fails to extend the discussion to the rehabilitation of the more mildly-afflicted majority. Also, the stressing of behavioural aspects leads to a neglect of other aspects of management such as drugs (except for carbamazepine in aggression).

The book could not be recommended to the generalist or the trainee, as better reviews can be found elsewhere (those of Miller or Levin *et al*, for instance). The clinical sections are probably of value to those involved in related work.

TOM DENING, *Research Fellow, Department of Psychiatry, University of Cambridge*

A HUNDRED YEARS AGO

The Superintendent of the Royal Edinburgh Asylum deals in a very practical manner with many points bearing on insanity in its social relations. Dealing with the question of the nature of the habitation which an asylum ought to provide for its inmates, he says: "It is now generally admitted that for a number of patients we cannot get classification and individualisation enough, if the whole institution is under one roof. We need distinct homes, and houses of different kinds. Many of the patients are capable of living ordinary lives in ordinary houses, *plus* medical supervision and skilled attendance. We need all through the *hospital* and the *home* idea — the hospital and its special arrangements predominating for patients in one state of mind, and the home for others in a different state." The death-rate was 7.7 per cent; and the recovery-rate on admissions was 38.5 per cent.

Reference

The Lancet, 31 December 1887, 1341.

Researched by Henry Rollin, Emeritus Consultant Psychiatrist, Horton Hospital, Surrey

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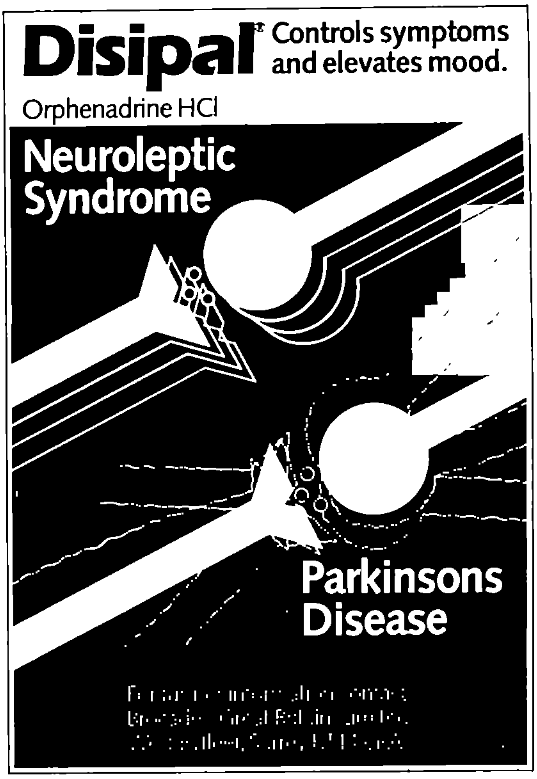

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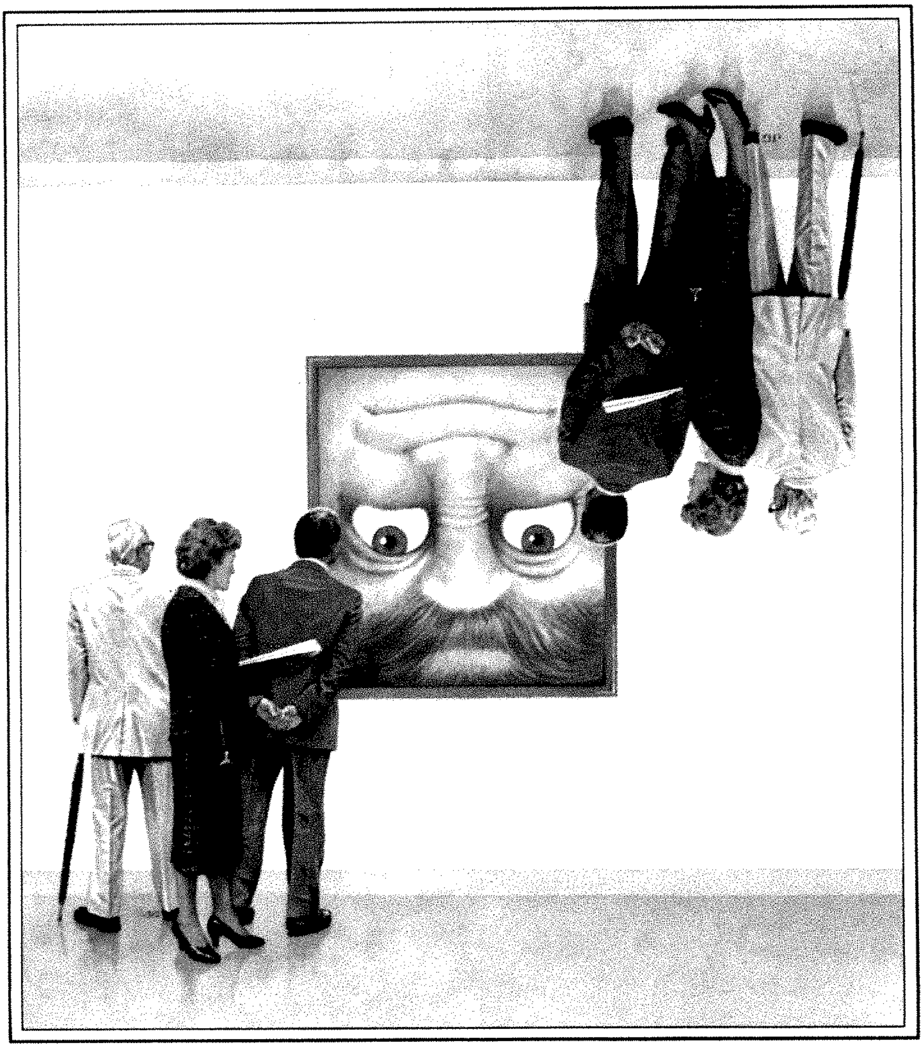

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MENTAL HEALTH

British Journal of Psychiatry (1987), **151**, 579–588

Review Article

AIDS-Related Psychiatric Disorder*

THOMAS W. FENTON

The background to HIV infection, its mode of transmission, and its neurological and psychiatric complications are described. The management of AIDS-related psychiatric disorder and problems encountered among staff involved in the management of patients suffering from AIDS are considered. There is a need for appropriate educational programmes. Although the incidence of AIDS in the UK has been appreciably lower than in many other countries, there are no grounds for complacency; psychiatric disorder associated with HIV infection will be encountered much more frequently in the future. Psychiatric staff are urged to inform and prepare themselves in anticipation of this development.

In the summer of 1981 the Centers for Disease Control (CDC) in Atlanta, USA, first notified the occurrence of illnesses that, in retrospect, were manifestations of the acquired immuno-deficiency syndrome (AIDS) (Centers for Disease Control, 1981*a,b*). It was later recognised, however, that cases of AIDS had been occurring in the USA since at least 1978. Hitherto, illnesses resulting from immunodeficiency had been encountered in patients receiving immuno-suppressant drugs for transplants and malignancies and in infants suffering from congenital immuno-deficiency. It was the reporting, in the USA, of increasing numbers of cases with an immunodeficiency syndrome but having no such cause as this, nor indeed any other apparent cause, that led to the recognition of a new disease entity.

In order to facilitate epidemiological surveillance, the CDC published a formal definition of AIDS in 1982 (Centers for Disease Control, 1982*a,b*). Essentially, this definition advised that the diagnosis of AIDS should be confined to patients having a severe disturbance of cell-mediated immunity resulting in a marked susceptibility to opportunistic infections, such as Pneumocystis pneumonia, or unusual neoplasms, such as Kaposi's sarcoma, but having no known cause of cellular immunodeficiency nor any other cause of reduced resistance reported to be associated with such deficiency.

Two further related syndromes were subsequently described, occurring in the same high-risk groups as does AIDS, but excluded as cases of AIDS by the CDC case definition. The first of these is characterised by persistent generalised lymphadenopathy (PGL), a condition occurring in the absence of symptoms, but subsequently found to be associated with the causal organism by serology. The second syndrome, termed the AIDS-related complex (ARC), is essentially a symptomatic infection with the causal organism in the absence of opportunistic infection or neoplasms. The AIDS-related complex is characterised by persistent generalised lymphadenopathy, severe lethargy and fatigue, significant weight loss, fever and/or nightsweats. Although PGL has appeared to date to have a relatively benign prognosis, approximately one quarter of those with ARC go on to develop opportunistic infection and/or neoplasms, thus qualifying them for the diagnosis of AIDS.

The causal organism was eventually found to be a virus, was first isolated in France in 1983, and named Lymphadenopathy-Associated Virus (LAV). It was subsequently also isolated in the USA and was termed the Human T-Cell Lymphotropic Virus Type III (HTLV-III). The latter nomenclature is also used in the UK. Further isolations were made in France and in the USA and labelled with a variety of acronyms. It has been agreed, however, that all of these viruses, although differently named, are closely related. The International Committee on the Taxonomy of Viruses proposed that the term Human Immunodeficiency Virus (HIV) be used to cover all of these isolates. This proposal has now achieved international acceptance. This paper will employ the term HIV hereafter. The organism is a lentivirus; that is, its replication and the evolution of its morbid pathological effects are very slow. It is also a retrovirus, and therefore transcribes its own genetic code back into the nucleus of the host cell, so modifying it that it continues to manufacture virus until it dies, resulting in a persistent infection. The virus infects the T4-helper lymphocytes which control immune effector mechanisms that attack and destroy invading organisms. T-helper cells become depleted and functionally defective, resulting in reduced stimulation of B-lymphocytes secreting specific antibodies, and reduced stimulation of macrophages and natural killer and killer lymphocytes which attack invading organisms.

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*Revised version of a paper presented at a meeting of the Midlands Division of the Royal College of Psychiatrists, April 1986.

Thus HIV undermines and compromises the body's defences against alien organisms, rendering it vulnerable to a wide range of infections. Micro-organisms in the environment, and especially those normally found dwelling within various tissues of the body, seize the opportunity created by a compromised immune system to proliferate. Patients are extremely susceptible to opportunistic agents such as viruses, fungi and protozoa. Immunodeficiency also leads to the development of certain types of neoplasm, including, in particular, Kaposi's sarcoma and non-Hodgkin's lymphoma. When HIV infection occurs the host develops antibodies to viral proteins, but these antibodies are unable to neutralise the virus. They do, however, serve as a useful indicator of infection in laboratory tests. The proportion of HIV infections that result in identifiable disease is still uncertain. Several weeks to months after infection occurs, most subjects develop antibodies. Seroconversion may be accompanied by a transient illness resembling glandular fever. For a variable period of time after seroconversion has occurred the virus remains in the body in a latent form. During this latent period infected persons are symptom-free but after an interval varying between several months to several years some go on to develop PGL, ARC or clinical AIDS.

Clinical AIDS appears to be the end stage of infection with HIV. The interval between infection and the development of AIDS is known to vary from one month (Jaffe *et al*, 1985*b*) to at least five years (Jaffe *et al*, 1985*a*). Estimates of the incidence of AIDS among those antibody-positive for HIV have to be based on the limited experience of the past few years. Earlier reports suggested that 9–14% of seropositive cases developed AIDS over a two-year follow-up (Melbye *et al*, 1984; Goedert *et al*, 1984). More recent reports have indicated an incidence of AIDS approximating 25% in seropositive individuals, and an incidence of 34% is reported in one study (Melbye, 1986). It seems likely that the higher rates of AIDS reported in later studies reflect a longer duration of the seropositive state in the subjects under review. While the outlook for those who are seropositive but asymptomatic is clearly uncertain at present, all must be regarded as able to transmit the infection.

Recently evidence has accumulated to indicate that HIV is not only lymphotropic but also neurotropic and can directly infect cells in the central nervous system (CNS) (Ho *et al*, 1985; Levy *et al*, 1985*a*; Shaw *et al*, 1985). A number of fascinating queries arise from this finding. Present evidence is that the occurence of CNS infection in patients with clinical AIDS is common. Is CNS infection in fact invariable? May CNS infection occur in the absence of immunosuppression, so that seropositive patients who escape AIDS or ARC may ultimately succumb to the direct attack by the virus on brain cells? If direct CNS infection can occur in the absence of immunosuppression, could the time-scale of its evolution span a considerably longer period than the evolution of immuno-deficiency? The answers to these questions must abide further experience with the disease and further research. What does seem clear, however, is that over the next decade the neurological consequences of the disease will acquire at least as great a prominence as its immunological consequences, and that the term AIDS – the acquired immunodeficiency syndrome – is no longer an adequate description of the pathological effects of the virus.

It is estimated that at the present time at least 1.5 million people are infected with HIV in the USA. To date nearly 20 000 have developed clinical AIDS (Curran, 1986), of whom about half have died. Although in the early years after 1981, a consistent doubling of cases every six months was reported, the Centers for Disease Control have recently published a report which indicates that, since the outbreak began, the period during which the number of cases of AIDS was doubled lengthened from 5 months in 1982 to 10 months in 1984–1985, and to 11 months in 1985–1986 (Centers for Disease Control, 1986). In other words, within the USA the rate of multiplication in the number of reported cases is declining; a significant factor in this decline may be the widespread adoption of 'safe sex' precautions by homosexuals (Altman, 1986; Golubjatnikov *et al*, 1983). About half a million people are believed to be infected in Europe; it has been conjectured that up to 10 million people may be infected in Central Africa. Since it was defined in 1981, AIDS has been reported throughout the world. In the UK the first case was reported at the end of 1981, and the number of cases reported monthly by the PHLS Communicable Disease Surveillance Centre (CDSC) in Colindale, London, has increased steadily, although so far the incidence of AIDS in the UK has been substantially lower than in many other countries. The CDSC estimates the number of people infected in the UK in 1986 to be of the order of 30 000. At the end of July 1986, 465 cases of clinical AIDS had been reported in the UK, with 234 deaths (CDSC, unpublished).

The virus has been isolated from blood, semen, cervical and vaginal secretions, saliva, tears, breastmilk, cerebrospinal fluid and brain. The principal mode of transmission is sexual intercourse. In the West this has to date been associated with intercourse between male homosexuals in particular, but by no means exclusively (Melbye, 1986). There is strong evidence that in Africa the dominant mode of transmission is heterosexual intercourse (Biggar, 1986; Piot & Mann, 1986). The virus is also transmitted by blood and blood products (Smithies, 1986). Therefore drug users who share needles, syringes and other equipment are at risk, as are haemophiliacs who have been treated with infected pooled factors VIII and IX. Blood transfusion has been a rare source of infection. The virus may be passed from an infected mother to the foetus during pregnancy (Lapointe *et al*, 1985), possibly also at birth and possibly in breastmilk (Ziegler *et al*, 1985). Infection has occurred through transplantation of organs from infected donors (Prompt *et al*, 1985), and through artificial insemination with semen from infected donors (Stewart *et al*, 1985). So far as health care workers are concerned, the risk of infection is probably limited to severe needle-stick injuries (Geddes, 1986). There have been many reports of needle-stick injuries occurring without the injured person becoming seropositive. So far, worldwide, there has been only one recorded case of a health care worker undoubtedly having contracted the virus; this followed a needle-stick injury resulting, at the time of the report, in seroconversion but not clinical AIDS (Lancet, 1984). There is no evidence that HIV is transmitted through casual social contact or by ingestion, droplet-spray or inhalation (Smithies, 1986). A study of 56 adult patients with AIDS or ARC and their 136 non-sexual household contacts over a period of 12 months revealed that no contact acquired infection via sharing of various household items, household facilities and close personal interaction (Saltzman *et al*, 1986).

The fear so universally evoked by AIDS has resulted from several well-established characteristics of the disease. Almost without exception it is a fatal illness; few patients with clinical AIDS live longer than about three years after the diagnosis is made and CDC reports have indicated that 80% die within two years. The diagnosis of AIDS is a catastrophic trauma for its sufferer, because it is known to run a rapidly deteriorating course with no prospect of definitive treatment and a hopeless prognosis. Since it was first identified the disease appears to have spread in a virtually exponential way. There is every reason to believe that those infected, which of course includes the many who are seropositive but symptomless, will remain infectious thereafter, acting as carriers of the virus, and may infect others without being aware that they are doing so. Taking these features into account, in conjunction with the widespread and often distorted publicity that they have received, the great alarm about AIDS which has manifested itself in society is easily understood. Agitation and fear among high risk groups, especially homosexuals, has resulted in a type of behaviour which has been termed 'AIDS panic' and which has led to homosexuals visiting their family practitioner or the local hospital demanding 'AIDS testing' on the first appearance of some skin lesion or of a persistent cough (Deuchar, 1984). AIDS-induced psychogenic states have also been described (Miller *et al*, 1985; O'Brien & Hassanyeh, 1985; Polan *et al*, 1985; Rundell *et al*, 1986; Weber & Goldmeier, 1983), in which individuals, not only those belonging to the high-risk groups, and known in some cases to be seropositive but in others not, have developed a terror of and an intense preoccupation with AIDS, leading to multiple somatic complaints with a conviction of suffering from the disease, together with associated mood disorder and occasionally a conversion reaction. Such patients may become intensely anguished, and preoccupied with thoughts of suicide, and the risk of a suicidal act may be very potent. Indeed, the risk of serious adverse reaction, including suicide, occurring in patients who have been informed that they are seropositive for HIV infection has led to the view being expressed that serological testing should not be carried out unless the patient can be provided with adequate counselling for the results (Miller *et al*, 1986). It has also been suggested that the availability of a widely used screening test creates a new population of patients who will be encountered with AIDS-related psychiatric symptoms resulting from adverse reactions to notification of a positive result (Rundell *et al*, 1986).

The media have had a key role in burdening the public with information which has often been inaccurate and misleading, has fostered the hysteria surrounding AIDS and has fed upon it in turn. Headlines such as "Gay Plague", "Lethal Scourge" and "Killer Epidemic" accompanying sensational accounts of AIDS with overtones of sin and retribution, together with descriptions of children and haemophiliacs as "the innocent victims", have stimulated the fearful imagination and fortified the prejudice of the public. Prejudice and discrimination against AIDS sufferers and their relatives and friends, and also against homosexuals and drug abusers, have been frequent and widespread.

Employers have sacked employees; landlords have evicted tenants suspected of carrying the disease. Dustmen have refused to collect refuse from the houses of known patients. Homosexuals have been refused life insurance; funeral directors have refused to handle the bodies of those who die of the disease. Prison inmates have gone on hunger strike, demanding that all homosexuals and intravenous drug abusers be banned from the kitchen; prison guards have asked for protective masks, and have demanded that all prisoners with AIDS be isolated. Parents have prevented their children from having any contact with a child known to suffer from AIDS, have kept them away from school, and have even kept them away from school where the relatives of AIDS patients attended. People have withdrawn their custom from restaurants, shops and hairdressers known to be frequented by those with AIDS. Hospital unions have tried to exclude AIDS sufferers from medical care; health care workers have been reluctant to care for them, change their beds, bathe them, take their meals or draw blood from them.

The overwhelming psychological trauma and sense of threat experienced by those who have contracted the disease is undoubtedly intensified by the frightening and often misleading media comment and by the irrational fears and negative discriminatory responses of the public. Taking account of the psychological stresses occasioned by awareness of being infected, and the interpersonal, intrafamilial, social and occupational consequences of the disease, together with the high incidence of neurological complications, that psychiatric disorder has proved to be a common accompaniment of AIDS is matter for no surprise. The American experience appears to have been that psychiatric consultations are most frequently requested to evaluate depressive symptoms, suicidal risk, and disturbed behaviour related to CNS impairment associated with dementia or delirium (Dilley *et al*, 1985; Holland & Tross, 1985). Mood disorder is a very common psychiatric complication (Rundell *et al*, 1986), and it is said that this has been most commonly diagnosed as an adjustment reaction with depressed or anxious mood (Holland & Tross, 1985). Psychotic depression is quite frequently encountered, although often, as might be anticipated, with a previous history of affective illness. Less commonly a neurotic depression is diagnosed. A mood of sadness, hopelessness and helplessness is characteristic, often associated with guilt, low self-esteem and feelings of worthlessness. Social withdrawal and isolation and also anticipatory grief are common accompaniments. Suicidal ideas may become prominent as the disease progresses, especially in those who have seen friends die from AIDS.

Obsessive–compulsive disorders are also encountered in the context of AIDS. Obsessional thoughts and behaviours may arise in the setting of depressed mood, but also occur independently. Such symptoms may arise not only in those suffering from clinical AIDS, but also in patients with ARC or those who are seropositive but otherwise well. Obsessional behaviour may take the form of repetitive checking of the body for skin rashes or other blemishes, a ritual which may occupy several hours every day (Miller, 1986). Obsessional thoughts may include unpleasant ruminations about death and dying, or a constant endeavour to recollect the past history of sexual practices and sexual partners, seeking to identify how and from whom the infection was contracted. They may also be concerned with a repetitive scanning of past events centred on fears that the patient may have accidentally transmitted infection to family or friends (Green, 1986).

There has been comment upon the common occurrence of certain psychological themes in patients showing psychiatric disorder, and which seem particularly relevant to the patient with mood disorder (Dilley *et al*, 1985; Holland & Tross, 1985; Nichols, 1985). Uncertainty surrounding the aetiology, course and treatment of the disease, and about modifications in life-style which may be required results in anger which may be directed towards caregivers. Social isolation is frequent, resulting from inability to follow occupation, diminution of sexual contacts and social withdrawal. Patients' fears that others seek to avoid them may be intensified by isolation precautions and by the failure of family and friends to support and visit them during their stay in hospital. For some patients the diagnosis of AIDS precipitates conflict about their sexual orientation. This may lead to the notion of illness being a retribution for promiscuity, and guilt about their sexual orientation. Such conflicts and related guilt may be accentuated as a result of shunning by others and may generate feelings of being unclean or dirty. Reactions of a kind experienced by those facing a life-threatening illness may also be shown. These include feelings of numbness and anger, fears of disability and of loss of body control, of pain and death. Above all there is a sense of loss: loss of physical stamina and health, of capacity for employment and income and often of relationships with others. A sense of time running out and of unfinished business in relation to interpersonal and family problems, and the possibilities of solving or easing these inevitably intensifies the depressed mood.

Neurological complications are a common feature of the disease (Levy *et al*, 1985*b*; Perry & Jacobsen, 1986; Snider *et al*, 1983). The most common forms of cerebral pathology related to

AIDS are encephalopathy, meningitis and neoplastic disease. At autopsy, evidence of encephalopathy is the most common CNS finding. This may be associated with post-mortem findings of various opportunistic infections such as the protozoal infection toxoplasmosis, producing a necrotic intracerebral mass associated with cyst formation; cerebral abscesses due to herpes-group viruses; and a progressive multifocal leukoencephalopathy due to papovavirus infection. Much the most common form of encephalopathy, however, and indeed the most common type of cerebral pathology affecting patients with AIDS, is that form which has been entitled 'subacute encephalitis' in the American literature (Levy *et al*, 1985*b*; Perry & Jacobsen, 1986; Snider *et al*, 1983). This encephalopathy is characterised by focal necrosis in grey and white matter, the formation of multiple microglial nodules in grey matter and to a lesser extent in white matter, gliosis of the cerberal cortex and subcortical nuclei associated with neuronal loss, microfocal demyelination and cerebral atrophy. This condition is now believed to be due to primary infection by HIV (Ho *et al*, 1985; Shaw *et al*, 1985). Meningitis is most often due to an opportunistic fungal infection such as cryptococcus. Aseptic meningitis has also been reported and is suspected to be due to one of the viruses of the herpes group. Although primary CNS lymphoma is the most common neoplasm encountered at autopsy, systemic lymphomas with CNS involvement have also been reported, including large-cell lymphoma, immunoblastic sarcoma, plasmacytoma and tumours resembling Burkitt's lymphoma. These systemic lymphomas frequently invade the meninges. Occasionally, cerebral and cerebellar metastases of Kaposi's sarcoma have been encountered (Levy *et al*, 1985*b*). Cerebro-vascular complications also occur, including cerebral infarction, intracerebral haemorrhage and subarachnoid haemorrhage, all presumably arising as a consequence of AIDS. Cranial nerve dysfunction and peripheral polyneuropathy may be additional manifestations of nervous system pathology associated with AIDS (Levy *et al*, 1985*b*; Snider *et al*, 1983).

Although dementia may result from cerebral opportunistic infections, it now seems certain that it is most commonly associated with primary HIV infection of the brain. This latter condition has been labelled the AIDS dementia complex (ADC). This complex is said to be the most common neurological complication of AIDS and to feature in the clinical course in the majority of AIDS patients, indeed on occasion being the predominant or even sole manifestation of infection by the virus (Price & Navia, 1986). ADC manifests itself as a combination of cognitive, motor and behavioural disturbances occurring with variable emphasis, cognitive impairment predominating in most patients, but motor or behavioural dysfunction being more prominent in others. Initial complaints of poor concentration and mental slowing, impairment of recent memory, lack of co-ordination and unsteady gait, and social withdrawal and apathy may be overlooked or regarded as features of depressive disorder. Motor disturbance may present early in the clinical course, with tremor, slowness of rapid alternating movements, and ataxia (Navia & Price, 1986). Over several months, but sometimes within the course of a few weeks, cognitive disturbance progresses remorselessly in the majority to a picture of profound global dementia associated with mutism, incontinence and eventual coma, death usually resulting from intercurrent infection. An EEG recording is the most useful investigation, eventually showing non-focal diffuse slowing, but sometimes revealing little abnormality in the early stages. Cerebrospinal fluid examination may reveal no abnormality or only a slight elevation of protein and slight cell increase. Computerised tomography reveals cortical atrophy in about two-thirds of cases.

A number of case reports have indicated the difficulty of diagnosing AIDS-related organic syndromes, especially in the early stages of the disease (Kermani *et al*, 1984; Nurnberg *et al*, 1984; Perry & Jacobsen, 1986; Thomas *et al*, 1985). Mood disturbance is common among sufferers from AIDS and this may mask the onset of dementia for some time, as may the symptoms of lethargy, apathy and withdrawal which are so frequently experienced by AIDS patients. There is the additional difficulty that dementia occurring in AIDS patients is quite frequently punctuated by episodes characterised by pronounced psychotic features which may mimic functional disorder such as schizophreniform psychosis (Perry & Jacobsen, 1986), acute paranoid psychosis (Perry & Jacobsen, 1986), depressive psychosis (Hoffman, 1984; Nurnberg *et al*, 1984; Perry & Jacobsen, 1986; Rundell *et al*, 1986), and hypomania (Kermani *et al*, 1985; Gabel *et al*, 1986; Perry & Jacobsen, 1986). Symptoms may include delusional thinking, hallucinations, suspiciousness, grandiosity, rambling and repetitive speech, and blunted affect. Mood disorder or psychotic episodes of this kind appear quite commonly to precede obvious cognitive deficit, sometimes by periods of several months. Two important considerations arise in relation to these findings. Firstly, it will be important to be alert to the possibility of AIDS-related psychiatric disorder when a major functional psychosis occurs in the context of a patient belonging to one of the known high-risk groups for AIDS. Secondly, when an apparent functional psychosis occurs in a known AIDS patient, the possibility will need to be borne in mind that the disorder reflects serious organic brain dysfunction.

There is an interesting parallel with another type of encephalitis which is also believed due to virus infection. The condition is generally referred to as subacute sclerosing panencephalitis, a collective term for several conditions which were previously identified by separate names, including Dawson's subacute inclusion body encephalitis and van Bogaert's subacute sclerosing leukoencephalitis (Lishman, 1978). In recent years evidence has accumulated to suggest that a virus similar to or identical with the measles virus is the cause of these conditions. The illness commonly presents with features of dementia but in some cases the presentation may resemble a functional psychiatric illness, and this has led to initial diagnosis of depression, schizophrenia or hysteria. The condition appears to be reminiscent of the syndrome associated with organic mental disorder related to AIDS infection.

It is not known whether CNS involvement is an invariable accompaniment of clinical AIDS. Certainly the occurrence of cognitive dysfunction is common (Price & Navia, 1986), and in one study was reported to have developed in more than 50% of patients (Holland & Tross, 1985). As well as dementia, the other type of organic mental disturbance quite frequently seen in association with clinical AIDS is a delirious reaction (Holland & Tross, 1985; Miller, 1986). This may occur in the setting of established dementia, or result from opportunistic infection of the respiratory tract, alimentary tract and central nervous system, or from malignancies in the brain.

When undertaking assessment of an AIDS patient presenting with psychiatric disorder, it will obviously be important at an early stage to determine, if possible, whether the condition has an organic basis. This will be a logical first step in the light of the known frequency of neurological complications such as encephalopathy due to HIV infection, progressive multifocal leukoencephalopathy and primary or secondary CNS neoplasms. When in-patient psychiatric care is indicated, the question arises as to what facility is best suited to the patient's needs. Those known to have AIDS or ARC and who are presenting with an acute psychotic state need to be investigated first of all in a medical ward to identify any possible organic lesion and to determine whether this is treatable or not. Once this has been done it will be appropriate for them to be transferred to a psychiatric unit, although they will need regular medical review by a physician having special experience in AIDS. Chronic psychotic patients who develop AIDS or ARC and who require in-patient care, and also AIDS sufferers who develop an intercurrent psychosis such as the depressive or manic phase of an existing manic–depressive illness, will be best managed in a psychiatric unit.

Patients will not need to be nursed in isolation unless certain problems arise which create exceptional risk of infection being transmitted (Geddes, 1986). The patients concerned will include those who are bleeding profusely, suffering from diarrhoea, or suffering from intercurrent infection with an infectious disease such as tuberculosis or salmonella; also patients who are acutely psychotic with disturbed behaviour likely to lead to violence by the patient or being offered to the patient by others. There are of course compassionate grounds for nursing in isolation those patients requiring terminal care. The risks which may be encountered in relation to managing AIDS patients in a psychiatric unit will include contamination risk from blood spilled in a violent incident or from self-injury, that risk being greatest where the person contaminated has also been injured or if contaminated blood contacts an already existing cut or graze; needle-stick injuries sustained by nursing staff when giving injections, or by doctors and pathology staff when carrying out venipuncture; and the possibility that the patient may infect others by promiscuous sexual behaviour. Staff caring for AIDS patients should exercise precautions aimed at reducing exposure to blood and body fluids and reducing transmission of opportunistic organisms (Geddes, 1986; Levy *et al*, 1985*b*). These will include the wearing of gloves and a plastic apron when attending to the patient, and when handling blood samples, tissue specimens, secretions or excretions, and items and surfaces which have been soiled by any of these. Sodium hypochlorite solution (10 000 ppm available chlorine) should be employed to disinfect after blood spillage and to disinfect work surfaces after use. Care must be taken to avoid accidental wounding with potentially contaminated material; needles should not be re-inserted into their original sheaths, but disposed of promptly in puncture-resistant containers. Contact of an open lesion with material from AIDS patients must be avoided.

When devising a strategy for the management of patients with AIDS-related psychiatric disorder, it will be important to bear in mind that characteristically AIDS is a chronic illness with acute exacerbations, although ultimately fatal. In the light of this longer perspective, counselling and a supportive type of psychotherapy will often be a very important aspect of case management, and a number of issues will require attention (Holland & Tross, 1985; Green, 1986; Miller, 1986; Perry & Jacobsen, 1986). It will be important to help the patient to understand current information about the disease, its cause, the modes of transmission, and the treatment that is available. Advice should be given on such precautions as may be necessary to prevent possible transmission of infection to partners, friends, household members, and family. Advice with regard to the adoption of 'safe sex' practices with partners will be of crucial importance. Such advice should encompass reduction in the number of sexual partners; the use of a condom and if need be extra lubrication for heterosexual intercourse; the total avoidance of anal sex, or the use of a condom and extra lubrication if anal sex is not avoided; avoidance of the transfer of body fluids, including 'deep kissing'; discussion of 'safe sex' guidelines with existing and prospective partners; the need for regular venereological screening; the desirability of keeping to body rubbing and mutual masturbation (Green, 1986). Even though it is highly unlikely that the advice will be followed, intravenous drug abusers should be firmly counselled that the only truly safe course of action for them is to renounce all forms of drug abuse. It will be essential to strongly advise that the patient should refrain from sharing drugs and equipment, and use only new needles, and also to advise about the sterilisation of equipment for injection. Interesting schemes are now being operated in Rotterdam and Amsterdam, Holland, whereby through the aegis of a private trust or the city council, drug-abusing prostitutes are offered the facility of free needle exchange on a clean-for-dirty basis (Beasley, unpublished). Such schemes have much to commend them, and it will be interesting to learn whether they will reduce the risk of seroconversion among the drug-abusing subculture.

Patients should be allowed to ventilate their anger about the negative reaction of others and public prejudice about the illness, and be helped to avoid acting out their feelings with self-destructive behaviour. They are likely to benefit from encouragement to discuss their feelings about the illness, and their fears for the future. Concern on the part of the staff for distressing symptoms and a compassionate understanding of emotional problems are obvious supportive measures. It will be helpful to offer realistic reassurance in the context of the current clinical situation, but the patient's awareness of a fatal outcome to his illness needs to be acknowledged, if expressed. This will allow appropriate attention to be given in counselling to practical matters such as arranging financial affairs, preparing a will, and perhaps seeking a reconciliation with family and friends. Patients may need and should be afforded an opportunity to discuss concerns about death, arrangements for terminal care, and a wish to seek the consolations of their religion. Discussions will also be needed with members of the family, the patient's friends and also carers in the community to provide sustained and structured social support. Referral to local self-help AIDS crisis organisations, where these exist, may prove very useful also (Nichols, 1983; Deuchar, 1984; Altman, 1986; Perry & Jacobsen, 1986). In view of the social implications of AIDS, early referral to a social worker is likely to be required for the planning of physical and financial assistance and referral to such self-help organisations.

Since mood disorder and severe stress may themselves exert immuno-suppressive effects (Stein *et al*, 1976; Bartrop *et al*, 1977; Cappel *et al*, 1978; Schliefer *et al*, 1983; Schliefer *et al*, 1984), the need for measures to alleviate anxiety and depressed mood becomes so much the stronger. With the great majority of patients, the management of anxiety is likely to play an important part in overall management (Green, 1986; Miller, 1986). Uncertainty about the disease and its treatment, preoccupation about its potential for a rapidly declining course to death, and alarm about any new physical symptoms are all potent causes for distress. The somatic accompaniments of anxiety may themselves be a source of further distress as they may be misinterpreted as additional evidence of a deterioration in physical health. Patients will need to be given an opportunity to air their anxieties about the illness, its treatment and its personal and social consequences. The nature of anxiety symptoms will require careful explanation. Relaxation training and other anxiety-management techniques will be useful adjuncts to management.

Since depressed mood is so common among AIDS patients, psychological techniques for its relief will be of importance also (Green, 1986; Miller, 1986). These are likely to involve encouraging an increase in social activity in order that benefit may be obtained from some renewal of physical stamina and the resumption of enjoyable activities and leisure pursuits. Where resumption of work is feasible, this should be encouraged as an activity tending to offer distraction from negative depressing preoccupations. Specific cognitive techniques may need to be employed to reduce negative thinking and to counter automatic assumptions that all efforts to improve the quality of life are pointless.

It will be important to be alert to the possibilities of suicide risk since many of the factors known to be associated with increased risk of suicide are operative with these patients (Holland & Tross, 1985; Green, 1986; Miller, 1986). Thus they have experienced recent trauma, including the diagnosis and consequences of the illness, many are very depressed, they may have suffered the loss of a loved one from AIDS, they may be socially isolated, their physical health is deteriorating, and they may have experienced recent financial hardship.

For very obvious reasons, patients with ARC are also likely to experience high degrees of psychological distress (Holland & Tross, 1985). The implications of the diagnosis and the uncertainty of outcome commonly dominate thinking, and indeed these patients may show even higher levels of distress than those with clinical AIDS. Counselling in regard to risk reduction by altering sexual behaviour and by taking appropriate precautions with regard to intravenous drug abuse will be of equal importance with these patients in order to limit progression of the disease and protect others. Patients with ARC may also require supportive psychotherapy informed by largely the same principles as obtain in the case of patients suffering from clinical AIDS.

Psychotropic drug therapy for AIDS patients with symptoms of anxiety, depression and psychosis is similar to that for patients with other affective and organic mental disorder. Anxiolytic medication and the use of antidepressants, particularly tricyclic drugs, may afford useful symptomatic relief, but side-effects such as postural hypotension, sedation and confusion may be more troublesome with these patients. Also, a high sensitivity to the extrapyramidal side-effects of phenothiazines and butyrophenones may be encountered, and here thioridazine may prove to be more useful than other major tranquillisers.

Unless staff are adequately prepared for the task, the management of patients with AIDS-related psychiatric disorder is likely to create a high level of anxiety, tension, and uncertainty among the staff themselves, with adverse consequences for their patients (Holland & Tross, 1985; Polan *et al*, 1985; Thompson *et al*, 1986); indeed, it has been remarked that even with optimal management of patients, psychiatric staff involved in their care are likely to experience persistent anxiety and uncertainty. Problems described include feelings of repugnance towards patients because of their illness or life-style, reluctance to nurse and avoidance of patients, a readiness to allow withdrawn patients to persist in their self-isolation, constant discussion about the risk of exposure to AIDS, fear of having developed AIDS progressing sometimes to AIDS panic (Polan *et al*, 1985). A survey of employees of a large urban hospital in America revealed that half of those responding believed that AIDS can be spread through casual social contact; 15% believed that AIDS is more common among health care workers than other groups; about half recognised that they spent less time with AIDS patients and about a third admitted that they actively avoided involvement with AIDS patients; a quarter of the workers expressed extreme anxiety about dealing with AIDS patients. Accurate knowledge about AIDS and its transmission was correlated to a high degree of significance with low anxiety as expressed by a self-rating anxiety scale, a willingness to work with AIDS patients and appropriate professional attitudes towards them (Gordin *et al*, 1986).

It seems clear then that the fears of health care workers will diminish appreciably if educational programmes and opportunities to ventilate those fears are offered to them. Educational programmes should afford opportunities to focus on certain key issues relating to staff attitudes and feelings. In this context there will be a need to encourage care workers to be aware of their own negative attitudes towards patients with AIDS, prejudice about alien life-styles and fears of contagion. They should be helped to acquire enough knowledge about bisexuality and homosexuality to understand the issues as they relate to transmission of the AIDS virus, and helped to be able to feel comfortable when discussing sexual matters with patients. The importance of being able to offer information and advice about factors contributing to AIDS infection will need to be stressed, with strong emphasis on the avoidance of moral posturing (Dilley *et al*, 1985; Holland & Tross, 1985).

Up to the present time the lack of information and preparation among psychiatric staff has been of little consequence in the UK because of the low incidence of the disease so far. It cannot be too strongly stressed, however, that there are no grounds for complacency on this score. When AIDS was first identified in the USA it seemed that it was confined to homosexual men, and so it was characterised by the media as a "Gay Plague". Soon after, it became apparent that intravenous drug abusers were contracting the disease because of their practice of sharing needles. Later it was observed that in Africa the ratio of men to women with AIDS is approximately one to one, that there is compelling evidence of heterosexual transmission, and indeed that in Africa this may be the dominant mode. There is now a growing incidence of heterosexual transmission in the West, with bisexual men, prostitutes and the partners of intravenous drug abusers acting as a 'cross-over group' in spreading the disease to the population at large. It is extremely likely that the virus is being spread rapidly by the re-use of unsterilised hypodermic syringes and contaminated needles for injections of vaccines and medicines, a practice which is widespread in Third World countries (Seale, 1986). Given these facts it has been very complacent to perpetuate the illusion that the disease will remain largely confined to the homosexual and drug-using subcultures.

AIDS is characterised by a very lengthy latent period and by lifelong infection. This creates the possibility of an enormous reservoir of individuals who, being asymptomatic and therefore unaware that they are infected, may infect others without being aware that they are doing so. The spread of HIV and the occurrence of AIDS are both increasing at an alarming rate, and it is obvious that so far we have only seen the tip of the iceberg, as it were. In some parts of Africa there is already evidence that up to 25% of the population are infected. The possibility that a significant proportion of the American population may become infected over the next decade, with a similar trend in Europe a few years delayed, presumably cannot be totally discounted.

There are indications that HIV infection of the central nervous system can occur before or in the absence of immuno-deficiency (Ho *et al*, 1985; Price & Navia, 1986). HIV shares with the measles virus and with the visna virus, an organism responsible for slowly progressive degenerative neurological disease of sheep, the property of combined lymphotropism and neurotropism. Although most of the experimental evidence relates to visna and measles, it has been suggested (Black, 1985) that probably all of these infections may persist in the central nervous system, even in the absence of clinical manifestations, for many years. If this is so, and since it appears that primary HIV infection of the brain can occur without immunosuppression, then pre-senile dementia from this cause may ultimately become a commonly encountered condition. The implication here is that if the immune system is not attacked by the virus, there may be a considerable likelihood that in the fullness of time, possibly several years later, encephalopathy and dementia may result from primary HIV brain infection. This is speculation, however; what we may confidently expect is that we will encounter AIDS-related psychiatric disorder with ever increasing frequency in the years to come. Unless an effective vaccine and/or drug therapy is discovered soon, and this seems unlikely, there is good reason to suppose that by the mid-1990s psychiatry will have become a 'front-line' speciality in the management of AIDS victims.

It is essential that mental health care workers prepare and inform themselves about the nature and scope of the problem and its attendant socio-cultural aspects. Only if they do so will they be able to manage their patients effectively and with dignity and compassion. It is hoped that this paper will be a contribution towards that end.

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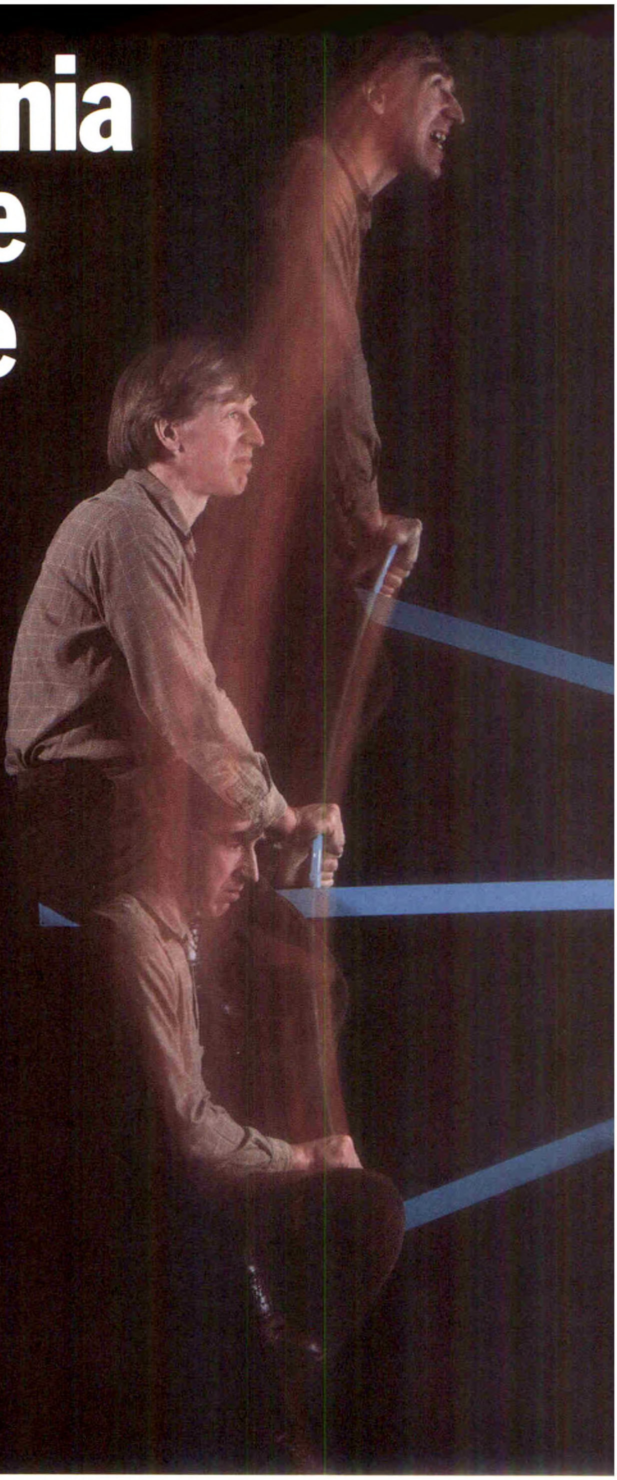

Schizophrenia therapy: the importance of balance

Too often, neuroleptic therapy swings the mood of the schizophrenic patient too far. Withdrawn patients' moods can be raised so high that they become agitated; whilst agitated patients may sink so low that they become withdrawn.

Experience shows that Piportil Depot treatment usually gets this critical balance right. Successful resocialization has been achieved even in patients who respond poorly to other neuroleptics.[1]

Piportil Depot contributes to putting the patient back into everyday life in other ways, too. Thanks to its relative freedom from extra-pyramidal effects,[2-5] and because injections need only be given every four weeks, patients hardly know they are being treated.

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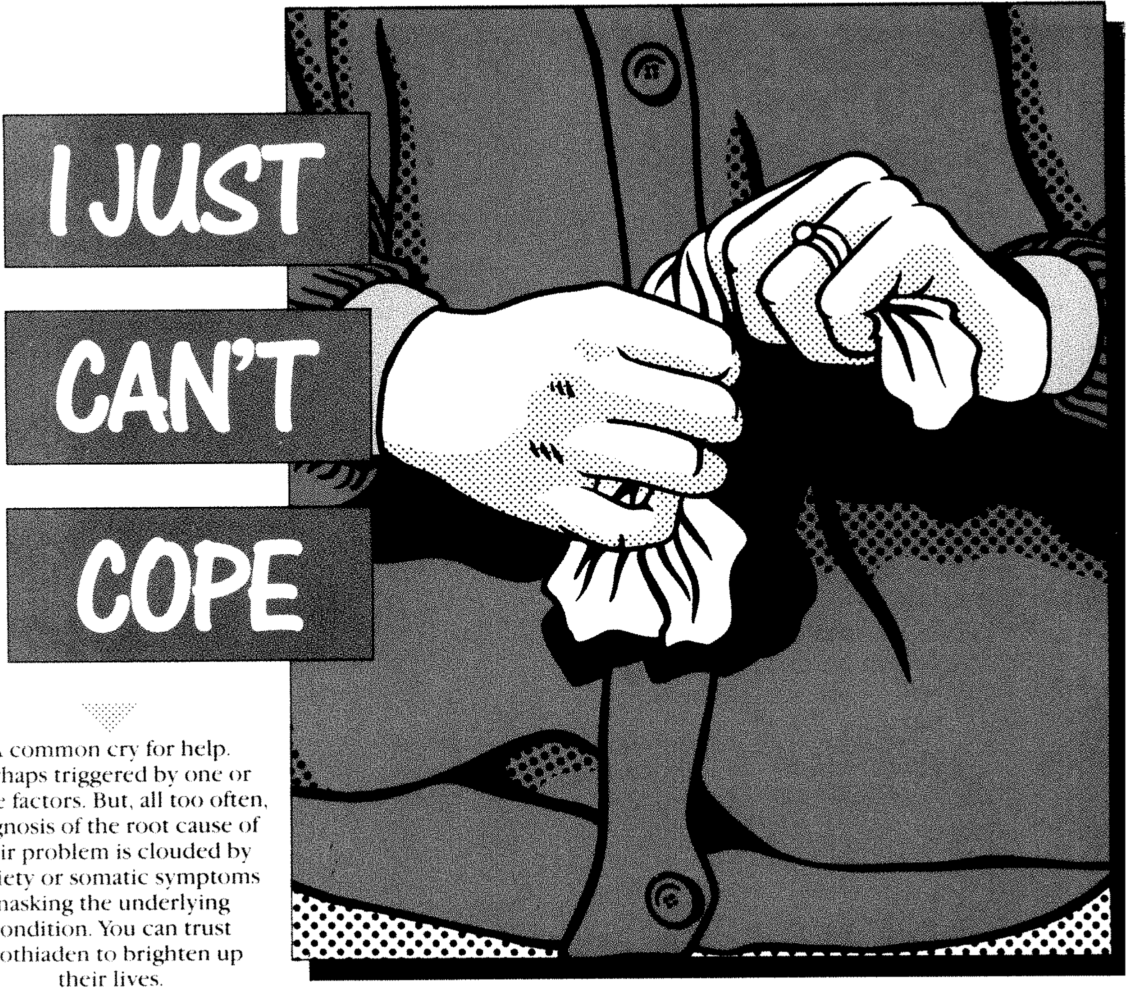
Presentation: Prothiaden is Dothiepin Hydrochloride B.P. an antidepressant of the tricyclic group. It is available as 25mg capsules and 75mg tablets. **Uses:** Prothiaden is indicated in the treatment of depression and associated anxiety. **Dosage:** Prothiaden should be given in a dosage of 75 to 150mg daily. based on response and severity. This may be taken as a single evening dose. In certain circumstances, i.e. in hospital use, Prothiaden has been given in dosages up to 225mg daily. Elderly – half the normal dose may be sufficient. **Precautions and Contra-indications:** Prothiaden has anticholinergic properties, therefore, it may precipitate urinary retention in susceptible individuals and its use should be avoided in patients with existing or potential urinary retention. Prothiaden should not be used in patients with closed-angle glaucoma. Caution is advised when treating epileptic patients and those with cardiovascular disorders. Prothiaden should not be used with MAO inhibitors nor within 14 days of ceasing such treatment. It may modify the action of some CNS depressants, catecholamines and hypotensive agents. **Side Effects:** These are usually mild and normally controlled by reducing the dosage. The following have been reported: dryness of mouth, constipation, disturbed accommodation, orthostatic hypotension, palpitations, somnolence, tremor, headache. **Basic N.H.S. Price:** 28 × 75mg £4.00 as at 1st Oct. 1987. **Product Licence Numbers:** PL0014/5941. PL0014/0209. The Boots Company PLC. Nottingham. England. Further information available on request. ETH 1174/9/87



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British Journal of Psychiatry (1987), **151**, 589–594

The Grammar of Psychotherapy
A Descriptive Account

JOHN P. COBB and STUART LIEBERMAN

The Grammar of Psychotherapy is a method of teaching both communication skills and the elements of psychotherapy. Supervised training is supplemented by self-monitored tasks. The course is economical in terms of tutors' time, and is particularly suitable for trainees in general psychiatry.

Psychiatrists spend much of their time talking with their patients. Even when not engaged in formal psychotherapy, the abilities to converse effectively and to understand their patients in psychological terms are essential psychiatric 'tools of the trade'.

Whether or not basic psychotherapeutic skills can be learned through technical training is keenly debated. The traditional view is that effective psychotherapy owes as much to non-specific factors as to technical expertise acquired through specific training (Frank, 1978). Research over the past 30 years has increasingly challenged this negative view. Rogers (1951) was the first to use audiotaped recordings of therapist–patient sessions as a means of both studying what went on in the sessions and training students. His colleagues (Truax & Carkhuff, 1967) identified specific effective therapist behaviours that could be acquired through relatively short training programmes. This approach was further refined and developed by a team of workers in Massachusetts, whose approach was labelled 'micro-counselling' (Ivey & Simek-Downing, 1980). A micro-counselling programme is structured to focus on teaching 'key' components of therapist behaviour. Control studies showed that specific skills such as attentiveness, accurate reflection, and summarisation of feelings occurring in the session can be taught by a five-hour structured training programme (Ivey & Simek-Downing, 1980). For example, after training, therapists interrupted less, spoke less and increased their response delay.

Similar work has also shown that specific interactional skills can be taught in both behavioural psychotherapy (Loeber & Weisman, 1975) and cognitive therapy (Beck *et al*, 1979). Several components are found in most training programmes. The training is introduced by a descriptive lecture, which is illustrated by a live or videotaped demonstration. Skills are then practised through role-play followed by feedback and discussion. Finally, supervision of patient interviews recorded on audio or video tapes provides a basis for discussion involving both supervisor and peer group.

This type of research has been criticised. Much of the early work done by the Rogerian school focused on therapist behaviour out of context of the interaction with the patient. More recent work has stressed the importance of looking at therapist behaviour from the patients' perspective, as well as from the view of independent judges. Much of the research has been done on neophyte therapist professionals or non-professional mental health trainees, such as undergraduates, parents, or teachers.

Several authors in the USA (Goin & Kline, 1974; Reiss, 1975; Friedmann *et al*, 1978) have discussed psychotherapy training for junior psychiatrists. Some favour traditional supervision; others found new techniques, such as videotape feedback, to be effective alternatives. In the UK, Cox *et al* (1981*a,b*) showed that psychiatric interviewing style could be studied effectively and conclusions drawn about the effects of various styles. Recently Goldberg *et al* (1984) and Maguire *et al* (1984) evaluated a teaching package which aimed to teach 'The Conversational Model' (Hobson, 1977) of psychotherapy to junior psychiatrists. This package included a booklet, two self-teaching videotapes, eight weekly seminars led by a trainer who supervised role-play, and videotape feedback of trainees' interviews. Basic psychotherapy skills can be taught to junior doctors using such a training package. Similar results have been found in teaching interviewing skills to medical students (Maguire *et al*, 1978).

Postgraduate psychotherapy training for psychiatrists may be viewed as a 'soft' area as far as data is concerned and thereby does not lend itself easily to scientific studies. Above all is the difficulty of determining the meaning of 'success' as far as training is concerned. Therapists' behaviour which is approved of by trainers may not necessarily be that which is effective in producing change with patients.

In any case, such change is difficult to measure. Because of the time-consuming nature of measurement, the number of trainee studies is likely to be small (Matarazzo, 1978). The population of patients varies from centre to centre, as does the general attitude towards psychotherapy as a method of treatment. Psychotherapeutic methods themselves vary widely, and the personality of teachers may be highly influential with trainees, but is difficult to quantify. The time available for training is variable, as is the commitment given by each junior doctor. Despite these objections, surveys of general consultant psychiatrists indicate that they recognise the importance of training in psychotherapeutic skills (Lieberman *et al*, 1978; Hafner *et al*, 1977; Scorer, 1985).

Course

Aims and structure

Faced with the task of teaching psychotherapeutic skills to over 100 psychiatric trainees scattered throughout the South West Thames Region, we have independently developed a systematic course whose content is derived from work done by Maguire *et al* (1978), Heron (1975), and Ivey & Simek-Downing (1980), and whose philosophy follows that of Sullivan, Freud and Rogers. The aims are to teach

(a) simple interview skills such as the collection of objective information, the communication of clear instructions and ways of structuring the interview
(b) complex psychological and interactional skills such as the ability to be sensitive to important psychological and emotional issues, the capacity to construct possible alternative hypotheses to understand the patient's problems in psychological terms; the ability to circumvent or modify rigid attitudes; and an awareness of the importance of the therapeutic relationship and the various factors which can modify this.

'Grammar' may be defined as the art and science concerned with a language's inflections or other means of showing relations between words as used in speech or writing, its phonetic system, and the established rules for using these. We use the word 'grammar' as a metaphor because it sums up our belief that we are teaching the study of components of that process of communication which is the basis of a working relationship between therapist and patient.

For teaching purposes we looked at three different levels of interaction. The most superficial of these focuses on the basic skills used by the therapist. Deficiencies in these basic skills may for example be demonstrated by the trainee who finds difficulty in getting patients to talk at the beginning of the interview and equal difficulty in stopping them talking at the end. The second level looks at a wider issue stressed in the writings of Ivey & Simek-Downing (1980) and Heron (1975). This is concerned with the therapist's intent in the interview and is one of the key factors which distinguishes a professional interview from an ordinary conversation. A common example of the way that this can go wrong is when a trainee who intends to take a history starts the interview with a series of confronting statements, and is then surprised that the patient clams up.

The final level of interaction looks at the interview as a whole and asks the questions "What was the therapist's understanding of the patient's problem?" and "What were the factors affecting the therapeutic relationship?".

General description

The Grammar of Psychotherapy course was planned to include 13½ hours of teaching time, starting with six consecutive weekly seminars. Three 1½ hour seminars were devoted to theoretical teaching using both lecture and role-play. Three written handouts were distributed and a multiple choice question (MCQ) test on the content was administered following the last theoretical seminar.

The theoretical seminars were followed by three 1½ hour audiotape supervision sessions in which small groups of students (3–6) presented their audiotapes for scrutiny by peers and one of the trainers.

Three follow-up supervision sessions took place six months later. These were consolidation sessions of a similar nature to the last three feedback sessions.

Specific components

The specific components included the interactograms, the role-play and the audiotape supervision sessions.

Reading material was prepared from various sources and presented as handouts. The first handout provided some theoretical information about the developmental aspects of verbal and non-verbal communication and interpersonal relationships. The second handout was about verbal and vocal communication skills. The third handout went into a number of factors which determine the way in which therapeutic relationships develop. Students were given an MCQ test which was based on this material.

Interactograms

The interactograms served to engage the students by involving them in a series of self-monitoring tasks. Attention was focused by those tasks on the students' current range of interactional skills, and psychological understanding. Trainees were encouraged to discuss their own interactograms and to pick out the aspects of their interviewing styles with which they were satisfied and also those things they would like to change. They were invited to show their interactograms to the rest of the group to ask for comment. Interactogram 1 deals with the simple basic skills of interview technique. Interactogram 2 deals with the intention of the doctor while doing his or her interview with the patient. Interactogram 3 deals with the doctor's mental set and the issues of transference and countertransference. These instruments are described in greater detail in the accompanying paper (Lieberman & Cobb, 1987).

Role-play

Role-play was used extensively in the teaching seminars, and during the feedback sessions if students had difficulty in recording live patient interviews. Role-play requires the student to attempt to see the world from the patient's viewpoint, as well as giving an opportunity to rehearse new skills. Earlier in the first training session one trainer took the role of a patient and the other the role of the doctor. The patient chosen was determined through discussion with the students about their own patients. The demonstration introduced the trainees to role-play as a technique and generated audiotaped material, which was then analysed with the group using the first interactogram.

Later the students were asked to role-play patient and doctor in exercises designated to restrict the doctor's repertoire. These 'restriction exercises' were based on previous knowledge of students' patterns of interaction. For example, if the use of silence was rare, students were asked to practise pausing for at least three seconds between the patient's response and the next therapist intervention. If students used a 'cross-examination' style consisting of an endless series of questions, the rules of the restriction exercise would confine the therapist to statements only.

Later in the course, restriction exercises gave the opportunity to examine problems associated with those intents that students found difficult. In particular, students wanted help with confrontation and catharsis. For example, as an exercise on confrontation, students are divided into groups of three with a protagonist, agonist, and observer. A 'doctor' is asked to confront the 'patient' in a clinical situation. The 'observer' marks the interactogram, including which particular microskills were used. The object of the exercise is to encourage the patient to look at his behaviour without damaging the doctor–patient relationship. After five minutes the 'doctor' discusses what he did, noting both good points and those points he would like to change. The 'observer' gives an account of what he sees. The 'patient' describes his feelings and his reaction to the therapist. He discusses whether he could see what the doctor was driving at and whether change was likely. This exercise is then repeated, with everyone swapping roles until all participants have had a chance to be 'doctor', 'patient', and 'observer'.

Seminar 1

The first seminar started with a role-played interview done by the seminar leaders, which was then analysed using the first interactogram. Invariably this promoted a lively discussion concerning the nature of a particular question or statement. Questions were easy to classify, although experience showed that the traditional split into leading/non-leading, open/closed questions is inadequate. Doctors often used the 'multiple choice question'; for example, "Do you think you're having problems because your work has been too difficult, or because your marriage is under strain, or because you are worried about your children at school?" where the patient has to respond to a forced choice. A different type of question is the 'multiple question' where the doctor asks a number of questions at the same time, leaving the patient to choose which will be answered. For example, "How have you been this week, have things worked out the way you expected, and have you had any side-effects from your medication?". The point was made during this discussion that there were no such things as 'good' or 'bad' interventions, but that some were effective and others counterproductive. Any strategy could have a use with certain patients at certain points in the interview. For example, direct leading questions, which are considered to be inadmissible by many, turned out to be used in practice by experienced therapists. They are inappropriate at the beginning of an interview where the interviewer is attempting to open up discussion, but they may be a perfectly acceptable way either of focusing on specific points of information or of establishing control in an interview which is getting out of hand. Statements fit less easily into neat categories, and during the evolution of our course we changed the labels on our statement 'pigeon-holes' several times. Discussion about classification made the trainees aware of statements that they used well and those that they used awkwardly or not at all. For example, some trainees never used empathic statements, while others employed so much polite social reinforcement that it became meaningless. Quite commonly trainees used nothing but questions in their interviews. When asked to role-play a patient being subjected to such an interrogative style of interviewing, many of them admitted that it felt like uncomfortable persecution.

An explicit aim was to encourage trainees to widen their range of basic skills. Time and time again trainees noted that they did not use 'silence'. They recognised that following even the slightest pause they intervened so quickly as to cut off any chance the patient might have had to think. Many admitted that they considered silence in an interview to be some kind of failure, although they could see there was no rational basis for this.

As well as building up a range of basic skills, trainees (and indeed the seminar leaders) became more aware of irritating idiosyncratic habits. A common example was the frequent use of a non-verbal utterance such as "ah-ah". Less common, although more damaging to the interaction, was the tendency for some therapists to answer their own questions!

Seminar 2

Very often, discussion of the first interactogram led the trainees to point out that the same intervention expressed with different emphasis or intonation might convey a very different meaning. This led on naturally to an examination of what the doctor was trying to achieve in this part of an interview. Therapist intent can be conveniently divided into information gathering, information seeking, catalysis, prescription, confrontation, catharsis, and support (Heron, 1975). Different basic skills were needed to achieve different intents at different points during the interview. An obvious example would be the use of a combination of open questions, silence, empathic statements, and reflections, in encouraging a patient to open up and explore a particular problem. Alternatively, a combination of feedback together with supportive statements might be an effective way of achieving a confrontation. When an interview was going wrong, trainees were asked to look first of all at what they were trying to achieve and secondly at what particular microskills they were using at the time. Garrulous or hypomanic patients often overwhelm the interviewer. A closer look at the microskills sometimes showed that skills appropriate for catalysis were being used, with the inevitable result! Another common difficulty was the trainee who grumbled that the patient seemed to run out of things to say. This was hardly surprising in the light of a microskills analysis which showed almost exclusive use of closed questions.

Seminar 3

By the time the trainees came to the third level they usually had become familiar with the task of looking at what they were doing in the interview rather than what their patients were doing. They were now asked to look not only at the question "What am I trying to do?" but "Why am I trying to do this?" Another way of approaching the same issue would be to ask the question "Why am I choosing to focus on this aspect of the patient's life rather than any of the other possibilities?" As a first step towards answering these questions we asked the trainees to 'brainstorm'. The object of this was to generate a number of alternative hypotheses to account for the patient's problems and symptoms. Such hypotheses could fairly easily be classified into the following categories: medical model; intrapersonal; interpersonal; and social. Alternative hypotheses were then discussed to see how much they were supported by information from the history or from observations in the sessions. If such evidence existed the hypothesis was termed a 'patient-focused hypothesis'. Very often we found little evidence to support an idea and we then had to conclude that the hypothesis came from the doctor's own head rather than being generated by the information available to him ('therapist-focused hypothesis'). A good example is the situation we termed the 'amateur psychoanalyst', in which appropriate and possibly damaging interpretations were made, often based on the latest book read by the trainee rather than on material presented by the patient. Examination of both the focus chosen by the doctor and the hypothesis used led easily to discussion of the way in which the doctor's own personality and experience was influencing interaction with the patient. Trainees were often intellectually aware of the meaning of countertransference but had not previously had experience of the way that it affected their own practice. Further evidence of this came from close analysis of sudden changes of style during an interview which at first instance appeared to be inexplicable. For example, a doctor by skilful and sensitive use of a range of microskills might encourage a patient to become emotional and to start to explore difficult and uncomfortable issues. Suddenly the tape-recording of the interview would document a dramatic change of style in that the doctor would revert to a traditional medical use of history-taking questions. Trainers were then able to ask the question, "Why at this particular point in the interview do you think you suddenly changed direction?" The other side of the coin, transference, could also be explored by following up similar openings.

Conclusion

We have run the Grammar of Psychotherapy courses for about 25 different groups of students. Most of the courses have been for trainee psychiatrists, but we have also taught other groups of paramedical personnel in both psychiatry and general medicine. Some of these courses were part of a study which compared students with matched control subjects. This allowed systematic evaluation of this teaching package, the results of which are now complete and will be reported in a subsequent paper.

Considering first the package as a whole, this was for many of our students the first time that they had ever listened to themselves talking to a patient. Laying aside for the moment the question of whether or not the specific items in our package were themselves of value, it did seem that the Grammar of Psychotherapy, with its individual 'interactograms', provides a framework which encourages trainees to tape-record sessions and then look critically at the way they interact with their patients. Merely listening to a tape of themselves can be for many trainees an intimidating and pointless procedure. However, by having to fill in an interactogram they are able to focus on certain issues which makes the process appear more worthwhile to them.

Informal feedback indicates that the course was well received by both students and clinical tutors. To quote:

> We normally do not write to individual lecturers thanking them for their contributions to our programme [statement] but in your case I feel I must write to thank you and say how impressed we all were with your series of lectures at Beechmont, St Francis Hospital. The trainees have found them extremely useful.
>
> [Silence]
>
> Our programme for next term is already arranged [another statement]. Do you feel you would like to come again to talk to the trainees during the autumn term [question]?

Courses were well attended. The structure enabled trainees to accept feedback and to involve themselves in lively discussion without feeling threatened. Surprisingly, some students gained insights into aspects of themselves through focusing attention on their own interactions with patients.

Many of the trainees attending our course are likely to go on to be general psychiatrists, and some will no doubt specialise in psychotherapy. Knowledge learnt through attending this course should be useful both to general psychiatrists and specialist psychotherapists.

In this course we avoided any particular theoretical stance and encouraged students to judge pragmatically whether a particular microskill or intervention was effective or not. The choice of course title was determined by the belief that the grammar of psychotherapy is the examination of the components that make up the relationship between therapist and patient, and that this is something that the plethora of schools of psychotherapy have in common.

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The Grammar of Psychotherapy

Interactograms: Three Self-Monitoring Instruments for Audiotape Feedback

STUART LIEBERMAN and JOHN P. COBB

This paper describes the development of three self-monitoring forms to enable students to make constructive use of audiotapes of their interviews with patients. Each interactogram is described in detail, with examples where necessary.

The Grammar of Psychotherapy course is a method of teaching communication skills and elements of psychotherapy, as described in the accompanying paper (Cobb & Lieberman, 1987). An essential element of the course is supervised training supplemented by self-monitoring tasks. During development of the course, audiotapes were used in supervision. Listening to audiotapes without a structured method of attending to the material was found to be unproductive. Forms were designed that students completed in the privacy of their homes or offices while listening to the interviews which they audiotaped with patients. These forms focused the attention of the students on their interactional skills and psychological understanding. We decided that we would call the three self-assessment forms 'interactograms' because they are meant to examine the interaction between the doctor and the patient. Three interactograms were constructed.

Interactogram I

The first interactogram (Fig. 1) was inspired by various authors who have developed structured methods of teaching communication skills and interviewing skills (Beckvar, 1974; Maguire *et al*, 1978). We were particularly influenced by Ivey (1971), who trained students in the use of attending behaviour, minimal activity responses, verbal following behaviour, open enquiry, and reflection of feelings. He used the term 'microskill' to describe the discrete verbal and non-verbal components of an interaction.

The first interactogram was designed to examine in minute detail each actual intervention which the therapist uses when interviewing a patient. The interactogram is divided into three major sections: questions, facilitations, and statements.

Glossary of terms

Leading questions. These often sound more like statements than questions. They imply that the questioner expects a certain answer and make it difficult for the respondent to disagree. The tone of the voice can be as important in this respect as the form of words. Leading questions are widely used in the media and in court. They tend to have a somewhat bullying quality; for example, "You *do* agree with me, don't you?"

Non-leading closed questions. In contrast to leading questions, non-leading questions do not expect any particular answer. Closed questions usually begin with the words "Is", "Are", "Do" or "Did". They can usually be answered briefly or with a "Yes" or a "No". They help to focus a discussion but they are a deterrent to wider exploration of any particular issue. Examples are, "Do you usually get angry at your boss?"; "Is there a particular type of woman that causes you problems?"

INTERACTOGRAM I
[Microskills analysis of therapist behaviour]

Questions

Non-leading open
Non-leading closed
Leading (open or closed)

Facilitation

Silence (3 seconds)
Noises

Statements

Orientation/introduction
Reassuring
Encouraging/empathic
Summarising
Checking/seeking clarification
Focusing/scanning/prompting
Self-revelation
Other (specify)

Play through a tape of your own and classify each of your interventions according to the above list. Think for yourself what impact your particular style of interviewing is likely to have on this patient.

FIG. 1.

Non-leading open questions. These usually begin with "What", "How", "Why" or "Could". They cannot be answered simply by a yes or a no, and invite wider discussion. They can make the person questioned feel more at ease and make the discussion less like a cross-examination. Examples are, "How have you been over the past week?"; "Could you tell me more about how you feel now?"

Silence. Though long silences are anxiety-provoking and can lead to a sort of battle, short silences give people time to think. The most useful length of silence in an average interview is 4–10 seconds, which strikes a reasonable balance between the two extremes. Students were encouraged to see that this was a general guideline and not an absolute rule. They were asked to think about what function they wanted the silence to serve. Under certain circumstances the general guideline might be ignored. So, in controlling a garrulous subject it might be best to reduce silence to a minimum. In contrast, if the therapist wanted to raise the patient's level of emotion, or allow time for deep thought at a sensitive moment in the interview, then a long period of silence could be appropriate.

Noises. These can be used either to encourage or discourage the other person from continuing to speak. They include noises like "Ahha" and "Umm" as well as short phrases like "OK", "I see", or simply "Yes". All of these are useful if, like salt, they are used sparingly. The danger is that with overuse they become either irritating or just plain ridiculous. Trainees soon become aware of this when listening to themselves on audiotape. In all communications the tone of voice may be as important as the semantic content. This is powerfully illustrated by discussion of the tone in which 'noises' in this group were produced.

Orientation/introduction. This refers to the initial part of the interview, which helps to set the scene. Trainees were taught to introduce themselves; to spell out the purpose of the interview right at the beginning and describe the amount of time available for the session. An example: "My name is Dr Smith and I am a psychiatrist. We have an hour and during this time I would particularly like to talk to you today about important relationships. I also want to make sure that we have time for you to tell me about things that are on your mind right now. Before the end of the session I hope we will be able to make some plans about future treatment."

Reassuring. Reassurance (Wilcox & Salkovskis, 1985) is much used by doctors, although frequently it is ineffective either because it is over-used or alternatively because it is patently untrue. Having said this, most people like to be rewarded and also to be admired and respected. Confirmation that they are doing a worthwhile job is usually welcome. The danger with reassurance, and this was often picked up by students during feedback sessions, is that the patient, eager to please and receive approval, will start to behave like a circus animal. Only those things which are likely to produce a reward are described. Thoughts, attitudes, behaviour, and emotions which the patient fears will provoke disapproval are then kept hidden.

Encouraging. This refers to any statement which is aimed at getting the other person to talk more. Examples would be, "Please tell me more"; "Please expand on that"; "I really want you to tell me more about those aspects of yourself that cause you guilt, anxiety and pain"; "Please go on from here . . . anywhere you wish". Simpler encouragement statements include "Go on" and "That's good". As has been pointed out already, statement categories overlap. Thus a statement such as, "I can see this is very tough for you but I'd like you to try to explore this further if you can", is both empathic and encouraging. For the purposes of the interactogram the student was asked to classify the statement according to the predominant intent. Lively discussion around this point might fail to produce agreement concerning the 'correct' category, but would serve to increase awareness of the impact of a particular statement on a particular patient.

Summarising. These are statements which give a précis of what the speaker has said. Provided they are brief and to the point, they show that the listener has been concentrating on what has been said. They are also useful in allowing the doctor to collect his thoughts, and highlight what appear to be key themes in the patient's description. An example is, "So in the past few minutes you've talked about this terrible fear and you feel that it is to do with the feeling that you could lose control completely and end up in a mental hospital."

Seeking clarification. Only a genius can understand everything the first time it is heard and unfortunately there are few geniuses around. For the rest of us it is important to be prepared to ask for clarification when something in the other person's communication is unclear. For example, "I think that you view things like this . . .". Beginners often assume that to admit "I didn't understand that" is to show weakness which will lower the doctor's image in the patient's eyes. Usually the reverse is the case, as illustrated by the awkward interview in which an intelligent patient got more and more entangled in obtuse and mutually contradictory self-philosophising. The therapist, bored and exasperated, eventually blurted out, "I'm sorry, I don't understand what you are saying." Whereupon the patient laughed and admitted, "Neither do I, this is the way I always carry on when I am anxious." This proved to be the beginning of more useful communication.

Checking. The main purpose of a checking statement is to ensure that the gist of the other person's discussion has been understood. For example, "From what you told me I can see that you are pretty happy at the moment but there seems to be great difficulty when it comes to finding a suitable long term goal", or "As I understand it, the key issue for you is not so much the depression itself, but that as a result of it your family will reject you." At one level a checking statement simply aims to ensure that the therapist has understood correctly. During supervision discussion, however, students realised that such statements convey other intents, such as confrontation. For example, "So you are saying to me that there is absolutely nothing you do from the moment you awake until you go to sleep which gives you even the tiniest bit of pleasure or satisfaction!"

Focusing/scanning. People often talk in paragraphs. During the course of a paragraph several different subjects may be mentioned. The listener *scans* the paragraphs and then has the opportunity to select one of the points to *focus* on for further discussion. For example, a patient may have mentioned that he is tired, it has been a long day, work has been particularly difficult and that he was called out of bed in the middle of the previous night to look after his daughter. The listener may think it is a good idea to encourage the patient to talk a little more about his tiredness, and respond by comment along the lines, "I don't know how you keep going when you must be feeling very tired." Alternatively, if interpersonal issues are a major problem, then the therapist may decide to focus on the daughter: "How did you feel about disturbing your sleep for her?"

Prompting. This refers to any device which may encourage the speaker to continue talking along the same lines. A well used technique is to repeat the last word or phrase of the speaker's sentence. An alternative is 'the unfinished sentence'. Here the therapist starts a sentence but dries up part way through. The natural response is for the listener to complete the sentence and continue talking: "So the death of your father . . ." or "The pills you are having . . .".

Empathic. An empathic statement indicates that the speaker has some idea of what the other person is feeling. To be effective, empathic statements need to be accurate and may need considerable skill in interpreting both verbal and non-verbal cues. For example, "Yes, you feel you've just about had enough today", "You are on the verge of tears right now", or "I think for two pins you would like to bash someone on the nose at this moment." Empathic statements have a powerful effect in enabling a patient to express emotions, especially if used in the here and now. Inappropriate use can cause difficulties. Expressed empathy near the end of a session may lead to a flood of emotion which has to be cut off when time runs out. Too much empathy, especially over past events – "You have been through it"; "It must have been awful" – can come over as patronising, a caricature of a caring professional.

Self-revelation. Used with care, self-disclosure can be a powerful way of encouraging people to talk more about themselves. Such self-revelation must be honest, and in ordinary circumstances ought to be limited so as not to overwhelm the patient. Self-revelation can involve feelings that the therapist is experiencing at the time of the interview; for example, "You know I'm feeling a bit apprehensive because I realise you are angry about being brought into hospital against your wishes". Alternatively they can involve experiences in the therapist's own life which seem relevant to the patient's difficulties; for example, "When my mother died I thought I knew all about mourning and could work it through myself. It was only six months later when I was in a real state that I was finally able to listen to a close friend and accept that I needed help."

Use of interactogram I

Interactogram I was used by trainees to monitor – at a microskill level – interviews with patients which had been tape-recorded either on the ward or in outpatients. Trainees were asked to classify each intervention they made and to record it on the interactogram form. Five-minute samples were analysed in this way. Often in different interviews, with different patients (or role-played patients) in different settings, characteristic patterns emerged in their interactograms time and again. Such patterns could be described as 'interactive fingerprints'. A good example (Fig. 2) is the typical medical model pattern which is probably the product of traditional medical student training (Maguire *et al*, 1978). Once completed, the interactogram provided the basis for feedback. Each trainee was asked to comment on his or her own form and to note the range and frequency of microskills employed, before showing the interactograms to other members of the seminar for their observations. Feedback was pragmatic. Trainees were discouraged from saying that any particular style was "right" or "wrong". Rather, they were asked to consider the questions, "What is the likely effect on the patient of this combination of microskills?" and "Is this style appropriate for the task in hand?"

INTERACTOGRAM I
[Microskills analysis of therapist behaviour]

Questions	
Non-leading open	✓
Non-leading closed	✓✓✓✓✓✓✓✓✓✓✓✓✓✓✓✓✓✓
Leading (open or closed)	✓✓
Facilitation	
Silence (3 seconds)	
Noises	✓✓
Statements	
Orientation/introduction	✓
Reassuring	✓✓
Encouraging/empathic	
Summarising	
Checking/seeking clarification	✓
Focusing/scanning/prompting	
Self-revelation	
Other (specify)	

FIG. 2. Typical 'medical model' interactogram, characterised by predominant use of non-leading closed questions.

Interactogram II

Interactogram II (Fig. 3) builds upon the work done in the previous stage of training. During categorisation of the different types of microskill, trainees became involved with the question, "Why is this particular skill, or this particular group of skills, being used in this interview at this particular time?" Teaching thus focused on the topic of therapist intent. The second interactogram is a device to aid this and is based on the Six Category Intervention Analysis (Heron, 1975). Heron claims that the six categories provide a comprehensive framework for all intentions manifest during the course of an interaction with a patient. As such they constitute a powerful analytic training tool for anyone who wishes to build up skills, and particularly aid the development of self-assessment and self-monitoring in the helping professions.

The six categories are prescriptive; informative; confronting; cathartic; catalytic; and supportive.

Authoritative categories

Prescriptive, informative, and confronting intents are authoritative, that is to say it is the therapist who is taking the overtly dominant or assertive role.

Prescriptive. The intent here is to give advice or to evaluate, be critical or judgemental; a prescriptive intent is one that seeks to direct the behaviour of the patient, especially behaviour that is outside the therapeutic relationship; for example, "This evening I would like you to try and persuade your husband

INTERACTOGRAM II
[Microskills analysis of impact of therapist behaviour on patient]

Therapist intent	*Microskill*	*Focus*	*Effect on patient*
(Prescriptive, informative, confronting, cathartic, catalytic, supportive)	Question, statement etc.	Patient/therapist	Mild/moderate/strong positive or negative
(e.g. Catalytic)	(e.g. Self-revelation)	(e.g. Therapist)	(e.g. Mildly inhibiting)

Fig. 3.

to join us next week. Make a note of how the discussion goes so that even if he can't or won't come, we can talk about the way you set about approaching him over difficult matters."

Informative. The informative intent is to teach, to instruct, to inform, to interpret, to impart new knowledge and information to the patient. Although trainees recognise that 'lecturettes' are poor ways of putting across information, many fall into the trap of 'talking at' their patients and hoping for the best. Diagrams, booklets, charts may be used, and above all the therapist is encouraged to check that the patient has understood key information; for example, "Just tell me what your tablets are called, how many you are going to take and at what times?"

Confronting. This is to give direct feedback, to challenge, to try to alter the attitudes of belief or behaviour of the patient, in a sense to hold up a mirror to show the patient what he or she is like, either directly in regard to the relationship between the patient and the therapist or in the way in which the patient has behaved in the past or in the present outside the therapy situation. Confrontation can easily degenerate into a battle. The therapist becomes exasperated and angry, and the patient alienated and even more stuck with unproductive attitudes and patterns of behaviour. Effective confrontations help the patient to take a good look at himself, without feeling 'got at' by the therapist. Careful balance of microskills may be crucial in making the difference; for example, "Well, we have talked for about an hour. I can see you are very keen to get help. Symptoms have almost overwhelmed you in the past but you've shown a lot of courage in your struggle to keep going. Yet, despite your need and your efforts, two previous attempts at therapy have failed. Something in you seems to drive away therapists in the same way you have driven away other people in your life. History has a habit of repeating itself unless we learn from the past. So, to avoid this happening again with us, I want you to ask yourself the question – what is this destructive thing in me, which pushes people away and leaves me isolated and miserable?"

Facilitative categories

The next three categories are considered facilitative because the therapist is less obtrusive, more discreet, and seeks to elicit some kind of state of being in the client.

Cathartic. The intent here is to get the patient to feel, to show his or her feelings, to react with or abreact painful emotion, encouraging for example laughter, anger, fear, crying. The use of silence, in combination with empathic statements and non-leading open questions, is helpful in achieving this intent. Sensitivity to non-verbal cues is important. Sudden alteration in the tone of voice or faltering over a certain word alert the therapist that the patient is approaching a possible point of catharsis.

Catalytic. Here the intent is to enable the patient to learn and develop through his or her own self-direction. The therapist acts as a catalyst and allows a patient to explore his own problems and to then discover his own solutions. Rogerian counsellors (Rogers, 1961) make great use of certain microskills to achieve 'catalysis'. Repetition of the last word of the patient's sentence is so well known as to be parodied (Rhinehart, 1971). Noises, silence, summarising and checking statements are all typical of this mode of interviewing (Ivey, 1971).

Supportive. The intent here is to give approval, confirming or validating the worth, the value of the patient, as a human being. The therapist is not trying to promote change, although practical advice and information may be one of the ways of providing support. It usually helps a great deal if the therapist likes and respects the patient. Well-meaning attempts to provide support can founder if these qualities are lacking. Discussion of this, using audiotapes in combination with the interactogram, can lead to awareness of the ways that therapists give themselves away as far as their true feelings are concerned. This leads without difficulty into a discussion of counter-transference in subsequent seminars.

Use of interactogram II

Interactogram II records not only the therapist's intent but also links in the microskills being used to achieve this. Blow-by-blow analysis of material from a patient–doctor session, with intent in mind, shows that some of the therapist's expressed purpose in the interview is based on an evident plan in the therapist's mind. At other times the therapist appears to be reacting to material produced by the patient during the course of the session. Trainees were asked to think about this and record their observations on the form. Finally, they were asked to consider the effect on the patient in contentious cases; role-play was often helpful here. Again the criteria were pragmatic: "How well did the therapist achieve his aim?"; "How much did the patient benefit from this stage in the interview?".

During a short segment of an interview it is quite possible to have two or three different intents. For example, a general practitioner often includes several intents: to be informative, to support the patient, and to prescribe within a six-minute consultation. The major intent in a psychotherapeutic interview may be catalytic, with an underlying intent of support. The primary intent in a behaviour therapy interview could be prescriptive, with a preliminary confrontation.

Interactogram II is used in conjunction with interactogram I so that the students are able to discover what particular interventions they use to further their intents.

Interactogram III

Interactogram III (Fig. 4) is an analysis of the relationship process. This interactogram is designed to make the therapist aware that it is not only his or her microskills and intent which govern what happens in the relationship with the patient. There are other factors which play a part in any human interaction. We use the term 'macroskill' to mean those skills which are used throughout the entire interview and relate to the broader process of the interview. The third interactogram is divided into two major sections, the first of which is related to the hypothesis. This is considered a working assumption about the interview which the therapist uses to understand the basis for the interview or part of the interview. Junior doctors were encouraged to establish working hypotheses to help them understand the difficulties that they were tackling. Developing a hypothesis can be highly therapeutic to the patient; alternatively, it can provide the basis for a plan of action to be agreed between the patient and the therapist. A skilled therapist would be one who avoids making 'divine revelations', and instead provides a series of hypotheses: educated guesses or ideas which would be the basis for further discussion. For example:

(a) "From what you've told me in this first interview I can see that you have already found out that if you stick with your anxiety in a difficult situation, it starts to decrease after a short while. Now, I wonder if we could make this the basis for our plans to help overcome the phobias." (*Basis for behavioural work*)

(b) "I know your father died over two years ago but I wonder if in a sense you are still haunted by him." (*Invitation to look at possible psychological basis for symptoms*)

(c) "Over the past weeks we've been through a lot together and I think that you're already starting to wonder how you are going to manage without these regular weekly visits." (*Invitation to look at meaning of psychotherapeutic relationship*)

Hypotheses can be very broad or very narrow. A broad hypothesis might be the idea that the patient has a psychological problem, rather than a medical illness. A narrow hypothesis might be that the patient's depression is being projected on to him by his wife, who is actually depressed and with whose depression he is identifying. A hypothesis is not meant to be a revelation of profound truth but a basis for communication between the patient and the therapist. Essentially, it is meant to be an approximation that can be open to negotiation from either side. If it is not acceptable to the patient it could be discarded. There is a process of learning to develop hypotheses. This process begins with the ability to observe and listen to the patient. When feeling stuck in an interview, or wondering what to do next, attention to the patient can help the therapist to develop useful working patient-focused hypotheses. For example, "From the way you describe your previous treatment I guess you have really got little faith left in doctors", or "Your manner of talking makes me wonder if part of your problem lies in feeling that no-one can ever understand you."

The next stage in making a hypothesis lies in the ability to link what the patient is communicating with material from earlier in the sessions, or from previous sessions and past history; for example, "You know, we keep coming back to the worries that you have about what other people will be thinking of you, yet it is all guesswork because you never try to find out what they are really thinking."

From this the therapist may make suggestions of ways in which both he and the patient are behaving which is playing a central part in the interaction; for example, "You know, I think you find my manner irksome because you have never liked being told what

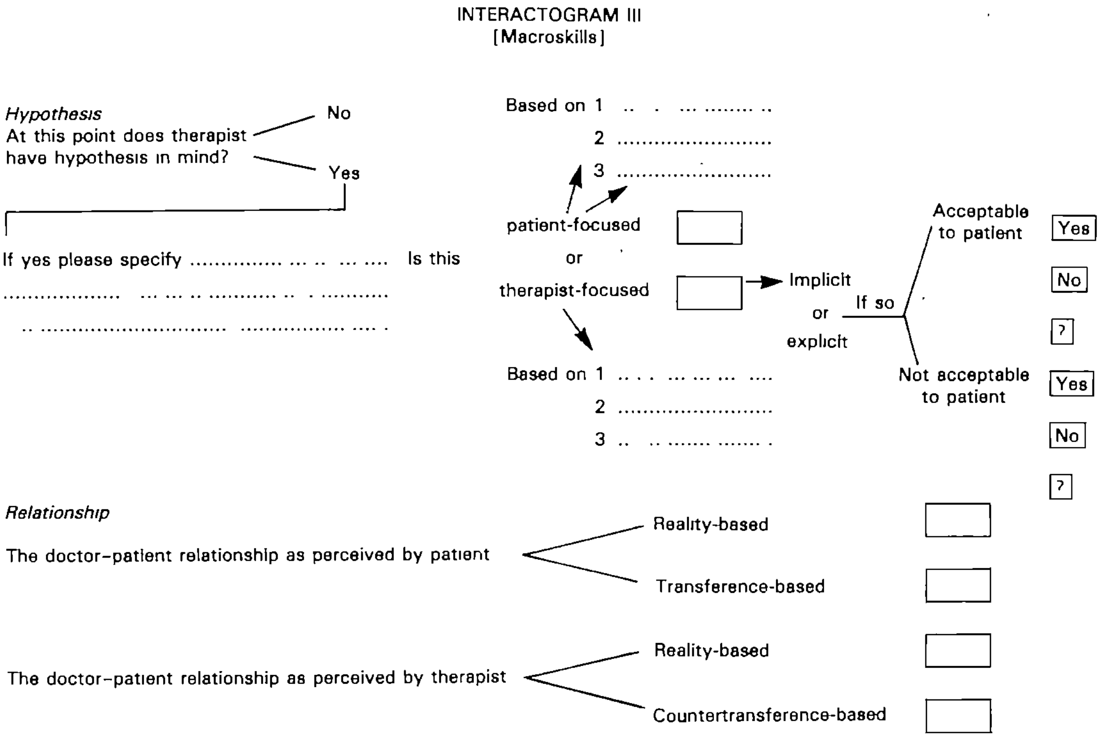

FIG. 4.

to do", or "We seem to be getting into difficulties and I think that may be because I have been encouraging you to think that I am going to take over responsibility for solving all your problems for you."

In the final stage the therapist starts to link what has been observed in the patient with his own theoretical ideas. These ideas come from watching other people working and from reading.

The number of different theories and the even greater number of individual variations of any one particular theory reflect the inventiveness of therapists in constructing hypotheses about the way in which their patients think, feel, and behave. If the therapist is more attentive to what is going on inside his own head than to what is going on inside the patient, the focus of the hypothesis will be therapist-based: "From my experience with other patients with anxiety like yours, I think . . .".

It is the ability and willingness to construct and use hypotheses which distinguish a psychotherapeutic style of interaction from plain interviewing. The skills can be most usefully acquired and developed by working with patients under supervision.

The second part of interactogram III focuses on the relationship between the doctor and the patient and introduces the concepts of transference and countertransference (Sandler *et al*, 1974). A number of factors influence the therapeutic relationship, both conscious and unconscious. The personality of the patient, the nature of the patient's difficulties, the personality of the therapist, the therapist's skill and experience, and the interaction between the patient and the therapist all play a part in defining the relationship. The doctor's ability to avoid imposing his own conflicts on the patient and distorting the material produced by the patient because of his own personality structure is an essential skill in the therapeutic encounter. Countertransference is both an essential part of therapy and an important phenomenon calling for self-awareness on the part of the doctor. We found that audiotapes of interviews, when specifically examined for evidence of countertransference, are a useful method of developing self-awareness.

Some aspects of patient and therapist are emphasised and other aspects minimised as the relationship develops. This process is unconscious and may be a re-enactment of previous patterns of relationship. The emphasis in the interactogram is that the relationship is a product of both participants, the doctor and the patient. What the doctor says and does and stands for are reacted to by the patient and vice versa, and an interpersonal feedback system quickly develops. This system must neither be ignored nor accepted blindly. The concept of transference is a special example of the way in which the feedback system operates when a patient reacts to the therapist as if he were representing some other person in that patient's past life, and the therapist is faced with what seem inexplicable responses to his words and actions. For example, one patient looked to a doctor as an authority on all subjects and depended on him to give advice on many areas of his life. This dependence and need for authority was a duplication of the nature of his relationship with his deceased father. He transferred his reactions from the relationship with his father to his relationship with the doctor.

By seeking information about past relationships, the therapist can identify the source of the transference. Without such knowledge the therapist can still be aware of the fact that the patient's reactions to the therapist are an unconscious re-establishment of some earlier relationship. The feedback system which develops between patient and doctor is a very important clue for the understanding of a patient's unconscious meaning. The reality-based perception of the doctor–patient relationship by both participants refers to the reason which brings together two participants in the interview: "We are here, you the doctor, I the patient, because I need help from you to deal with a particular problem or illness."

Conclusion

We believe that the use of these three interactograms is a helpful adjunct to psychotherapy supervision and provides a method for junior doctors to examine their audiotaped interviews with patients. This systematic approach, we believe, helps bridge the gap between traditional medical training and the psychotherapeutic approach which is an integral part of modern psychiatric practice.

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Effectiveness of Time-limited Therapy Carried Out by Trainees

Comparison of Two Methods

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Common neurotic and personality disorders account for widespread personal restriction and suffering. The need for effective, economically feasible treatment methods is widely acknowledged and the range of conditions successfully treated by available methods needs to be established. This study is concerned with two methods of brief psychotherapy; our aim was the essentially pragmatic one of investigating how far relatively long-term NHS out-patients with neurotic, personality, and interpersonal problems could be successfully and economically treated by the kind of therapists available in NHS settings. It is generally believed by psychoanalytically-oriented therapists that brief therapy requires high levels of training and experience; this is tested here, in that all the therapies were strictly time-limited, and none of the therapists had completed advanced training.

The research design differs from the currently favoured model of psychotherapy research in which an (ostensibly) homogeneous group of patients (usually suffering from depression) is exposed to a (supposedly) standardised therapeutic input. This model is somewhat remote from the realities of clinical practice and teaching, and ignores the extent to which groups of patients, however classified, differ importantly in respect of the issues addressed in therapy. Moreover, much current research employs a very restricted set of change measures. For example, most cognitive therapy research considers the mutual reinforcement of depressed thinking and depressed mood characteristic of the depressive cycle (Teasdale, 1983), but pays little attention to the life events and life strategies that predispose a person to become or remain depressed (Ryle, 1986). Psychotherapy and pharmacotherapy can both modify the depressive cycle, but psychotherapy, in most cases, seeks also to modify the patient's long-term interpersonal strategies and assumptions, his personality structure, and his self-attitudes, and measurement of these changes needs to be attempted.

In the study reported here, response to therapy was measured by using symptom inventories and also by measuring changes in assumptions and interpersonal strategies, using for this purpose a form of repertory grid technique reported in previous small-scale studies (Ryle, 1979*b*, 1980), and patients' ratings of individual targets. There is nothing innately illogical in this combination of nomothetic and ideographic measures, as Shapiro (1966) points out; exclusive reliance on the former in group studies can fail to detect the specific individual changes that are the aim of therapy.

Method

The patients' characteristics

The patients studied in the present research project were seen in the out-patient department of Guy's Hospital. Most were referred to the project by psychiatrists in the Guy's district; a small number were directly referred by GPs. The therapy and testing were completed by 48 patients, of whom 8 were aged under 25, 22 were aged 25–34 and 18 were aged 35 or over. Twenty-seven (56%) were receiving or had completed higher education or post-school professional training. These patients were classified retrospectively, from referral and research data, according to the Research Diagnostic Criteria (RDC). There were 37 (77%) who had had previous contact with psychiatric services, of whom 18 had received some form of psychotherapy. On referral, six patients were being treated with tranquillisers, seven were on tricyclics and one on MAOI antidepressant medication, and one was on a beta-blocker. The duration of the presenting complaint was as follows: 0–3 months, 7; 3–6 months, 0; 6–12 months, 9; 1–2 years, 10; 2–5 years, 12; over 5 years, 10. Thirteen patients completed pre-therapy assessment but did not complete the project. Of these, two did not accept the offer of therapy; two were taken out of the project and admitted for treatment, one for a manic and one for a depressive illness; one was taken on for long-term therapy and was subsequently admitted to hospital; four dropped out during therapy; and four failed to complete post-therapy testing.

The therapists and their supervision

Therapy was carried out by 15 different therapists: five trainee psychiatrists, with slight to substantial supervised therapy experience; six social workers, with varying amounts of casework experience but no formal therapy training; three nurses, one with a behavioural and two with a counselling training; and one trainee lay psychotherapist.

None had experience of time-limited therapy. On entering the project, a brief exposition of the two methods to be employed was offered, and it was recommended that two books describing the methods should be read (Mann & Goldman, 1982; Ryle, 1982); most attempted this. Weekly supervision in groups of 4–6 was the main learning method; this focused on general issues and on the specific features of the contrasting approaches.

The two therapeutic methods

All patients received 12 sessions of therapy, usually at weekly intervals. All were aware of the research component in the project. The two methods were labelled 'interpretive' therapy (INT) and 'cognitive-analytic' therapy (CAT).

The INT approach was based on the work of Mann (Mann & Goldman, 1982). This is a strictly interpretive, psychoanalytically based method, laying emphasis on the fact of termination and on the early formulation of a 'central issue'. This formulation aims to encapsulate the patient's 'chronically endured pain' in a description indicating the conflicts or assumptions related to it. Unlike the form of focusing employed by other brief therapists (e.g. Malan, 1976; Sifneous, 1972), this focus for treatment is shared with the patient, it being argued that accurate understanding conveyed in this way 'reaches through' the defences and engenders an early, intense involvement of the patient in the therapy. In this respect Mann's approach is closer to the CAT method than are other psychoanalytically-based methods.

The CAT approach (Ryle, 1979*a*, 1982) aims to integrate concepts and methods from psychoanalytic, cognitive, and behavioural schools. Focusing, or re-framing, is carried out with the patient, and an agreed, written list of target problems (TPs) and target problem procedures (TPPs) is formulated in the course of the first 3–4 sessions. A 'procedure' (Ryle, 1982) is the repeatedly used sequence of conscious and unconscious mental processes, including anticipation and feedback, which generates and monitors an individual's purposive acts and role behaviour. Neurotic procedures are characterised by being ineffective and resistant to revision. In formulating a target problem procedure (TPP), the aim is to identify the procedures causing and maintaining the patients' difficulties. The description of a procedure may refer to assumptions, beliefs, predictions about the efficacy and the consequences of the proposed actions, the choice of means, the action, and the evaluation of the outcome and consequences of the action. The 'Psychotherapy File' (see Ryle (1982) for an early version of this) was given to patients after the first or second interview. This includes instructions in self-monitoring of moods or symptoms, similar to those developed by Beck (1976), and descriptions of common TPPs, classified as Traps, Dilemmas and Snags, as in Ryle (1979*a*). A list of TPs and TPPs was agreed between patient and therapist at the fourth session. Patients were then encouraged to keep diaries recording their recurrence as a means of enhancing recognition and control, and they rated change on their TPs and TPPs on visual analogue scales at subsequent sessions. Therapists were free to employ a range of therapeutic methods, but all interventions were related to the issues summarised in the TPs and TPPs. Cognitive and behavioural methods were aimed at the processes described in these formulations, and interpretations of transference issues were made by reference to the TPPs.

TABLE I
Features of INT and CAT approaches

	INT	*CAT*
Early phase	Identify and name central issue	Patient self-monitors and reads therapy file
	Expect positive ± intense transference in most cases	Joint preparation of written list of TPs and TPPs
		Positive tranference plus active participation from patient
Middle phase	Ambivalent transference	Patient involved in learning to recognise TPPs (diary-keeping etc.)
	Interpret around central issue and termination	TPPs may manifest in transference
Termination phase	Transference recapitulation of earlier losses and disappointments	Continue TPP recognition; initiate change
		Termination transference issues linked with patient's new tools for self-help

Table I summarises the main features of the two treatment conditions. The psychodynamic issues interpreted in the INT approach were also considered in the CAT therapies, although their presentation and the therapeutic use made of them was influenced by the other activities included in the therapy.

No direct monitoring of the therapist interventions was carried out. Given that the formal instruction was minimal, it is likely that very variable levels of skill and compliance were displayed. However, the use of the psychotherapy file, instruction in self-monitoring and diary-keeping, the joint elaboration of written TPs and TPPs and the use of homework assignments were strictly confined to the CAT condition. With two exceptions, all therapists treated patients in both conditions, their first case being randomly allocated and subsequent cases being treated alternately in the two modes. A few more severe cases were allocated to the more experienced therapists; this allocation was made (by AR) blind to the mode to be employed.

Research design

Two hypotheses are being tested: (1) that patients will show significant improvement after 12 sessions of therapy carried out by relatively inexperienced trainees, and (2) (a null hypothesis) that no difference will be found between the two methods in respect of the type or degree of change. The basic design is summarised in Fig. 1. No waiting list or placebo control group was formed in view of the ethical and design problems involved in this group of patients, most of whom had long histories of symptoms and of treatment (see Shapiro, 1981). Given the brevity of the intervention, the long histories of most patients, and the specificity of the aims of therapy, it seems legitimate to attribute change, with respect to individually identified issues, to the treatment.

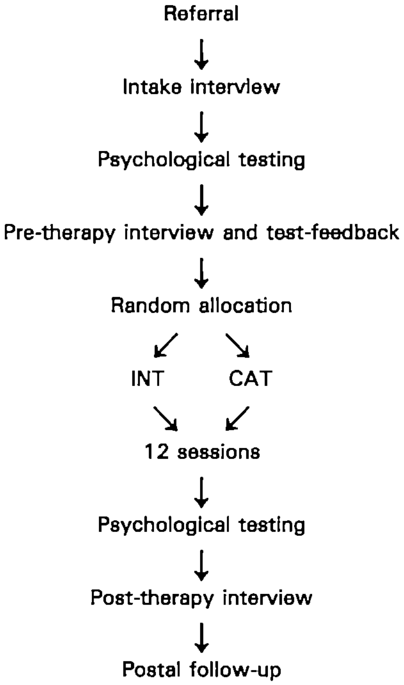

FIG. 1 Research design.

As regards the relative effectiveness of the INT and CAT methods, the use of the same therapists, the same testing and induction process, the same duration of treatment and random allocation provides a basis for attributing any differences found to differences in the therapeutic input. It should be noted that the two methods were supervised together; therapists' transfer of skills from one condition to the other would tend to diminish the contrast between the methods. It should also be noted that therapists were supervised by the originator of the CAT approach and this could be a source of bias.

In terms of psychotherapy research design, this project can be seen as an example of the 'dismantling' procedure (see Stiles *et al*, 1986) in which the CAT group received the full treatment and the INT group received this minus the cognitive, homework and written elements.

Procedure

Pre-therapy psychological testing

Testing was carried out by psychology students.

Symptom levels were assessed using three standard symptom questionnaires: the Beck Depression Inventory (BDI) (Beck *et al*, 1961), the 60-item General Health Questionnaire (GHQ) (Goldberg, 1972) and the Crown–Crisp Inventory (CCI) (Crown & Crisp, 1979).

Attitudes to self were measured from a semi-standard dyad grid of the form used in previous studies (Ryle, 1980). In this, patients rated their parents' relationship, and the role of self-to-other and of other-to-self in eight relationships (including with parents) and their relationships with themselves, on ten provided and six elicited constructs. The grids were analysed on the Ingrid program (Slater, 1972). A full account of this use of grid technique can be found in Ryle (1979*b*, 1980). Two measures of self-attitudes were derived as follows: the Positive Self Attitude Score (PSA) is the sum of the angular distances of the element self-to-self from the constructs 'looks after', 'respects' and 'forgives' (high score = low positive attitude); the Negative Self Attitude Score (NSA) is similarly derived from the constructs 'guilty to', 'blames' and 'is cross with' (high score = low negative attitude).

Specific cognitive change was assessed as follows. On the basis of the pre-therapy grid, predictions of desirable change were recorded for certain construct correlations. Some of an individual's assumptions about relationships are indicated by high or low construct correlations. Population norms for the angular distances between the constructs 'looks after', 'is dependent on', 'controls', 'gives in to' and 'is cross with' are available (Ryle, 1981). Where these values differed in a given patient's grid by more than one standard deviation from the norm, and where (as was usual) clinical data and discussion with the patient in the pre-therapy interview (see below) indicated that this was related to interpersonal difficulties, a prediction was recorded of the desirable direction of change. Other construct correlations, linked with the patient's specific problems and sometimes including the elicited constructs, were also, in some cases, made the subject of directional predictions. The grid prediction score (GPS) (see below) was derived in this way from the analysis of a grid of self-to-other elements only, in view of the fact that values in the self–other and other–self grids for these angular distances can differ considerably.

Pre-therapy interview

After initial testing, patients were interviewed (by AR). On the basis of the previous history and the assessment interview, and the discussion of the test results with the patient, a list of Research TPs and TPPs was drawn up with the patient, describing the problems and procedures which therapy was concerned to modify. This list was not identical with that drawn up by patient and therapist over the first four sessions in patients treated in the CAT condition, but obviously there was often close similarity with that list and with the 'central issue' identified in the INT condition patients.

Post-therapy psychological testing and interviews

After therapy, patients completed the same test battery and also the Therapy Experience Questionnaire (TEQ) (see Appendix 1). They were then interviewed (by AR) and they rated how far they felt they had resolved the issues identified at the pre-therapy assessment on the agreed list of Research TPs and TPPs. Rating was the change in millimetres on a ±20 mm visual analogue scale, of which the midpoint represented the level of difficulty at presentation; improvement scoring positively, deterioration negatively. From this was derived a mean TP and mean TPP score. The Grid Prediction Score (GPS) was derived by comparing the pre- and post-therapy grid values for the identified construct angular distances, counting predicted change as positive and the opposite as negative, and taking the mean of all predictions recorded.

Follow-up

Some months after completion, patients were contacted by post and were asked to repeat the tests and to indicate whether any significant life changes had occurred and whether they felt further treatment would be helpful. Unfortunately, for a number of reasons, the interval to follow-up varied from a minimum of 8 months to a maximum of 22 months.

Summary of research measures

The list of data available and coded on all patients at the start of therapy is given in Appendix 2.

Results

Comparability of the INT and CAT samples

Therapy and post-therapy testing was completed by 30 CAT and 18 INT patients. This discrepancy in numbers is partly a reflection of the higher attrition rate in patients in the INT sample (7 of the 11 non-completors who had started therapy were INT patients), but is largely due to chance in the balance of methods used by therapists seeing odd numbers of patients. The attrition group had higher mean initial symptom scores than did patients completing the project (BDI 24.2, GHQ 29.0, CCI 56; see Table IV for the lower values of the treated patients in the CAT and INT samples). The distribution by diagnosis in the INT and CAT samples (Table II) is relatively similar; in view of the small numbers in most of the categories, no breakdown by diagnosis is offered in terms of outcome. Despite the random allocation, indications of greater severity were more frequent in the CAT sample, as follows.

TABLE II
Diagnosis of CAT and INT samples

Diagnostic category on RDC	*CAT*[1]	*INT*	*Total*
1. Minor Depressive Disorder	12	3	15
2. Minor Depressive Disorder with significant Anxiety	11	8	19
3. Intermittent Depressive Disorder	0	1	1
4. Recurrent Unipolar Major Depressive Disorder	1	0	1
5. Generalised Anxiety Disorder	3	1	4
6. Generalised Anxiety Disorder secondary to Phobic Disorder	0	1	1
7. Obsessive Compulsive Disorder (one with Minor Depressive Disorder, one with generalised Anxiety)	2	1	3
8. Other Psychiatric Disorder (Bulimia Nervosa)	2	0	2
9. Alcoholism, plus Minor Depressive Disorder with Anxiety	1	0	1
10. Never Mentally Ill	1	3	4

1. CAT sample includes three cases with two diagnoses: i.e. Minor Depressive Disorder and Obsessive–Compulsive Disorder; Generalised Anxiety Disorder and Bulimia Nervosa; Minor Depressive Disorder and Bulimia Nervosa.

Medication. On entry to the project, four of the INT patients were on minor tranquillisers. Of the CAT sample, two were on minor tranquillisers, one was on a beta-blocker, one was on a MAOI and seven were on tricyclics. Treatment aims included reducing or ending tranquilliser use; antidepressant medication was usually continued. No patients were started on medication after referral.

Age. The proportion of patients aged over 35 was higher in the CAT sample.

Obsessionality. All but one of the 13 patients scoring 11 plus on the CCI obsessionality scale were in the CAT group ($\chi^2 = 6.33$, $P < 0.05$).

Scores. Mean BDI and CCI scores were higher in the CAT sample, but GHQ scores were marginally lower (see Table IV).

Patient characteristics associated with outcome

In order to investigate the influence of age, sex, education, duration of symptoms and initial inventory scores on outcome, patients were divided into response and non-response categories on five outcome measures. The criteria used were such that approximately one patient in three was classed as non-responding. The criteria of response were as follows.

Symptom response was measured by dividing initial scores into four categories on both the BDI and the GHQ (0–13, 14–20, 21–26 and 27 plus); failure to go down to a lower category at post-testing (unless initially in the lowest), on either inventory, was classified as non-response.

TABLE III

Relations between initial patient characteristics and outcome measures significant at 10% or better

Patient characteristics and baseline scores	*Outcome measures*	*Association*	χ^2	P<
Age 35+	PSA improvement	–	4.67	0.05
	NSA improvement	–	5.61	0.05
	Symptom response	–	3.06	0.1
Symptoms over 5 years	PSA improvement	+	3.57	0.1
Initial BDI 20+	PSA improvement	–	3.36	0.1
	NSA improvement	–	2.86	0.1
Initial GHQ 34+	Target ratings	–	2.73	0.1
	PSA improvement	–	7.99	0.01
Initial CCI–T (total) 45+	Symptom response	–	4.75	0.05
Initial CCI–O (obsessionality) 11+	Symptom response	–	3.37	0.1

Target rating improvement response was defined as a score of +5 or more on both TP and TPP ratings.

Grid Prediction Score response was defined as a GPS of plus 20 or more.

Positive Self Attitudes response was defined as a PSA score reduction of 20 or more (indicating more positive self-attitudes).

Negative Self Attitudes response was defined as a NSA score increase (indicating less negative self-attitudes).

The associations between patient characteristics and response to the five measures were tested using 2×2 χ^2 tests; associations significant at 10% or better are listed in Table III. Neither sex nor education were related to outcome on any measure at this level of significance. The seven patients with histories of less than 6 months showed similar changes to those with longer histories; the greater improvement on one measure of long term patients is surprising. The other associations are expected; older patients and iller patients showed less change.

Relation between outcome measures

Rank order correlations between the TP, TPP and GPS scores and changes in the PSA and NSA showed only one association significant at the 5% level: that between mean research TP and TPP ratings ($r=0.84$). A sum score was derived from the different response/non-response categories using the criteria listed above. Scoring 1 for response and 0 for non-response, patients could achieve a 'sum score' of 0–5 from symptom response, target rating response, GPS, PSA and NSA scores. It seems reasonable to assume that patients showing improvement in more of these independent measures will have benefitted more from therapy. This sum score was significantly correlated with high TEQ scores indicating a positive evaluation of therapy by the patient ($\chi^2=7.9$, $P<0.01$).

Comparison of change measures in INT and CAT patients

Nomothetic measures

Pre- and post-therapy mean scores for five measures are given in Table IV. Mean scores on the BDI and GHQ dropped significantly in both INT and CAT samples. CCI total score dropped significantly in the CAT sample. Patients in the CAT group showed significant improvement in PSA scores. Improvements in NSA (reflected in larger scores) did not reach significance in either group.

TABLE IV

Pre- and post-therapy mean scores for CAT and INT samples on five nomothetic scores

CAT (n=30)	*Pre-therapy mean*	*Post-therapy mean*	*Significance (Wilcoxon)*
BDI	17.0	12.4	$P<0.01$
GHQ	22.5	13.7	$P<0.02$
CCI	45	33	$P<0.01$
PSA	325	276	$P<0.01$
NSA	190	214	NS
INT (n=18)			
BDI	10.8	6.7	$P<0.01$
GHQ	24.7	9.3	$P<0.01$
CCI	34	31	NS
PSA	288	272	NS
NSA	186	192	NS

Ideographic measures of change

Mean TP and TPP ratings and GP scores are given in Table V for three groups, namely INT and CAT patients, and a sample of CAT patients matched with the INT group on initial BDI and CCI–O (obsessionality) scores. TP and

TABLE V

Mean scores on three ideographic change measures for INT, CAT, and matched CAT samples

Sample	*INT (n=18)*	*CAT (n=30)*	*Matched CAT (n=17)*	*Significance of difference between INT and CAT*
TP	10.6	8.7	10.6	$P<0.01$
TPP	11.1	9.7	11.1	$P<0.01$
GPS	23.6	33.0	33.2	$P<0.01$

TPP ratings are significantly higher in the INT than in the whole CAT sample, but no different from the scores found in the matched CAT sample. GPS scores are the same in the matched and unmatched CAT samples, being significantly higher than in the INT sample.

Comparative effect of INT and CAT methods

It has been shown, starting from different initial mean score levels, that both INT and CAT samples showed significant reductions on BDI and GHQ scores, and the CAT sample also showed significant reductions in CCI-T (total) scores. When matched on initial BDI and CCI-O (obsessionality) scores, the two approaches had the same effect on TP and TPP ratings; the more depressed and obsessional CAT patients, excluded in this matching process, did less well. The mean GPS score was higher for CAT sample patients and was independent of the initial symptom level. The effect size of the CAT over the INT sample in improving self-attitudes was calculated using the formula proposed by Smith & Glass (1977), whereby effect size equals difference in mean change between the samples divided by the standard deviation of the INT sample. The value of this was 1.0 in respect of changes in PSA scores, but when patients with initially high scores (336+) were omitted, to give a sample matched on initial PSA (mean 290), the figure became 0.53. For the NSA, where pre-therapy levels were similar in CAT and INT patients, the effect size was 0.38. The standard deviations of construct angular distances are in the range 17–25 (Ryle, 1981); on this basis the effect size in respect of the GPS approaches 0.5.

The mean sum scores (see above) were 3.7 for the CAT sample and 3.1 for the INT sample ($t=4.3$, $P<0.02$). This difference reflects similar effectiveness in symptom reduction and target problem ratings, and a greater effect of the CAT approach in modifying specific cognitive features expressing individual assumptions about relationships and in altering attitudes towards the self.

Follow-up

Postal follow-up was attempted on all patients, but satisfactory returns were received from only 25 (62%), due to address changes and some refusals. The time from end of therapy to follow-up was variable (INT sample range 6–22 months, mean 14; CAT sample range 8–18 months, mean 12). The mean score values on seven measures of the whole sample post-therapy, and of the follow-up sample post-therapy and at follow-up, are given in Table VI. The follow-up sample's mean scores post-therapy were virtually the same as the scores of the whole sample. A small deterioration in mean scores between post-therapy and follow-up testing was recorded on two measures in both the INT sample (BDI and GHQ) and the CAT sample (GHQ and NSA); improvements in mean scores, of modest size, were recorded on the remaining measures. The two worst outcomes, showing improvement from presentation to follow-up on only one measure, were in men aged over 35 with initial CCI–O scores of 11 or more.

Discussion

In the use of individualised goal and outcome measures, in the degree to which treatments, therapists and patients are described, and in the use

TABLE VI

Post-therapy and follow-up mean scores of INT and CAT samples

	n	*BDI*	*GHQ*	*PSA*	*NSA*	*GPS*	*TP*	*TPP*
INT								
Whole sample post-therapy	18	6.7	9.3	272	192	24	11	11
Follow-up sample post-therapy	11	5.6	9.3	269	203	28	12	13
Follow-up sample at follow-up	11	5.5	9.8	259	235	32	14	15
CAT								
Whole sample post-therapy	30	12.4	13.7	276	214	33	9	10
Follow-up sample post-therapy	14	12.6	12.5	273	218	31	8	9
Follow-up sample at follow-up	14	10.4	17.8	240	201	34	9	10

of diverse outcome measures, the design of this study is in accordance with the recommendations of Fiske *et al* (1970). It would, however, have been strengthened by the inclusion of independent assessments of change.

The INT and CAT methods of treatment chosen for comparison were intentionally similar in important respects, both being characterised by brevity and by the sharing of the focus with the patient, both being preceded by the same assessment process and the treatments being carried out by the same therapists. The therapists' prior experience and outlook were fairly equally spread along the active versus interpretive spectrum. Both INT and CAT approaches involved them in the unfamiliar task of producing, after 3–4 sessions, formulations encapsulating the issues to be addressed by the therapy. This task, and the research component in the project, probably served to heighten therapist involvement. Patients in both conditions had the same experience of psychological testing and received feedback on these procedures; it is likely that this (largely cognitive) input had some therapeutic effect. The CAT approach differed, firstly, in the use of written material; secondly, in the form in which the issues addressed in the therapy were formulated; thirdly, in the involvement of patients in serial ratings of change on the defined issues; and fourthly, in the inclusion of cognitive/behavioural tasks in the therapy, notably self-monitoring of identified TPPs. These additional factors could be seen to maximise the pressure on patients to accept responsibility for their defined problems (Försterling, 1980; Keller, 1984). While therapists avoided direct advice, they were more active in the CAT mode in offering concepts and setting cognitive behavioural tasks designed to help the patient initiate change, and in these respects offered more of the factors associated with positive outcome according to Murphy *et al* (1984).

Unfortunately the random allocation of patients between the two modes produced poorly matched samples, the bias being in the direction of the CAT sample being more severe. The choice of outcome measures was determined by the conviction that therapy should affect both the presenting symptoms and difficulties complained of by the patient, and the underlying 'dynamic factors' reflected in these complaints. While measures of these two sorts of change may co-vary to a degree (Mintz, 1981), at least in the short term, it was considered that the underlying assumptions and strategies about self and relationships were the most appropriate target for therapeutic change. Attempts by psychoanalytic researchers to assess these factors clinically have been of low reliability (DeWitt *et al*, 1983). In the present study the repertory grid has been used to yield measures related to the 'dynamic' issues, in line with the general work reviewed by Ryle (1976), and the specific applications of this grid method to previous outcome research reported by Ryle (1979*b*, 1980). The measurement of positive and negative self-attitudes, achieved by embedding the element self-to-self among the range of self-to-other and other-to-self elements in the dyad grid, provides indications of change in self-attitudes. The GPS represents a specific ideographic measure of the patient's changing of identified, non-adaptive, idiosyncratic assumptions, in most cases concerning the relationship between control, looking after, dependency, anger, and giving in. The psychological processes indicated by these identified construct correlations, about which predictions of change were made, can be seen to be those described as dilemmas (Ryle, 1979*a*, 1982), and, in object–relations terms, as related to splitting and projective identification.

The evidence for the overall efficacy of these two forms of brief therapy rests on the comparison of the 'before' and 'after' measures. Given the relatively long duration of presenting problems in most cases and the individualised nature of some of the outcome measures, it seems reasonable to claim that both approaches were effective in a substantial proportion of the cases treated. Significant changes in many group mean scores occurred between pre- and post-therapy testing; these were maintained but did not show further significant change in the period between post-therapy testing and follow-up, a period which was longer than the duration of therapy itself. As regards the relative impact of the INT and CAT approaches, the evidence suggests that in cases of equivalent severity the two approaches have an equal impact on symptoms and self-report ratings, while in all cases, independent of severity, the CAT approach had a greater effect on some grid-derived measures of cognitive change. As therapists were supervised in both modes by AR, the possibility of biased supervision must be acknowledged. However, the supervision of dynamic issues was common to both conditions. It is more likely that any advantage of the CAT approach was minimised by the design, in that therapists working in the INT condition may have been influenced by their CAT training, and patients in the INT condition had experienced cognitive input during their test completion and pre-therapy interview.

Luborsky *et al* (1980) concluded that prediction of outcome of psychotherapy is difficult; it is rare to demonstrate associations between pre-therapy measures and outcome strong enough to be of any clinical value. This is true of the present study. It is, however, noteworthy that neither educational level nor duration of symptoms were related to therapeutic response. That some patients of greater age, higher obsessionality and higher symptom scores at outset did less well is worthy of note, and suggests that the response to treatment of patients with these features should be carefully monitored; it is possible that longer or different intervention may be indicated.

As a result of the research reported here, the CAT approach is being further evaluated, but with some changes in practice, notably the following.

Firstly, therapists attend a two-day training workshop. This lays particular emphasis on the process of reformulation as it is our impression that, if this is adequately carried out, therapists of relatively little experience can combine therapeutic methods effectively, and can make use of transference when appropriate, within the terms outlined by the target problem procedures.

Secondly, it is considered that patients showing major depressive symptoms should be treated pharmacologically before psychotherapy is commenced, and failure of less severe depression to recede after the first few sessions is also taken as an indication for medication (see DiMascio *et al*, 1979). Thirdly, the meaning of common psychosomatic and mood symptoms can often be decoded early in therapy with the use of self-monitoring, but some patients with more structured disorders (e.g. severe eating disorders, obsessive–compulsive disorders) may require a more energetic treatment of the symptom before the underlying issues can be addressed in therapy.

Fourthly, while the approach is usually adequate for crisis intervention or for patients with common neurotic disorders, patients with poorly integrated personalities will usually need more than a brief intervention. However, even with this group of patients, a time-limited approach has many advantages as a first therapy, providing an opportunity to see how much use patients can make of which aspects of therapy, while guarding against the risk of regression. In such cases the initial 'package' of therapy will not attempt to address all the identified problem procedures.

We believe that the CAT approach, with its demand on both therapist and patient for early conceptual work in describing accurately the repeatedly used, ineffective procedures underlying the patient's difficulties, is an effective therapeutic method and one which provides a suitable framework for trainee psychotherapists and a good basis for training general psychiatrists in psychotherapy. Further research will aim to identify with increasing accuracy the features which can be identified at different stages of the therapy that may indicate the possibility of briefer, or the need for more extended, or different, forms of treatment.

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Appendix 1
Therapy Experience Questionnaire

The Therapy Experience Questionnaire is reproduced in Table VII. There are four four-item subscales, items being presented in turn

TABLE VII
Therapy Experience Questionnaire

1. My therapist is/was really friendly and informal	+ +	+	±	–	– –
2. I am convinced that my therapist respects/respected me as a person	+ +	+	±	–	– –
3. I hardly ever think about myself in ways I learned in therapy	+ +	+	±	–	– –
4. Therapy certainly stirs/stirred up my feelings	+ +	+	±	–	– –
5. My therapist's general attitude is/was rather cold and distant	+ +	+	±	–	– –
6. I feel my therapist is/was genuinely interested in helping me	+ +	+	±	–	– –
7. Therapy has certainly made me think	+ +	+	±	–	– –
8. I have not felt emotionally involved in my therapy	+ +	+	±	–	– –
9. I have felt there is/was really a good deal of warmth in the way my therapist talks/talked with me	+ +	+	±	–	– –
10. I never really feel/felt fully accepted by my therapist	+ +	+	±	–	– –
11. I seldom consciously refer to what I have learned in therapy	+ +	+	±	–	– –
12. I do not manage my feelings any better as a result of therapy	+ +	+	±	–	– –
13. The tone of my therapist's statements tend/tended to be rather cold	+ +	+	±	–	– –
14. I never fully trust/trusted my therapist's integrity as a person	+ +	+	±	–	– –
15. Therapy has given me some clear new understandings about myself	+ +	+	±	–	– –
16. Therapy has helped me get in touch with my feelings	+ +	+	±	–	– –

+ + Definitely true; + True; ± May or may not be true; – Not true; – – Definitely not true.

for therapist warmth (W), therapist respect (R), therapy as a learning experience (L) and therapy as a feeling experience (F). The first two subscales were modified from Strupp *et al* (1969). The L and F scales were selected, after piloting, on face value, with the aim of exploring whether the more 'cognitive' CAT approach was sensed by patients as involving less feeling and/or more learning. Each item scores 1–5. Mean values on the INT and CAT samples are shown in Table VIII.

TABLE VIII
Mean values on the INT and CAT samples

Sample	*n*	*W*	*R*	*L*	*F*	*Total*
INT	18	16.6	17.2	16.5	15.8	66.1
CAT	28	17.5	17.7	16.5	16.2	67.9

Appendix 2
Summary of research measures

1. Age: (classified as <24, 25–34, 35+).
2. Sex.
3. Education (EDN-coded tertiary education or equivalent training, or not).
4. Duration of presenting problem (DTP-coded as under 3 months, 3–6 months, 6–12 months, 1–2 years, 2–5 years, over 5 years).
5. Score on Beck Depression Inventory (BDI).
6. Score on General Health Questionnaire (GHQ).
7. Score on Crown–Crisp Inventory (total – CCI-T).
8. Score on Crown–Crisp Inventory (obsessionality subscale – CCI-O).
9. Positive Self-attitude Score (PSA).
10. Negative Self-attitude Score (NSA).

Measures 5–10 were repeated at the post-therapy testing, at which point the following were available in addition:

11. Grid Prediction Score (GPS).
12. Research Target Problem rating (mean – TP).
13. Research Target Problem Procedure rating (mean – TPP).
14. Therapy Experience Questionnaire sum score (TEQ).

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The DSM–III Concepts of Schizophrenic Disorder and Schizophreniform Disorder

A Clinical and Prognostic Evaluation

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Consecutive new patients presenting at a Nigerian psychiatric unit over a 14-month period and fulfilling the DSM–III criteria for schizophreniform disorder, (active) schizophrenia and residual schizophrenia were studied. There were no differences between the three groups with respect to background social data. The schizophreniform patients had a lower incidence of previous psychotic episodes. There were no differences between the first two groups with respect to the distribution of PSE symptoms and signs, Schneider's First Rank Symptoms, or severity of psychopathology. Initial clinical outcome, and longer-term clinical and social outcome, assessed 25–38 months after index presentation, was best in the schizophreniform patients and worst in the patients with residual schizophrenia. The findings are consistent with schizophreniform disorder lying on a spectrum of schizophrenic disorders. They also highlight inadequacies in mental health services in Nigeria and other developing countries.

The third edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM–III; American Psychiatric Association, 1980) makes a distinction between schizophrenic disorder and schizophreniform disorder; a duration of at least six months is required for the former, and between two weeks and six months for the latter. This distinction highlights the controversy over the possible existence of a separate disorder of short duration which, although manifesting with schizophrenic symptoms, carries a better short-term as well as long-term prognosis than schizophrenia *per se* (Langfeldt, 1937; Slater & Roth, 1969; Smythies, 1973; Spitzer *et al*, 1978; Fox, 1981). An alternative approach would be to view such a disorder as a form of schizophrenia which because of its relatively acute nature and earlier presentation would respond more rapidly and more favourably to treatment. The DSM–III categorisation does not differentiate between a schizophrenic patient who has been ill only for a short period up to the time of diagnosis and 'schizophreniform disorder' (assuming the latter actually exists). The above consideration might appear to make the term schizophreniform disorder meaningless in terms of therapeutic implications.

The present paper describes a study of consecutive new patients presenting at a psychiatric unit over a 14-month period, in whom the diagnosis of schizophrenic disorder (active or residual) or schizophreniform disorder was made. The three groups were compared in an attempt to detect any differences, apart from duration of illness, between them.

Method

Consecutive new patients presenting at the psychiatric unit of the Wesley Guild Hospital, Ilesa, Oyo State, Nigeria between 1 November 1982 and 31 December 1983, and in whom a diagnosis of schizophrenic disorder or schizophreniform disorder was made according to DSM–III criteria, were studied. The schizophrenic patients were further categorised into those with active symptoms and those with residual schizophrenia, according to the DSM–III criteria.

For each patient, a psychiatric case-history was taken and a full mental state and physical examination carried out. Background data were collected on a specially prepared *pro forma*. The Present State Examination (PSE; Wing *et al*, 1974) and a World Health Organization modification of the Brief Psychiatric Rating Scale (BPRS; Overall & Gorham, 1962) were also administered. The modified BPRS contains three items additional to the original 16 – for pressure of speech, excitement, and disorientation. Where the patient was too disturbed for the PSE to be administered fully on presentation, sections 18–20 for observed behaviour, affect, and speech were administered and the other sections administered later, in all cases within one week. Specific note was made of the presence of those First Rank Symptoms (Schneider, 1959) which are not clearly differentiated in the PSE. Other data were also collected over a series of interviews in some cases. The first author was the consultant responsible for the management of all the patients studied.

Between 25 and 38 months after index presentation, the patients were traced and re-examined. Although a

TABLE I

Background data in the three groups of patients[1]

	Schizophreniform disorders (n = 34)	*Active schizophrenia (n = 66)*	*Residual schizophrenia (n = 16)*
Age at presentation (mean ± s.d.): years	27.9 ± 8.4	28.4 ± 9.4	33.6 ± 11.0
Age at first episode (mean ± s.d.): years	27.6 ± 7.6	25.6 ± 8.0	23.4 ± 7.0
Patients with previous episodes:* number and proportion of group	4 (0.1)	31 (0.5)	4 (0.25)
Male patients: number and proportion of group	16 (0.5)	40 (0.6)	9 (0.6)
Female patients: number and proportion of group	18 (0.5)	26 (0.4)	7 (0.4)
Duration of illness (mean ± s.d.):** months	1.7 ± 1.2	23.2 ± 37.2	67.0 ± 71.2

1. There were no apparent differences between the three groups with regard to occupational group (classified according to Boroffka & Olatawura, 1976), marital status, religion or educational status.

*$\chi^2 = 13.03$, $P<0.01$.

**$F = 15.77$, $P<0.01$ (ANOVA).

TABLE II

Frequency of occurrence of commonest PSE symptoms and signs in the patients with active schizophrenic symptoms and schizophreniform disorders

PSE item	*Sign/symptom*	*Schizophreniform disorder (n = 34)*		*Active schizophrenia (n = 66)*	
		Number of patients	*Proportion of group*	*Number of patients*	*Proportion of group*
107[1]	Social impairment due to psychotic condition	32	0.9	64	1.0
104[2]	(Lack of) insight into psychotic condition	29	0.9	51	0.8
74[3]	Delusions of persecution	24 (5)	0.7 (0.1)	43 (6)	0.7 (0.1)
62	Voices commenting on thoughts/actions and voices discussing/arguing about subject	23	0.7	38	0.6
63	Voices speaking to subject (non-affective)	20	0.6	32	0.5
13	Autonomic anxiety due to delusions, etc.	19	0.6	24	0.4
1[4]	Adverse subjective evaluation of physical health	18	0.5	22	0.3
35	Delayed sleep	18	0.5	30	0.5
8	Restlessness	14	0.4	18	0.3
23	Depressed mood	12	0.4	20	0.3
40	Irritability	9	0.3	16	0.2
81[3]	Delusions of alien forces penetrating the body	10 (1)	0.3 (0.0)	20 (2)	0.3 (0.0)
2	Presence of physical illness or handicap	10	0.3	14	0.2
71[3]	Delusions of control	8 (1)	0.2 (0.0)	15 (2)	0.2 (0.0)
79[3]	Delusional explanations in terms of paranormal phenomena	7 (0)	0.2 (0)	16 (0)	0.2 (0)
108	Self-neglect	7	0.2	15	0.2
6	Tiredness or exhaustion	5	0.2	16	0.2
60	Non-verbal auditory hallucinations	3	0.1	16	0.2

1. Refers to scores of 2 (moderate interference with function) or 3 (severe interference with function).
2. Refers to scores of 2 (partial insight) or 3 (no insight).
3. Figures refer to full delusions; partial delusions in brackets.
4. Refers to scores of 2 (unwell but not seriously incapacitated) or 3 (feels seriously incapacitated).

Note. None of the differences was statistically significant (χ^2 test).

proportion of them were currently attending the psychiatric unit at the time, the majority had to be contacted in the community. This involved journeys to 21 different towns, the furthest being 110 km from the psychiatric unit. At the follow-up interview, information was obtained both from the patient and, where possible, from the relatives. In the nine cases where the patient was dead, information was obtained from relatives. Such information was supplemented from the patients' case files. A specially designed *pro forma* for recording details of occupation, marital, family, and social data as well as clinical course was used. In addition, a full mental state and physical examination was carried out. The PSE and modified BPRS were administered and the Clinical Global Impression (McGlasham, 1973) was recorded.

All interviews and evaluations were carried out by the first author, both at presentation and follow-up. No other

TABLE III

Frequency of occurrence of Schneider's First Rank Symptoms in the patients with active schizophrenic symptoms and schizophreniform disorder

Symptom	*Schizophreniform disorder (n=34)*		*Active schizophrenia (n=66)*	
	Number of patients	*Proportion of group*	*Number of patients*	*Proportion of group*
Voices arguing/discussing about subject	23	0.7	34	0.5
'Made' volitional acts[1]	7	0.2	13	0.3
External influences playing on the body	3	0.1	8	0.1
Voices commenting on thoughts/actions	2	0.1	5	0.1
Thought insertion	0	0	5	0.1
Thought extraction	1	0.0	3	0.0
Thought echo	0	0	3	0.0
Thought broadcasting	0	0	2	0.0
Delusional perception	1	0.0	2	0.0
'Made impulses'[1]	1	0.0	2	0.0
Total number with FRS	28	0.8	40	0.6

1. 'Made' affects were not described by any patient.
Note. None of the differences was statistically significant (χ^2 test).

adequately skilled and trained personnel were available in the psychiatric unit; a reliability check on the DSM-III diagnoses was therefore not possible.

Results

There were 66 patients with active schizophrenic disorder, 16 with residual schizophrenia, and 34 with schizophreniform disorder. Background data (Table I) indicate that in addition to the significantly greater duration of illness among the schizophrenic patients ($P<0.01$) there was also a significantly greater number of previous episodes of the disorder among the two schizophrenic groups ($P<0.01$).

Clinical features

The frequencies of the most common PSE symptoms and signs in the schizophreniform and active schizophrenic groups are given in Table II. There were no apparent differences between the two groups. There was a somewhat higher frequency of occurrence of Schneider's First Rank Symptoms in the schizophreniform group (not statistically significant) (Table III). In both groups the experience of voices arguing about or discussing the subject in the third person was the most common, followed by 'made' volitional acts.

The clinical severity of the disorder, as estimated by modified BPRS scores, was similar in both groups with active symptoms, mean score ± s.d. being 30.0 ± 10.1 in the active schizophrenic group and 31.6 ± 8.9 in the schizophreniform group. There were no differences in the occurrence of a history of drug abuse (13 in the active schizophrenic, four in the residual schizophrenic, and five in the schizophreniform group). Only four active schizophrenics, two residual schizophrenics and three schizophreniform patients had a positive family history of mental disorder.

Initial clinical outcome

The initial clinical outcome, as determined by the interval between presentation and complete disappearance of symptoms, was much better in the schizophreniform group and worse in the residual schizophrenics (Table IV).

Outcome at 25–38 months

Only a small proportion of patients attended follow-up clinic regularly. The majority defaulted within six months (Table IV). In a number of cases, patients who had defaulted resumed attendance, usually after relapse.

Out of the original cohort of 116 patients, the authors were successful in tracing 94 cases (84%), made up of 31 (proportion 0.9) of the schizophreniform group, 51 (proportion 0.8) of the active schizophrenic group and 12 (three-quarters) of the residual schizophrenic group, at between 25 and 38 months after index presentation. Mean interval ± s.d. between initial presentation and follow-up interview was 31.8 ± 3.9 months in the schizophreniform group, 31.9 ± 3.9 months in the active schizophrenic group and 32.2 ± 4.1 months in the residual schizophrenic group (ANOVA, $F=0.025$, NS). Proportions 0.4 of the schizophreniform group, 0.7 of the active schizophrenic group, and 0.5 of the residual schizophrenic group had further episodes of active functional psychosis, usually of schizophrenic type (Table IV). These differences were statistically significant for further active schizophrenic episodes but not for further active psychotic episodes overall. (Some episodes diagnosed as 'atypical psychosis' were probably schizophrenic in nature, but this could not be ascertained because of inadequate information.) In addition, six of the active schizophrenic group and eight of the residual group continued to suffer from residual schizophrenic symptoms. There were no significant differences in the incidence of physical problems (Table IV).

TABLE IV

Clinical outcome over 25–38 months after index presentation: except where indicated, figures refer to the 94 patients contacted at 25–38 months

	Number of patients			*Statistical analysis*
	Schizophreniform group	*Active schizophrenic group*	*Residual schizophrenic group*	
Time taken for initial full clinical recovery (all 116 patients)	*(n = 34)*	*(n = 66)*	*(n = 16)*	
One month or less	18	25	2	One month or less *v*. over one month: $\chi^2 = 6.51$, $P < 0.05$
1+ to 3 months	13	20	2	
3+ to 6 months	1	5	1	
Did not recover[1]	1[2]	11	9	
Not known	1	5	2	
Further episodes of mental disorder after index, presentation	*(n = 31)*	*(n = 51)*	*(n = 12)*	
Active schizophrenic episodes	9	33	6	Further active psychotic episode(s) *v*. no further episode: $\chi^2 = 0.94$, NS Further active schizophrenic episode(s) *v*. no further episode: $\chi^2 = 9.87$, $P < 0.01$
Atypical psychotic episode	7	6	1	
Residual schizophrenia[3]	0	6	8	
Non-specific mental symptoms	7	8	1	
No mental symptoms[4]	7	9	1	
Physical disorders after index presentation				
No problems	21	30	7	No problems *v*. further problems: $\chi^2 = 0.74$, NS
Moderate problems	5	13	3	
Severe problems	1	3	2	
Died	4	5	0	
Length of initial follow-up after index presentation (all 116 patients)	*(n = 34)*	*(n = 66)*	*(n = 16)*	
Continuous (no defaulting)	6	5	4	
Over 12 months	4	6	4	
6+ to 12 months	3	16	2	
6 months or less	18	36	6	
Transferred to another unit	3	3	—	

[1] Did not recover within 6 months.

[2] Died during course of illness after defaulting; cause of death unknown.

[3] Persistence of residual schizophrenic symptoms beyond one year after index presentation.

[4] Excluding insomnia, which occurred at some stage in almost all patients.

Four of the schizophreniform and five of the active schizophrenic patients had died. One (a schizophreniform patient) had died during the index episode, of unknown causes, having defaulted from treatment. Another schizophreniform patient committed suicide. One schizophreniform and one schizophrenic patient had been beaten to death by night guards who had found them wandering, following relapse. The other five patients had died in traditional healers' establishments following relapse. It is very possible that some of those who could not be traced had also died.

At follow-up, the great majority of the schizophreniform and active schizophrenic groups were not receiving any form of treatment in a psychiatric unit. At the follow-up interview, a significantly higher proportion of the schizophreniform group were without any current mental disorder and a significantly higher proportion were not suffering from active psychotic features (Table V). The schizophreniform group also scored lower on the two indices of psychopathology employed, the modified BPRS (not statistically significant) and the Clinical Global Impression ($P < 0.05$).

TABLE V

Current clinical situation in the 94 patients evaluated at 25–38 months after index presentation

	Number of patients			*Statistical analysis*
	Schizophreniform group (n = 31)	*Active schizophrenic group (n = 51)*	*Residual schizophrenic group (n = 12)*	
Current source of treatment				
Nil	18	28	4	
The psychiatric unit	7	11	7	
Other hospital	0	2	1	
Traditional/religious healer	1	2	0	
Other	1	3	0	
Dead	4	5	0	
Current clinical situation				
No current mental disorder	19	22	2	No current mental disorder v. currently mentally ill: $\chi^2 = 9.91$, $P < 0.01$ Not actively psychotic v. currently actively psychotic: $\chi^2 = 3.64$, NS
Active schizophrenia	4	16	3	
Atypical psychosis	1	2	0	
Residual schizphrenia	0	4	7	
Other[1]	3	2	0	
Dead	4	5	0	
Modified Brief Psychiatric Rating Scale score: mean ± s.d.	5.33 ± 8.92	11.33 ± 12.19	8.92 ± 8.21	$F = 2.64$, NS (ANOVA)
Clinical Global Impression: mean ± s.d.	2.04 ± 1.81	3.13 ± 2.16	3.42 ± 1.56	$F = 3.56$, $P < 0.05$ (ANOVA)

[1] Neurotic disorder or non-specific symptoms.

The prognosis with regard to recovery of social and occupational functioning was distinctly better in the schizophreniform patients. These differences were statistically significant in terms of interval between index presentation and resumption of work (or school), of whether the individual was currently working at the time of follow-up interview, and of difficulties in family and social relationships, but not in marital relationships (Table VI).

Discussion

Since Kraepelin and Bleuler described schizophrenic disorders at the beginning of the century, their diagnosis has always carried the implications of chronicity and poor prognosis. Suggestions that a more acute disorder with similar symptoms might carry a better prognosis were formalised in the description of 'schizophreniform psychoses' by Langfeldt (1937). This concept has been incorporated into DSM–III. However, Langfeldt's concept of schizophreniform disorder is somewhat different from that of DSM–III, which is employed in this study. Langfeldt described an acute disorder with an obvious precipitant, marked by prominent clouding of consciousness and affective, hysterical or paranoid colouring as well as typical active schizophrenic symptoms. The ICD–9 (World Health Organization, 1978) concept is similar to that of Langfeldt. DSM–III differentiates schizophrenia from schizophreniform disorders merely on the basis of duration of symptoms. In fact it is likely that there is considerable overlap between these different concepts of schizophreniform disorder.

In the present study, apart from the longer duration of current illness among the two schizophrenic groups compared with patients suffering from schizophreniform disorder at the time of index presentation, there was also a significantly greater number of patients with a history of previous psychiatric episodes among the schizophrenic patients. However, the two groups with active psychotic symptoms (the schizophreniform and active schizophrenic groups) did not differ significantly in terms of degree of psychopathology, frequency of PSE symptoms, or signs or frequency of First Rank Symptoms.

Patients with schizophreniform disorder had a much better prognosis than the two schizophrenic groups in all parameters employed. This included

TABLE VI
Occupational and social data obtained on the three groups of patients 25–38 months after index presentation

	Number of patients			*Statistical analysis*
	Schizophreniform group (n=31)	*Active schizophrenic group (n=51)*	*Residual schizophrenic group (n=12)*	
Index presentation and interval between resuming work				
0 to 6 months	16	14	2	0–6 months *v.* over 6 months: $\chi^2=6.82$, $P<0.05$
6+ to 12 months	5	8	3	
12+ months	2	5	1	
Work not resumed	8	24	6	
Current occupational status				
Currently working	16	16	2	Currently working *v.* not currently working: $\chi^2=7.42$, $P<0.05$
Not working	11	30	10	
Dead	4	5	0	
Number of months absent from work since resumption				
Nil	15	9	2	No period of absence *v.* period of absence (the two schizophrenic groups combined): $\chi^2=3.86$, $P<0.05$
6 months or less	4	10	1	
6+ to 12 months	1	6	1	
More than 12 months	2	0	1	
Not applicable	9	26	7	
Marriage				
Satisfactory	5	9	1	No problems *v.* problems present: (the two schizophrenic groups combined): $\chi^2=0.02$, NS
Problems	3	3	1	
Complete breakdown	5	7	3	
Not applicable	18	32	7	
Relationship with family				
Satisfactory	21	22	5	Satisfactory relationship *v.* problems present: $\chi^2=6.73$, $P<0.05$
Moderate problems	5	10	4	
Severe problems	1	12	3	
Not applicable	4	7	0	
Social relationships				
Satisfactory	19	22	0	Satisfactory relationships *v.* problems present: $\chi^2 15.62$, $P<0.001$
Moderate problems	5	11	6	
Severe problems	3	13	5	
Not applicable	4	5	1	

initial clinical outcome, clinical outcome over a 2–3 year follow-up period, and outcome with regard to social and occupational functioning over the 2–3 year follow-up period. These findings are in agreement with the contention that schizophreniform disorders carry a better prognosis than schizophrenia *per se* (Fox, 1981). However, they do not necessarily support the contention that the two categories of disorder are different entities. If they were, it would surely be expected that there would be some differences with regard to clinical symptomatology (i.e. with regard to the frequency of occurrence of individual symptoms). Also, although the schizophreniform patients did have a better prognosis, they still suffered from a relatively high proportion (over 0.4) of relapses and of occupational, marital, family, and social problems. It is therefore equally possible that the two terms, schizophreniform disorder and schizophrenia, do not describe two different entities, but represent different degrees of acuteness of the same disorder. The apparently better prognosis in the schizophreniform group would then be a reflection of the more acute nature of the illness and the earlier presentation and treatment; both these factors are widely acknowledged as good prognostic indicators in schizophrenics (Slater & Roth, 1969; Bemporad & Pinsher, 1974; World Health Organisation, 1979). Residual schizophrenia, which in the present study carried the worst prognosis and in which duration of illness at index presentation was highest, would then represent the other, more chronic, extreme.

The commonest of Schneider's First Rank Symptoms encountered in both groups of patients with active symptoms was the experience of voices discussing or arguing about the subject, followed by 'made' acts. Other First Rank Symptoms occurred much less commonly. This is in marked contrast to findings from other populations (Mellor, 1970; Carpenter & Strauss, 1974; Chandrasena & Rodrigo, 1979; Bland & Orn, 1980). Unfortunately, the distribution of First Rank Symptoms in Nigerian schizophrenics found in the International Pilot Study of Schizophrenia (IPSS) (see Carpenter & Strauss, 1974) are not available for comparison. The overall frequency of occurrence of First Rank Symptoms in the two groups was comparable to that found in the IPSS.

Follow-up studies in industrialised nations over the current century have confirmed Kraepelin's and Bleuler's original observations that schizophrenia was associated with a poor clinical as well as social prognosis. (The studies are summarised by the World Health Organization (1979).) The international 2–3 year follow-up study carried out by the WHO (World Health Organization, 1979) in five industrialised and four developing countries found that schizophrenia was associated with a better prognosis in terms of clinical outcome in developing countries. The best outcome was recorded from Nigeria. Social outcome, assessed as a single item, was also better in the developing countries. The present study records a much worse outcome – clinically, occupationally, and socially – among schizophrenic patients than found by the WHO study for the developing countries. One reason for this is most certainly the diagnostic criteria employed. Patients were included in the WHO study on the basis of a diagnosis of schizophrenia on ICD-8 criteria (World Health Organization, 1973), which are looser than those of DSM-III, and no minimum duration of illness was employed; indeed, patients whose index illness had been present for more than three years or who had been in hospital for more than two years in the previous five years were excluded from the study. Since 73% of the patients overall (94% of the Nigerian patients) had been ill for less than five months, the great majority would have been given a DSM-III diagnosis of schizophreniform disorder rather than schizophrenia. Indeed, the WHO findings for developing countries, particularly Nigeria, are much more comparable with those of the present study for the schizophreniform group.

The patients described in this paper profoundly illustrate the failure of medical services in Nigeria to deal effectively with the problem of mental disorders. A high level of continued morbidity, both medically and socially, was observed in the majority of cases, even in the so-called better-prognosis 'schizophreniform' group. Clearly, the medical services offered represented merely an interlude, albeit with some temporary relief, in the course of a chronic or recurrent disorder which produces severe disturbances of social and economic functioning. A major problem lies in the fact that psychiatric services in developing countries, with few exceptions, consist of hospital units offering strictly medical services and without the personnel and facilities required for proper rehabilitation and after-care. In reality, patients are being given acute medical treatment of active symptoms, accompanied by rehabilitative measures which consist mainly of counselling of patients and families on the ward. After-care consists essentially of follow-up attendances at the out-patient clinic; the time, expense, and inconvenience to the patient almost invariably results in early failure of patient compliance. The heavy defaulting notable in the present study is therefore not surprising. It is obviously impractical to expect that developing nations can develop the elaborate rehabilitative and after-care facilities found in industrialised nations. However, it should be possible to improve services by developing community-based services manned by less skilled personnel, which would be backed up and supervised by the more highly skilled professionals. This subject is discussed in more detail elsewhere (Makanjuola, 1986). Various such schemes have been advocated but never put into effect. Until they are, the management of schizophrenia (and other mental disorders) in developing countries will continue to be ineffective, and the disorder a source of crippling long-term medical and social morbidity.

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The Incidence of Schizophrenia in Nottingham*

J. E. COOPER, D. GOODHEAD, T. CRAIG, M. HARRIS, J. HOWAT and J. KORER

Attempts were made to identify, and include in a two-year follow-up study, every patient living in the catchment area of the Mapperley group of psychiatric hospitals in Nottingham (population 390 000) who made their first-ever contact with the psychiatric services for a potentially schizophrenic illness during a two-year period (1 August 1978 to 31 July 1980). Screening was based upon symptoms rather than diagnosis, covering both in-patient and out-patient services; a consensus diagnosis using ICD–9 was made by the project team. The Nottingham Psychiatric Case Register was used in a retrospective Leakage Study which added nine cases to the 99 identified by the screening procedures. Incidence rates are given for both broad and narrow concepts of schizophrenia, and for DSM–III diagnosis. The Nottingham incidence rates are similar to those reported from other UK centres, and are near the middle of the range found in the other collaborating centres in the WHO study on Determinants of Outcome of Severe Mental Disorders. At entry to the study, 27 patients were out-patients, and 11 were never admitted to hospital at any time in the two-year follow-up period. Reasons for believing that the Nottingham administrative incidence may be close to the incidence in the community are discussed.

The WHO Collaborative Study on Determinants of Outcome of Severe Mental Disorder is an international study between 11 centres, designed and co-ordinated by the Division of Mental Health of the World Health Organization, Geneva.

An important aim of the study is to investigate further some of the findings of its predecessor, the International Pilot Study of Schizophrenia (IPSS) (World Health Organization, 1973, 1975, 1979), in which centres in nine countries took part (Czekoslovakia – Prague; Denmark – Aarhus; Colombia – Cali; India – Agra; Nigeria – Ibadan; Taiwan – Taipei; UK – London; USA – Washington; USSR – Moscow). The two-year follow up of the IPSS patients (Sartorius *et al*, 1977; World Health Organization, 1979) raised the important possibility that schizophrenic patients in developing countries (Nigeria, India, and Colombia) have a better outcome than patients in developed countries (UK, USA, USSR, and Denmark), even though their illness appears to be similar at onset. Other less extensive studies from individual centres have suggested a similar difference in outcome (Rin & Lin, 1962; Murphy & Raman, 1971; Waxler, 1979), but no single study can be regarded as giving definitive findings on this very important possibility. The IPSS, although on a large scale and using standardised methods of assessment and diagnosis (it included a total of 1200 patients, 800 of whom received a diagnosis of some form of schizophrenia), was not designed on an epidemiological basis, and it is possible, although not evident, that varied and unknown selection factors in the different centres were behind the different outcomes.

It was therefore decided to carry out another study using an extended network of collaborating centres, with patients collected in such a way as to obtain in each centre as complete a sample of patients as possible from a geographically defined population. As before, the study is basically a two-year follow-up study, and because of the demonstrated importance in the IPSS of social and demographic influence in determining outcome, steps were also taken to improve the quality and quantity of the information about the personal background, family structure, and social circumstances of the patients.

A joint report on the sociodemographic, clinical and diagnostic description of the study patients in each centre, together with the 'administrative incidence' in each centre, has been prepared by the World Health Organization (1986). The present paper deals with the Nottingham data on incidence rates in more detail, and illustrates how incidence rates may be presented in several different ways.

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*A report from the UK Field Research Centre of the World Health Organisation Collaborative Study on Determinants of Outcome of Severe Mental Disorders

Specific aims

The aim of the Nottingham study was to identify, and include in a two-year follow-up study, every patient living in the catchment area of the Mapperley group of psychiatric hospitals who made their first-ever contact with the psychiatric services because of a potentially schizophrenic illness during a two-year intake period (1 August 1978 to 31 July 1980).

Method

Method of screening for case-finding

The procedures for screening and assessment were as follows.

A purposely over-inclusive screening schedule (see Appendix 1) was used to identify patients with hallucinations or delusions of any type, or with clearly abnormal behaviour suggestive of psychotic illness, such as social withdrawal, or unexplained severe anxiety, aggression, or excitement. Patients with a clear organic cause for their symptoms, such as brain damage, toxic confusional states, alcoholic psychosis, epilepsy, or severe mental retardation, were not included in the study. All the psychiatric staff covering the psychiatric services of the Mapperley hospital group agreed to apply the screening schedule to all contacts with new patients during the study period. To facilitate this, meetings were arranged with groups of psychiatric staff of all grades of seniority, at which the purpose of the study and the design of the screening process were described. Copies of the screening schedule were deposited in all the out-patient clinics, and they were displayed prominently on the desks at every clinic session; copies were also placed on all in-patient admission wards. All nursing and psychiatric staff were encouraged to contact the project staff if they had the slightest query about a patient's eligibility for inclusion in the study. The instructional sessions were repeated at intervals throughout the study period, particularly after the twice-yearly intake of new junior staff.

Project team members made regular (usually weekly) checks with all the acute admission wards, to see if potential study patients, not already notified, had been admitted.

In addition, Medical Records staff (often doing joint work with the Psychiatric Case Register staff) were asked to notify the project whenever they came across the case-notes of a patient who had made their first contact during the study period.

After the intake period was over, the Mapperley Psychiatric Case Register was used to provide lists of all patients with selected diagnostic categories who had made their first contact during the study intake period. This exercise is described separately later, as part of the 'Leakage Study'.

Method of assessment

Mental state and behaviour

All patients whose screening schedule suggested that they should be in the study were interviewed as soon as possible by a project psychiatrist, who completed the Present State Examination (PSE) (Wing *et al*, 1974). In the great majority of cases, this was done within a few days of completion of the screening schedule; for those patients who were picked up as out-patients or on domiciliary visits, the delay was usually longer (about a week or so).

Psychiatric and Personal History Schedule and social disability

As soon as possible after the PSE schedule had been completed, a different interviewer completed the rest of the schedules with a close relative or other informant, covering the psychiatric, personal, family, and social background (PPHS) and social disability (DAS). In the great majority of cases this was done within one to two weeks. Since one of the issues in the data analysis is the relationship (or lack of relationship) between symptoms and social disability, the PPHS and DAS interviews were always done by an interviewer who had not done the PSE.

Project diagnoses

Project consensus ICD-9 diagnosis. When all the schedules had been completed, the Diagnostic and Prognostic Schedule (DPS) was completed at the next weekly project meeting. At least two (and usually three) project psychiatrists, together with the PHSS and DAS interviewer, discussed all the information available about the patient and family, and also perused the hospital case-notes. A consensus diagnosis was chosen, according to ICD-9, if necessary (but rarely) by a majority vote. Main, subsidiary, and alternative diagnoses were allotted as necessary, as described in the instructions for users of ICD-9 (World Health Organization, 1978). The diagnosis was made using only the information available at entry to the study, which was usually three to four weeks before the team meeting at which the diagnosis was made. In a small proportion of the patients, one or more of the team psychiatrists was also concerned in the clinical care of the patient, and so inevitably was aware of the patient's progress since admission to the study, but this information was not used in making the team consensus diagnosis.

Project diagnosis for certainty of schizophrenia. The presence of main, subsidiary, and alternative diagnoses allows the project diagnosis to be expressed in several different ways. In the presentation of results which follow, it is convenient to use a 'certainty of schizophrenia' code, devised as follows.

1. Schizophrenia certain: the main diagnosis is any of the schizophrenic categories except schizoaffective type (295.0–295.9, except 295.7), and no alternative diagnosis given.
2. Schizophrenia very likely: the main diagnosis is as for 1, but an alternative diagnosis is also given (any code).
3. Schizophrenia probable: the main diagnosis is schizo-affective psychosis (295.7) or a paranoid psychosis (including psychogenic) (i.e. 297.0–297.9, 298.3, 298.4).

4. Schizophrenia possible: the alternative diagnosis is a schizophrenic or paranoid category as in 1–3, but the main diagnosis is not.
5. Not schizophrenia: neither main nor alternative diagnosis is any of the schizophrenic or paranoid categories.

Leakage study

After the study intake period was over, several different types of check were carried out to try to find any cases missed by the screening system just described. These comprised checking with local psychiatrists who engage in private practice; checking with the two private psychiatric hospitals that might have admitted schizophrenic patients from Nottingham during the study period; and checking the records of the Nottingham Psychiatric Case Register.

No previously missed patients were found as a result of checks done by the local psychiatrists who accepted private patients and by the private psychiatric hospitals.

The Nottingham Psychiatric Case Register provided lists of all patients making their first contact with the services in the study period with a hospital diagnosis of Schizophrenia (295), Paranoid States (297), non-affective varieties of Other Organic Psychoses (298.3–298.9), and Paranoid Personality (301.0). The hospital case-notes of all these (*n* = 140) were examined and 13 patients found who had apparently been missed by the screening procedure. Of these 13, two had epilepsy in addition to schizophrenia, one also had an organic syndrome due to alcohol, and in one it was finally decided that the evidence for the presence of hallucinations or delusions was insufficient. This left nine patients who had been legitimate candidates for inclusion in the screening process. The reasons for missing these patients were as follows.

(a) Five patients – seen only as out-patients and never admitted; no special reason apparent why the screening schedule had not been completed.

(b) Two patients – very unwilling patients; seen only once as out-patients by a National Health Service clinician, and refused further appointments offered (with hints of future litigation in one case).

(c) Two patients – brief admissions lasting less than 48 hours, one over a week-end and one over a public holiday.

Sufficient clinical information was present in the case-notes of these nine patients to allow a reasonably confident diagnosis to be made, so a consensus ICD–9 diagnosis was made by two of the project psychiatrists in the same way as for those included in the screening process. Eight of these nine patients had certainty of schizophrenia codes of 1–4, so they were included in the calculation of incidence rates (but not followed up). It is worth noting that only three of the nine cases from the Leakage Study fall into the 'certain' or 'very likely' codes for schizophrenia already described, so their addition makes very little difference to the incidence rates of the more definitely schizophrenic diagnoses.

Results

Number of patients and rate of intake to the screening procedure

A total of 99 patients were taken into the study by means of the above screening procedure, 53 in the first year of intake and 46 in the second. The rate at which the patients entered the study was fairly steady, averaging out over the two years at just under one patient per week. In most months, between two and five patients were found, with few extremes (one month with ten cases and two months with one case); the range of 2–5 cases per month covers more than half of the months of the study. This group of

TABLE I
Diagnostic Certainty of Schizophrenia (ICD–9) at entry for Total Screening Intake group (n = 99) plus the Leakage Study patients (n = 9); two-year intake (total n = 108)

	Certainty of schizophrenia at entry					
	Certain	*Very likely*	*Probable*	*Possible*	*Not schizophrenia*	*Total*
Total Screening Intake Group						
Male	26	19	7	9	6	67
Female	11	11	4	2	4	32
Total	37	30	11	11	10	99
Leakage Study						
Male	1	0	2	1	0	4
Female	1	1	2	0	1	5
Total	2	1	4	1	1	9
Total						
Male	27	19	9	10	6	71
Female	12	12	6	2	5	37
Total	39	31	15	12	11	108

99 patients is referred to as the Total Screening Intake group. The Diagnostic Certainty of Schizophrenia is shown in Table I for the 99 patients in the Total Screening Intake group, plus the nine patients found in the Leakage Study (total $n = 108$).

The screening schedule was designed on a symptomatic basis as already noted, so as to produce a somewhat over-inclusive group from the diagnostic point of view. This is evident in that for ten of the 99 patients, all their main, alternative and subsidiary diagnoses were in the affective, neurotic or adjustment reaction categories; nine of these ten had a main diagnosis of Mania or Depression with mood-congruent delusions or hallucinations. The rates that are discussed below for the diagnosis of schizophrenia at various grades of confidence do not include these ten patients. The remaining patients were divided up in several ways for calculation of the incidence rates that follow, according to the narrowness or breadth of the diagnostic concept of schizophrenia that is under discussion.

The monthly variations in the rate of intake to the study of the total of 97 potentially schizophrenic patients (certain = 37 + 2 leakage; very likely = 30 + 1 leakage; probable = 11 + 4 leakage; possible = 11 + 1 leakage:total = 97) were not significant. The rate of intake to the study is only of administrative interest, and does not necessarily have a direct relationship to the time or type of onset of the illness. These aspects will be described in detail in later reports, but it is of interest to note that one-third of the patients had an illness onset that could be clearly identified in the four weeks preceding admission to the study.

Incidence of schizophrenia

Several different incidence rates for schizophrenia can be derived, depending upon different definitions of schizophrenia. The joint report from all the field research centres in this study (World Health Organisation, 1986) uses the CATEGO data derived from the PSE ratings. This is the most standardised type of diagnostic statement available, and is appropriate for comparisons between centres even though it does not have quite the same meaning as a set of clinical diagnoses expressed in terms of ICD-9. The CATEGO data will not be discussed here again in detail, apart from a brief mention in the discussion section. The rates given here are based upon the project ICD-9 consensus diagnosis, which probably bears a close resemblance to diagnoses made by many general psychiatrists in the UK. Rates are given for both broad and narrow definitions of schizophrenia using ICD-9, and for schizophrenia using DSM-III.

Using a broad definition of schizophrenia

The top section of Table II shows the age- and sex-specific rates for the summed ICD-9 categories of

(a) all categories of Schizophrenia (295)
(b) Paranoid States (297)
(c) non-affective types of 'Other Non-Organic Psychosis' (298.2–298.9) (often called 'Reactive Psychoses').

There are 85 patients in this group, which will be referred to as the Total Incidence of Schizophrenia group. It is derived from the Total Screening Intake group ($n = 99$) by taking out those patients with a main diagnosis in the Affective Psychosis or non-psychotic categories ($n = 10$) and then adding the seven patients from the Leakage Study with schizophrenic-related main diagnosis. (This is the same as adding together the first three 'certainty of schizophrenia' groups, of certain, very likely and probably schizophrenia, and seven patients from the Leakage Study.) The rates are markedly higher for young men than for young women, with a less marked reversal of this difference in the 40–49 age group. For men and women together, the rates are highest (2.8 per 10 000) in the 20–29 year age group.

For the ICD-9 definition of Schizophrenia (295.0–295.9)

The middle section of Table II shows the rates for men and women together derived from the 76 patients (52 men and

TABLE II

Mean annual incidence of new cases of schizophrenia: Nottingham (August 1978 to July 1980)

	Rates per 10 000 (numbers in brackets)					
	Age 15–19	*Age 20–29*	*Age 30–39*	*Age 40–49*	*Age 50–54*	*All ages 15–54*
ICD-9 broad concept of schizophrenia[1]						
Male	2.1 (7)	4.0 (27)	2.6 (13)	1.4 (6)	1.4 (3)	2.8 (56)
Female	2.4 (8)	0.7 (4)	1.9 (8)	2.2 (9)	0.0 (0)	1.4 (29)
Male and female	2.3 (15)	2.8 (31)	2.2 (21)	1.8 (15)	0.7 (3)	2.2 (85)
ICD-9 Schizophrenia (295 only)						
Male and female	2.2 (14)	2.6 (29)[2]	1.9 (18)	1.4 (12)	0.7 (3)	2.0 (76)
DSM-III Schizophrenic Disorder						
Male and female	0.3 (2)	1.4 (16)[3]	1.1 (10)	0.7 (6)	0 (0)	0.8 (34)[4]

1. 295, + 297, + 298.2–298.9.
2. Male 26, female 3.
3. Male 16, female 0.
4. Male 23, female 11.

24 women) with ICD-9 Schizophrenia (295) as a main diagnosis. The same differential pattern for age and sex exists as for the broad definition.

An even narrower definition of schizophrenia can be achieved by omitting the six patients with a main diagnosis of Schizoaffective Schizophrenia (295.7), but this makes little difference to the overall rates, and does not affect the sex and age differences.

Incidence rates using DSM-III

A DSM-III diagnosis (American Psychiatric Asociation, 1980) was also made for all the patients in the study. This was not done at the time of entry, since the final form of DSM-III was not then available. Some time after the study was completed, two psychiatrists, one of whom (JEC) was familiar with the study and the patients, made themselves familiar with DSM-III, and then made independent diagnoses on all the study patients on DSM-III (Gregory & Cooper, 1983). All the special study documents (schedules and narrative summaries) and the hospital case-notes were used. An agreed DSM-III diagnosis was then reached by discussion of any differences in the independent diagnoses.

Because of the DSM-III requirement of the presence of symptoms for at least six months to qualify for a diagnosis of Schizophrenic Disorder, only 34 patients received this diagnosis. The incidence rates derived from these patients are shown in the bottom section of Table II. At entry to the study, the 76 patients with an ICD-9 diagnosis of Schizophrenia (295) were divided between the DSM-III diagnoses of Schizophrenic Disorder ($n=34$), Schizophreniform Disorder ($n=20$), Schizoaffective Disorder ($n=4$) and Atypical Psychosis ($n=18$). Some of these patients fulfilled the six months duration of symptoms criterion during the follow-up period, so it is possible to derive a compound 'retrospective incidence' rate for DSM-III Schizophrenic Disorder by adding together the 34 patients qualifying at entry and those qualifying later (ten men and five women). This gives an overall age-specific rate of 1.7 per 10 000 for men ($n=34$) and 0.7 per 10 000 for women ($n=15$), and an overall rate for men and women together of 1.3 per 10 000 ($n=49$).

Rates per total population

All the rates quoted above are age- and sex-specific. Since new cases of schizophrenia are rare below the age of 15 and above the age of 54, and there are marked sex differences at some ages, age- and sex-specific rates give a more complete picture of the incidence of schizophrenia than total population rates. But some studies quote only total population rates, so these are needed in order to make comparisons. For Nottingham, this means that the population base rises from 202 214 aged 15-54 years to 380 028 total population of all ages. The rates therefore fall to just over half of the age- and sex-specific rates; the overall rate (men plus women) for the broad ICD-9 concept falls from 2.2 to 1.2 per 10 000, the ICD-9 Schizophrenia (295 only) falls from 2.0 to 1.1 per 10 000, and the DSM-III rate for Schizophrenic Disorder at entry falls from 0.8 to 0.5 per 10 000 (the DSM-III compound retrospective incidence rates for men and women together falls from 1.3 to 0.7 per 10 000).

Discussion

Administrative incidence and the completeness of the sample

The effort put into obtaining as complete a sample as possible for this study was aimed primarily at providing the best possible group for follow-up, rather than being able to quote very accurate incidence rates. Nevertheless, the comparison of the Nottingham incidence rates with those found in other similar studies is of considerable interest, since it is likely that the Nottingham sample is close to being complete.

As the joint paper (World Health Organization, 1986) emphasises, the aim in this study is to estimate the 'administrative incidence'; that is, to obtain a complete sample of patients making their first-ever contact with those services or agencies which were identified in each centre as usually being the most important early point of contact for an individual with severe mental illness; in the UK this was taken to be contact with a psychiatrist. The study does not have the more ambitious aim of estimating the total community incidence. In some of the study centres it is difficult to estimate precisely how big the difference between administrative incidence and community incidence might be, although detailed study of this point often suggests that the difference may be surprisingly small. In the UK, and in Nottingham in particular, there are a number of reasons for believing that the administrative incidence and the incidence in the total community may be very similar. The Nottingham rates can also be regarded as those of a typical English urban community, as far as such a concept is useful. The reasons stem from the nature of the Nottingham community, the structure of the UK National Health Service, the particular structure within this of the psychiatric services of the Mapperley group of hospitals, and the presence of a psychiatric case register.

The nature of the Nottingham community

The town of Nottingham has a comparatively stable population with little outward movement, and the population indices for such items as social class and type of housing are close to the national average. The proportion of the population born overseas or of recent overseas origin is stable at around 7%.

The structure of the UK National Health Service

Virtually everybody in the UK is registered with a general practitioner in the National Health Service, to whom referral is quick, easy, and free. It is very unusual in the UK for general practitioners not to refer patients to the psychiatric services if they suspect a serious mental illness. This was confirmed by one of the authors (JEC) when making personal contact with over 20 general practitioners (for the purposes of another study) during the intake period. When asked if they ever treated patients who might possibly have schizophrenic or other serious mental illness without requesting a specialist opinion as soon as possible, without exception the general practitioners reported that they always sought psychiatric advice without delay.

There is very little private psychiatric practice (i.e. outside the National Health Service) in Nottingham. At the time of the study, only two of the 12 consultants covering the catchment area accepted private patients, and then only for a maximum of one-tenth of their working week. In any case, patients with serious and potentially chronic illness will almost always be treated within the National Health Service after the first consultation. As noted, checks on the local private practitioners were included in the Leakage Study.

The structure of the psychiatric services of the Mapperley group of psychiatric hospitals

The psychiatric services centred upon Mapperley Hospital cover the majority (390 000) of the population of the town of Nottingham (population about 400 000, depending upon how it is defined). The hospital catchment area is defined exactly, and no other psychiatric services operate within the area. In-patient, day-patient and out-patient services (including domiciliary visits) were all covered by the Mapperley-based psychiatrists, working in sector-orientated clinical teams. At the time of the study, there was an increasing emphasis on sectorisation, and on community-based care and close collaboration with general practitioners. It is very unusual, as subsequent checks in the Leakage Study showed, for patients of any sort to be referred to psychiatrists outside the hospital catchment area.

Nottingham in relation to the other field research centres in the WHO study

Nottingham is around the middle of the range of rates found in the eight field research centres for which incidence rates can be calculated with confidence (Aarhus, Chandigarh - urban, Chandigarh - rural, Dublin, Honolulu, Moscow, Nagasaki, and Nottingham). These rates and other incidence data are presented in more detail in the joint report (World Health Organization, 1986). It is sufficient for present purposes to note that, for instance, of the 85 Nottingham patients in the 'broad definition of schizophrenia' group, 66% have a CATEGO class of S^+ (this class indicates the presence of one or more 'first rank' symptoms (Schneider, 1959, 1974; Mellor, 1970)). A further 16% have CATEGO classes of S?, P^+ or O^+, so a total of 82% of Nottingham patients have CATEGO classes known to be very strongly correlated with a clinical diagnosis of schizophrenia. This proportion puts Nottingham into fourth place among the above eight centres.

The separate age- and sex-specific rates are shown in Table II only for the largest group (the broad definition of schizophrenia), since the numbers in the smaller age- and sex-specific groups become very small when the total in the group drops. However, in spite of the small numbers, the pattern of sex and age distribution seen in the broad definition group is maintained throughout; that is, markedly more young men than young women, and slightly more women aged 40–49 than men. The same pattern is also found in 10 of the 12 centres in the overall WHO report, so this pattern, although not an invariable finding in previous studies, is common enough to be regarded as fairly typical.

Comparisons with other incidence studies, and the accuracy of case register information

The Nottingham total population rates derived from this study are lower than those from the Salford Case Register, and the same as those from Camberwell; for 1969–1973, the Salford and Camberwell mean rates were 1.4 and 1.1 per 10 000 respectively (Wing & Fryers, 1976). The uncorrected Nottingham Psychiatric Case Register rate (for the total population) is the same as for the Salford Psychiatric Case Register.

Comparison with rates from other studies done in other countries is of interest, but there is no point in going into great detail since these previous studies were done under different (usually more difficult) service conditions, with different diagnostic criteria and with less standardised methods of assessment and diagnosis. When reviewing these problems in relation to incidence rates, Cooper (1978) noted rates of 7.2 per 10 000 in New York State (Babigian, 1975), 5.4 in Mannheim (Hafner & Reimann, 1970) and 5.2 in Dublin (Walsh, 1969). He predicted correctly that "more rigorous definitions of a new case might result in much reduced estimates", and it seems likely that the Nottingham, Camberwell, and Salford case register rates, all of the same order of magnitude and markedly lower than most other rates quoted in the literature, represent the effect of efforts in this direction. A more recent report from Ireland (O'Hare *et al*, 1984) also seems to fulfil this expectation.

Incidence rates using DSM–III criteria

No other DSM–III figures are yet available for comparison. The DSM–III requirement that symptoms must have a duration of at least six months before the patient can qualify for 'Schizophrenic Disorder' raises a new type of problem in psychiatric epidemiology. Even the complex 'retrospective incidence rate' reaches only 0.7 per 10 000 total population, and all the age- and sex-specific rates on DSM–III criteria are much lower. Whether or not it is justified to include a criterion of long duration of symptoms in the definition of schizophrenia, or any other disorder, needs to be debated elsewhere, but the effect on incidence rates is immediately clear. An incidence rate no longer has the same meaning as for other disorders that do not possess a criterion of long duration, and unless a survey of such a disorder continues as a follow-up study for at least as long as the specified duration of symptoms, the determination of incidence rate cannot be complete.

Effects of ICD–9 criteria and the service setting of this study

In contrast to the DSM–III criteria, the ICD–9 description of schizophrenia contains no restrictions on duration of symptoms: schizophrenia can range from very acute to chronic, and the symptoms need to last only long enough to bring the patient to the attention of those making the assessment.

Particular care was taken to follow the ICD–9 description for all the schizophrenic and paranoid categories, so the diagnoses arrived at represent the cross-sectional descriptive approach that is typical of most parts of ICD–9.

A number of the Nottingham patients have illnesses of very acute and recent onset, with clear possible causes; it is very likely that some psychiatrists would be reluctant to make a diagnosis of schizophrenia in such patients and would prefer labels which, in their particular view, carry different implications about aetiology and prognosis.

If this point is taken together with the likelihood that very few patients from the catchment area population have been missed, it can be concluded that this group of patients is an unusually complete sample of those suffering from psychiatric illnesses that can be called schizophrenia quite legitimately according to the cross-sectional descriptive approach used in ICD–9. Both acute and less acute disorders are included, with and without clear aetiology, and irrespective of their service status after being identified. For instance, 27 patients were out-patients at entry to the study, and 11 were never admitted to hospital at any time in the two years of the follow-up. This study group is therefore suitable for investigating the implications of the ICD–9 concept of schizophrenia and related illnesses, in both cross-sectional and longitudinal studies. Much effort was put into the collection of information about the social background, family composition, and social disabilities of these patients, and the main interest of the study lies in the relationship during the follow-up period of these additional types of information with the clinical state.

Acknowledgements

We are most grateful to the consultant staff of the Mapperley group of hospitals for permission to study their patients. Medical, clerical and administrative staff gave considerable help in finding and contacting the patients during the period of study, for which thanks are due. The study was supported in part by grants from the Division of Mental Health, World Health Organisation, Geneva, and from the Department of Health and Social Security.

Appendix 1
Extract of main points covered by the screening schedule

Section A. Temporary residents, visitors and persons of no fixed abode were included only if they had lived in the catchment area for at least six months in the year preceding the contact.

Section B. Exclusion criteria (any of 1–3):
1. Clinically manifest organic cerebral disorder (confusion etc.)
2. Severe or moderate mental retardation
3. Organic CNS damage (encephalopathy).

Section C. Inclusion criteria (any of 1–5):
1. Hallucinations or pseudo-hallucinations in any modality
2. Delusions
3. Marked thought and speech disorder
4. Marked psychomotor disorder
5. Emergence of marked exacerbation of bizarre and grossly inappropriate behaviour.

Section D. Inclusion criteria – a definite change of personality and behaviour during the last year, manifested in any of the following:
1. Marked reduction or loss of interests, initiative and drive
2. Emergence or marked exacerbation of social withdrawal
3. Severe excitement, purposeless destructiveness or aggression
4. Episodic or persistent states of overwhelming fear or severe anxiety
5. Gross and persistent self-neglect.

Section E. Exclusion criteria: admission to hospital, or diagnosed or treated for a psychotic disorder similar to or continuous with the present illness, at any time before the last three months.

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Ecological Structure and the Distribution of Schizophrenia and Affective Psychoses in Nottingham*

J. A. GIGGS and J. E. COOPER

The 800 census enumeration districts of Nottingham were used as a basis for the production of 12 distinct 'clusters' or neighbourhood types. Plotting the place of residence of the 68 patients with 'certain' or 'very likely' diagnoses of ICD-9 schizophrenia, identified in the World Health Organization Study of Determinants of Outcome of Severe Mental Disorders, showed the expected concentration in central areas of low social status. A more varied distribution was found for 132 patients with a case-register diagnosis of affective psychosis, contacting the psychiatric services for the first time during the same two-year period as the schizophrenic patients. Some of the highest rates for affective psychosis were found in new housing estates of middle and low status at some distance from the town centre.

The study of the geographical distribution of psychiatric disorders in urban populations has a quite substantial pedigree. It is generally accepted that Faris & Dunham (1939) established the methodological foundations of modern ecological psychiatric investigations, for in addition to providing a comprehensive review of then existing research, they also showed that, within Chicago, there were substantial spatial variations in both the incidence and prevalence rates of psychiatric hospital admissions. For every diagnostic group except manic-depression, the highest rates were found in the populations living in central city neighbourhoods. With increasing distance from the inner city the rates declined regularly and progressively, in a broadly gradient-like fashion. The methodology of this pioneer study has since been repeated frequently in many countries (Freeman, 1984). In almost every case the geographical patterns identified have been substantially the same as those revealed for Chicago.

Although the techniques employed in all these studies have been essentially geographical, it is a regrettable fact that geographers themselves have contributed only modestly, and recently, to this important field (Giggs, 1973, 1980, 1983; Smith, 1980, 1984). However, despite the relative novelty of their involvement, geographers have been able to demonstrate that the analysis of spatial variations in the distribution of mental disorders within cities is characterised by both serious problems and considerable potential. These issues are discussed in detail elsewhere (Giggs, 1979, 1983; Smith, 1980, 1984). A report on the incidence of schizophrenia in Nottingham (Cooper *et al*, 1987) provides the opportunity to exemplify some of the specifically geographical contributions to the analysis of the spatial distribution of this severe mental disorder.

In attempting to identify variations in the geographical distribution of mental disorders within large urban areas, the researcher is faced with a seemingly bewildering array of choices concerning which particular set of sub-areas to choose. Thus for Chicago, Faris & Dunham (1939) used several spatial frameworks for their maps, ranging from 120 'subcommunities' (i.e. blocks of neighbouring census tracts) at one end of the scale, down to only 11 'natural area' types, differentiated by contrasting social and housing attributes. In contrast, most British researchers have used electoral wards. A recent analysis of psychiatric hospital admissions in Bristol (Ineichen *et al*, 1984) typifies this strategy. Regrettably, however, electoral wards are too gross, since they generally contain markedly divergent social and housing attributes. Their boundaries rarely coincide with those of the social and housing areas found within cities. Moreover, from the 1966 population census onwards, in Britain, it has been possible to obtain census data for the so-called 'small areas' (i.e. enumeration districts, with an average population size of 500 persons). Using well-tested statistical methods (Knox, 1982; Davies, 1984), geographers have shown that these small areas can

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*A report from the UK Field Research Centre of the World Health Organisation Collaborative Study on Determinates of Outcome of Severe Mental Disorders

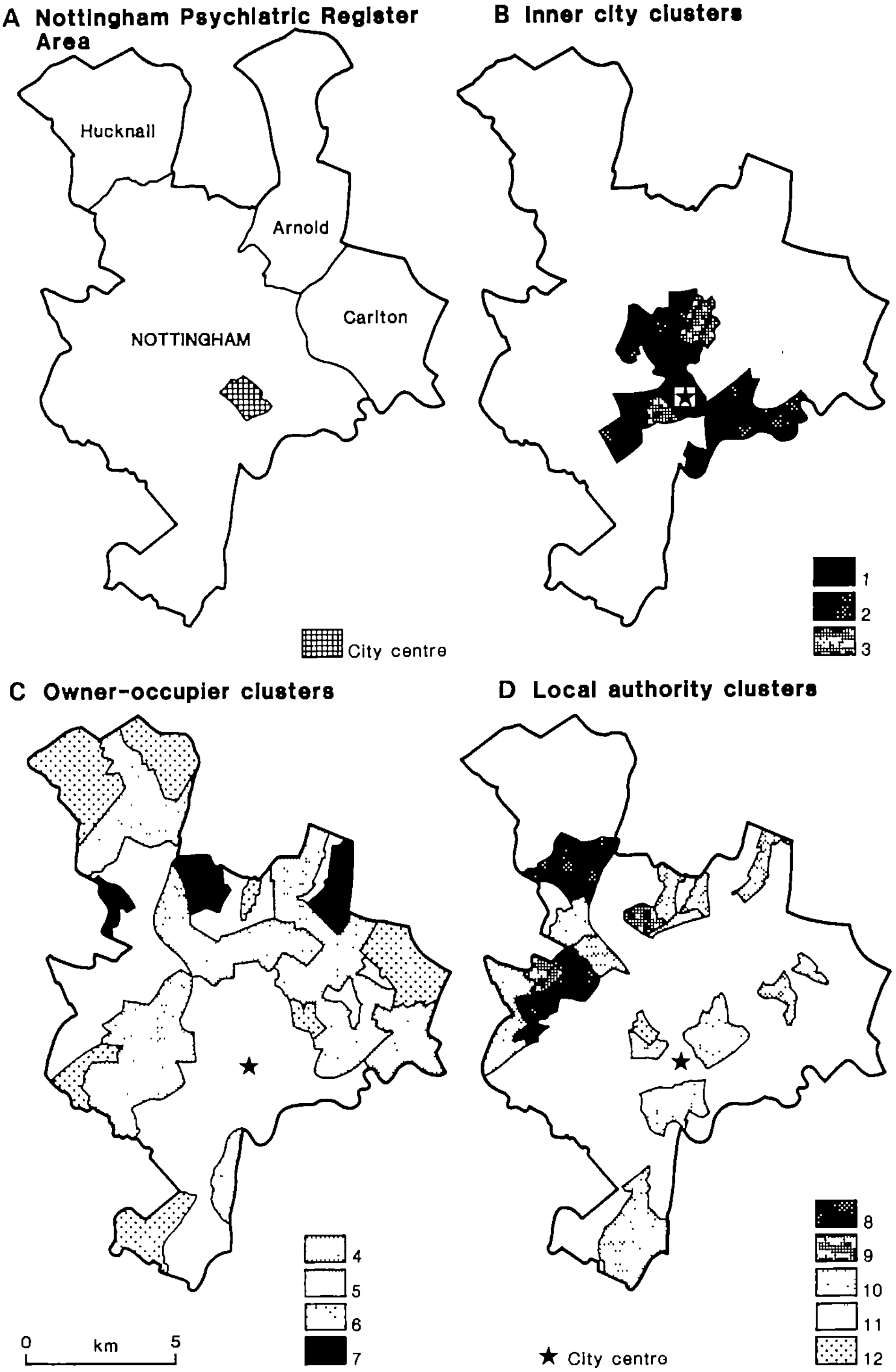

FIG. 1 Nottingham Psychiatric Register area and its neighbourhoods.

be grouped into larger units possessing uniform social and housing characteristics. Thus the analysis by Short (1978) of ten social and housing variables in Bristol identified eight neighbourhood types which would provide a contextual framework superior to electoral wards for the analysis of the geographical distribution of mental disorders.

Method

For the present investigation, 36 of the 1981 census variables and four variables derived from local authority sources (Payne, 1983, 1984) were collected for the 800 enumeration districts located within the Nottingham area. This data matrix was then analysed using Principal Components Analysis and a clustering program (Giggs & Cooper, 1986). These methods revealed that Nottingham is characterised by 12 distinct 'clusters', or neighbourhood types (Fig. 1, parts B–D). Some of the major social and housing attributes of these neighbourhoods are presented in Table I. It is suggested that these statistically derived areas provide an effective contextual framework for the analysis of spatial variations in the incidence of mental disorders. The computer-based data bank of the Nottingham Psychiatric Case Register has recently been matched with an address-based computer file (Giggs, 1984*a*). Consequently it is now possible to code the place of residence of every patient (from 1975 onwards) by enumeration district, and then to map the distributions. This has been done for both the 68 schizophrenics and the 132 patients with affective psychosis. At the time of the study, the Nottingham Psychiatric Case Register recorded all face-to-face contacts between psychiatrists and psychiatric patients living in the catchment area of the psychiatric services based on the Mapperley group of hospitals (population 380 000). This catchment area (defined exactly, street by street) contained the great majority of the town of Nottingham, and so was virtually all composed of inner-city, urban or suburban environments. Because of the nature of the National Health Service, the structure of the Nottingham psychiatric services, and the absence of alternative types of psychiatric care, there are good reasons to presume that the 'administrative incidence' of schizophrenia and other serious mental illnesses in Nottingham (Cooper *et al*, 1987) is close to the incidence in the community as a whole, particularly for schizophrenia.

The schizophrenic patients examined here comprise the 68 who were given study diagnoses of 'certain' or 'very likely' schizophrenia in the latest World Health Organization collaborative study (Cooper *et al*, 1987). At the time of first contact these patients were living in private households (i.e. in non-institutional settings) within the Nottingham Case Register Area (part A of Fig. 1). In addition, for comparative purposes, the analysis includes a consideration of the 132 patients with a hospital diagnosis of ICD-9 296.0–296.6 (i.e. the more specific forms of affective psychosis) who contacted the local psychiatric services for the first time during the same two years as the schizophrenic group (August 1978 to July 1980). These patients were obtained retrospectively from the Mapperley Psychiatric Case Register, so their diagnoses were not derived from the same consensus method as the diagnoses of the schizophrenic patients. However, the way in which the hospital psychiatrists are known to use the diagnoses of affective or manic-depressive psychoses means that it can be said with confidence that the group is composed almost entirely of patients with severe depression or obvious hypomania. This was also confirmed when the case-notes of these patients were examined during the 'Leakage Study' to ensure that there were no schizophrenic patients among them (Cooper *et al*, 1987).

TABLE II
Average annual inception rates of schizophrenia and affective psychosis in the populations of Nottingham's neighbourhoods

	Schizophrenia		*Affective psychosis*	
	Annual rate/1000	*Standardised rate*	*Annual rate/1000*	*Standardised rate*
Neighbourhoods				
1. Low status, transient	0.489	289.3	0.611	180.9
2. Low status	0.328	194.4	0.383	113.3
3. High status, transient	0	0	0.575	170.0
4. Elderly and post-child	0.103	55.9	0.327	88.8
5. Mature and post-child	0.105	61.8	0.196	58.1
6. Mature families, working women	0.050	29.3	0.174	51.3
7. Young families	0.079	46.7	0.751	222.2
8. Ageing families, low status	0.102	60.1	0.711	150.1
9. Mature families, low status	0.096	57.1	0.096	87.7
10. Mature families, middle status	0.199	117.5	0.275	81.4
11. New estates, low status	0.304	242.3	0.270	72.8
12. Newest estates, low status	0.795	313.6	0.530	392.0
Nottingham Register Area	0.169	100.0	0.338	100.0

TABLE I

Selected housing and social characteristics of Nottingham's neighbourhoods; 1981

Neighbourhoods	*Housing*			*Population*			
	Owner-occupied: %	*Local authority:* %	*Share amenities:* %	*Unemployed adults:* %	*Unmarried adults:* %	*Moved house 1980–1981:* %	*Population change 1971–1981:* %
Inner city, mixed tenure							
1. Low status, transient	47.0	11.4	17.0	19.2	20.6	17.5	−30.8
2. Low status	46.3	24.8	11.8	14.9	13.3	12.5	−28.9
3. High status, transient	60.5	3.9	6.4	4.9	18.8	14.9	−5.2
Middle–high status, mainly owner-occupied tenure							
4. Elderly and post-child families	58.4	30.4	4.3	7.3	6.1	8.4	−7.5
5. Mature and post-child families	70.3	20.3	3.2	5.9	7.2	7.7	+9.3
6. Mature families, working women	59.7	30.0	1.2	6.7	5.9	8.4	+47.8
7. Young families, post-1970 housing	64.8	32.9	0.2	7.4	6.4	10.2	+89.8
Low–middle status, mainly council tenure							
8. Ageing families, low status	18.4	78.5	6.5	12.1	5.5	7.3	+5.6
9. Mature families, low status	8.0	90.1	1.0	22.4	5.1	7.7	+9.1
10. Mature families, middle status	30.2	66.2	3.0	9.8	7.2	7.0	+1.5
11. New estates, low status	13.1	79.1	6.1	18.2	12.9	12.2	−44.7
12. Newest estates, low status	15.4	56.5	13.1	36.7	26.1	19.1	−43.6

Results

The average annual incidence rates of both schizophrenia and affective psychosis for the 12 neighbourhood types are presented in Table II, together with the standardised rates (i.e. the rate for each neighbourhood type expressed as a ratio of the rate for the whole of Nottingham). The standardised rates for schizophrenia and affective psychosis are also mapped in parts A and B of Fig. 2. It is evident that there are substantial spatial variations in the incidence of these two severe mental disorders. In the case of schizophrenia the pattern is the familiar one of inner city dominance (Fig. 2, part A), with rates exceeding twice the Nottingham average. For affective psychosis, however, the distribution pattern is strikingly different. Thus the highest rates are found in certain suburban areas, namely the cluster 7 tracts (compare Fig. 1, part C and Fig. 2, part B). These are typified by middle status, early to mature families (i.e. children, parents under 45 years old), high population turnover, and post-1970-built owner-occupied housing. Above-average rates are also found in several inner city neighbourhoods. This more varied distribution pattern has also been found in many other large urban areas (Faris & Dunham, 1939; Hare, 1956; Levy & Rowitz 1973; Bagley & Jacobson, 1976), but the comparatively high social status of the tracts with the highest rates of affective psychosis is a new finding.

Although many researchers have demonstrated that there are substantial variations in the distribution of various mental disorders within cities, few have subsequently tested the *significance* of these findings. In the few instances where statistical tests *have* been used, the spatial units employed have been highly aggregated (e.g. Gerard & Houston, 1953; Jaco, 1954; Hare, 1956). Thus Hare (1956) grouped the electoral wards of Bristol into only three units. The dearth of statistical tests in the literature of psychiatric ecology is a serious issue, because the testing of results is an important stage in epidemiological research. Cooper (1973) has argued that "The basic aims of epidemiology are to estimate rates of (disease) inception and prevalence in populations, *and to test for differences between the rates for defined subgroups*".

The distributions of schizophrenia and affective psychosis in Nottingham were therefore analysed, using the Poisson

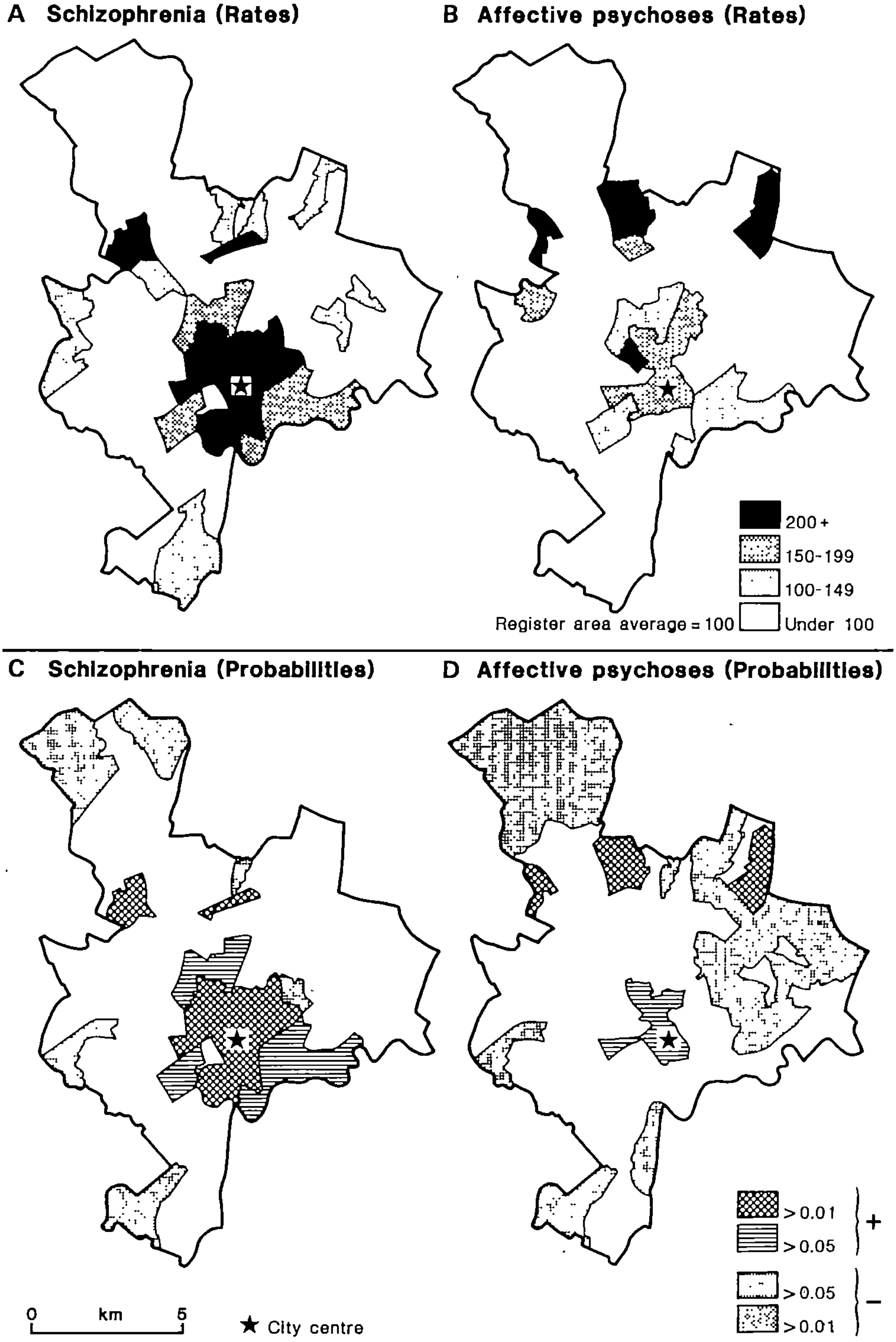
A Schizophrenia (Rates)
B Affective psychoses (Rates)
200+
150-199
100-149
Under 100
Register area average = 100
C Schizophrenia (Probabilities)
D Affective psychoses (Probabilities)
>0.01
>0.05
+
>0.05
>0.01
−
0 km 5
★ City centre

FIG. 2 Distribution of schizophrenia and the affective psychoses.

probability test (Norcliffe, 1977). It is thus possible to determine whether the incidence of the two disorders is significantly higher or lower in any of the 12 neighbourhood types than in the population of the entire study area. The results of these tests are shown in parts C and D of Fig. 2. The analyses confirm that the inner city neighbourhoods of Nottingham have significant excesses of schizophrenics (Fig. 2, part C). In contrast, the significant excesses of affective psychotics are found chiefly in three middle-status suburban tracts ($P<0.01$), but also in the central, low-status, 'rooming house' district (i.e. neighbourhood type 1, $P<0.05$; compare Fig. 1, part B and Fig. 2, part D).

Discussion

The distribution of new schizophrenic patients in Nottingham during the late 1970s was very similar to that in Chicago some 50 years ago (Faris & Dunham, 1939). However, there are some important differences between the findings for Nottingham and those of Chicago and other cities. The Nottingham study has a more complete sample of the total possible cohort of schizophrenics in the community (11 of our patients were never admitted to hospital), and the information on the Nottingham patients and, in particular, the project diagnosis, was of a higher quality and reliability. In contrast, the Chicago study and virtually all subsequent investigations were based exclusively on hospital admissions and the collected hospital admission diagnosis, whose quality must have been difficult to assess.

The Nottingham study also differs from previous studies because the spatial distribution of schizophrenia has been analysed and tested within the context of an objectively derived set of distinctive neighbourhoods. These areas provide a more accurate characterisation of the significant social and housing areas of cities than the large administratively defined areas (e.g. electoral wards) which customarily have been used in intra-urban ecological studies.

Despite these important differences, the essential similarity of the findings of the present study and existing research is marked. The apparent stability of the intra-urban spatial distribution of the incidence of schizophrenia across both time and nationality is striking. Two earlier investigations of the incidence of schizophrenia within Nottingham confirm the marked stability of the high-rate inner city neighbourhoods over nearly two decades (Giggs, 1973, 1983). This local stability is also striking. Between 1971 and 1981 the total population of the whole of Nottingham remained fairly stable, declining by only 5.6%. However, this general picture conceals highly divergent local variations within the study area. Thus the four working class inner city neighbourhoods (i.e. groups 1, 2, 11 and 12 – see Table I and parts B and D of Fig. 1) experienced population losses of between 28.0% and 44.7%. Moreover, these are not population losses overall: between 1961 and 1971 the nineteenth century slum housing in the tracts comprising neighbourhoods 11 and 12 was completely demolished and replaced by new inner city local authority housing estates and deck-access flattened complexes. During the decade, the former residents were rehoused in suburban estates and very few subsequently returned to the renewed inner city housing areas. Despite these profound changes in the size and character of the population and the housing stock between 1971 and 1981, the incidence of schizophrenia during 1978–1980 was remarkably similar to that during 1969–1973 (Giggs, 1983) (but it must be noted that the 1969–1973 rates were based upon uncorrected case-register diagnoses).

For the diagnosis of affective psychosis, the geographical distribution is very different from that for schizophrenia. Instead of a single major central city concentration of high rates, there is an apparently random scatter (Fig. 2, parts B and D), but a separate investigation of the ecological correlates of this disorder (Giggs & Cooper, 1986) suggests that the high rates of manic-depressive psychosis are correlated with high levels of population mobility. Thus the highest rates of this particular psychosis are concentrated in the new suburban estates (i.e. neighbourhood 7) and the inner city 'rooming house' district (i.e. neighbourhood 1), both of which are known to be characterised by high population turnover. Similar findings have been reported elsewhere (Giggs, 1984*b*).

The distribution of schizophrenic patients that we have found is broadly what we expected, but there are two main points of special interest. Firstly, the findings are unusually clear-cut, probably because of the unusually precise and comprehensive characterisation of both the schizophrenic cohort and the neighbourhoods within Nottingham. Secondly, we shall be exploring the connections between place of residence (i.e. neighbourhood type and characteristics) and follow-up. We shall be analysing the extensive information that we have on clinical state and social disability at the one-year, two-year and later follow-up points. Some interesting findings concerning residential mobility behaviour have already been identified (Giggs & Cooper, 1986).

The geographical data, like that on incidence rates, is of interest in its own right, but it has greater practical importance when set against outcome. The main purpose of this collaborative study is to try to learn more about what determines outcome in schizophrenia. To do this we need to examine carefully and systematically as many attributes of the patients, their families, and their neighbourhood settings as we can.

No particular aetiological clues connected with these different geographical distributions emerge from the data of this study at the present state of analysis. Limitations of manpower and finance made it impossible to carry out formal studies of pre-morbid life events, which could perhaps be associated with the different styles of life in the ecologically different areas. Looking at the possibility using information collected at the ordinary clinical level of detail and accuracy, there was no particular suggestion of unusually high rates of stressful events or physical illnesses. The new finding of high rates of manic-depressive illness in areas of high population mobility and a reasonable level of material resources suggests that it would be justified to study the pre-morbid personality and social mobility of this type of patient and family.

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Double-Blind Comparison of Half-Dose and Standard-Dose Flupenthixol Decanoate in the Maintenance Treatment of Stabilised Out-Patients with Schizophrenia

D. A. W. JOHNSON, J. M. LUDLOW, K. STREET and R. D. W. TAYLOR

A double-blind controlled trial of 50% dose reduction in maintenance treatment in stable out-patients with low BPRS scores and good social function shows a significantly higher relapse rate in the low-dose group at 12 months ($P<0.05$). After an interval of 24–36 months from dose reduction, 56–76% had experienced a relapse and 76–79% had resumed their former dosage. No clear advantage was shown for the lower dose in either a reduction of side-effects or improved social function, but a reduced prevalence or lower rate of symptom emergence for tardive dyskinesia was suggested.

The value of long-term maintenance neuroleptic treatment in preventing psychotic relapse has been well demonstrated (Leff & Wing, 1971; Hogarty *et al*, 1973, 1974, 1976; Hirsch *et al*, 1973; Davis, 1975; Rifkin *et al*, 1977; Johnson, 1979, 1984; Schooler *et al*, 1980; Kane *et al*, 1982; Johnson *et al*, 1983), with the knowledge that responders should continue on regular medication for an unknown period in excess of 5 years. The longer-term experience with these drugs has made us more cautious, since they have the potential to produce unwanted side-effects such as extrapyramidal symptoms, tardive dyskinesia, weight gain, and akinesia. In all these unwanted effects, either the current dose or the total dose over time has been found to be a dependent variable. It is essential, therefore, to establish the lowest dose possible for the successful maintenance of patients, although the range is likely to be substantial, since no single dose will be correct for all patients.

Reviews of the actual regimens prescribed in controlled trials have led to the conclusion on theoretical grounds that much lower doses than commonly used might be equally effective (Baldessarini & Davis, 1980; Teicher & Baldessarini, 1985). In those controlled trials that permitted estimates of the equivalent dose of chlorpromazine to be plotted against reduction of relapse, no significant dosage effect in the range 100–2000 mg/day was found, and no mean difference in outcome at dosages above compared with dosages below 310 mg/day. It has also been demonstrated that some chronic patients can be successfully maintained on very small doses, relapsing when their drug is discontinued (Capstick, 1980).

In a trial of only 10% of the existing dose prescription of fluphenazine decanoate, Kane *et al* (1983) compared a low-dose group (1.25–5.0 mg per 2 weeks) with a standard-dose group (12.5–50.0 mg per 2 weeks) over 12 months, the subjects being out-patients. The relapse rate was significantly higher in the low-dose group (56% *v*. 7%). Kane (1984) subsequently studied a group of patients on 20% of the standard dose and reported a reduced relapse rate of 20% over 12 months. Marder *et al* (1984) reported a double-blind comparison of two groups of out-patients – a low-dose group on 20% of the previous dose, and a standard-dose group (5 mg *v*. 25 mg fluphenazine decanoate per 2 weeks). At the end of 12 months, the two doses appeared to be having equal effect. During the second year, the low-dose group became significantly disadvantaged, with a wide separation of the survival curves after 15 months. The standard-dose group had a negligible relapse rate in the second year, while the low-dose group had a fairly constant relapse rate throughout both years (31% *v*. 64% at 24 months). The apparent equal outcome at 12 months may be an artefact of the entry procedure into the trial, since most relapses in the standard-dose group occurred within the first 3 months. It is likely that some early relapses occurred before stabilisation on the standard-dose therapy had been established.

The low-dose studies all suggest that if relapse is the sole criterion of assessment, then standard-dose therapy is superior. However, suggestions have been made that the risks of side-effects may be less and the comfort and function of the patient improved by use of low doses. It has also been suggested that any relapse as a consequence of a reduced dose may have a less dramatic onset than relapse following complete withdrawal of medication, so remedial therapy may be offered. These practical advantages have not yet been established.

So far, there have been no studies investigating a dose reduction of less than 80%, nor any using the depot injection flupenthixol decanoate. The present study compares half dose and standard dose flupenthixol decanoate maintenance therapy over 12 months, and reports the 2–3 year outcomes of reduced medication.

Method

The sample was of consecutive out-patients who had been diagnosed as suffering from schizophrenia by their psychiatrist and who met the Feighner criteria (Feighner *et al*, 1972). All patients were on the normal treatment regimens used by their psychiatrist. In addition, they had to score no more than 3 on the total score of the Brief Psychiatric Rating Scale (BPRS) (Overall & Gorham, 1962), to have been maintained on a relatively low dose of flupenthixol decanoate over the last 12 months, and to have been on a stable dose of no more than 40 mg every two weeks for the past 6 months (in practice all patients had been on this stable dose for 12 months or longer before commencement of the trial). No patient was accepted with organic brain disease, physical illness, alcohol or substance abuse, below normal IQ, or additional mental illness. All patients had to give informed consent. Any patient might be withdrawn at his own request or at the discretion of the psychiatrist.

Patients fulfilling these criteria were allocated to one of two patient groups on the basis of a sealed computer randomisation. Group A continued on their pre-trial dosage for 12 months and then had their dose reduced to half. Group B had their pre-trial dosage reduced to half at the onset.

For both groups follow-up continued for three years. For the first 18 months, assessments every 3 months included administration of the BPRS and Clinical Global Impression (CGI) (Guy, 1976) by a psychiatrist; a side-effects checklist (Guy, 1976), the Extrapyramidal Rating Scale (EPRS) (Simpson *et al*, 1964), and the Abnormal Involuntary Movements Rating Scale (AIMS) (Guy, 1976) by a second psychiatrist; the CGI and EPRS by a nurse; and a Social Scale (Stevens, 1973) by a social worker. The assessors were blind to the evaluations of other assessors and all previous assessments of their own. All assessors were blind to the depot dosage throughout, as this was managed by a separate psychiatrist. From 18 months to 36 months, assessment intervals were increased to 6 months, and assessments were made by a single psychiatrist on mental state alone, using the BPRS and CGI. At the conclusion of the trial, the hospital notes and prescription sheets of all patients were examined. In addition, the patients were seen at any time at their request, or if changes were noted by any treating agency.

Relapse was defined as a definite increase in or re-emergence of symptoms resulting in either a CGI score of 3 (moderate severity) or an increase of the BPRS score of >3.

Wilcoxon's Matched Pairs Signed Ranks and Mann–Whitney *U* tests were used for statistical analysis throughout, with one exception, which has been identified, and on that occasion the chi-squared test with Yates' correction was used.

Results

Essential data was lost on one patient (group B (half dose)), so the analysis is based on the remaining 59 patients.

The demographic characteristics of the sample are presented in Table I. No significant differences were found between the two groups on entry into the study (demographic features, previous treatments, previous dose of drug, mental state, symptoms, or side-effects).

TABLE I
Demographic features of the patient sample

	Group A	*Group B*
Age: years		
Range	20–66	20–65
Mean	42	40
Sex		
Male	12	13
Female	19	15
Illness duration: years		
Range	2.5–29	2.5–26
Mean	11.2	10
Total time as in-patient: months		
Range	2–22	2–72
Mean	7.4	12.3
Total time on drugs: years		
Range	1.2–17	1.75–15
Mean	5.6	6.5

For the first 12 months, the doses in weekly equivalents were as follows:

Group A	range 4–20 mg;	mean 9 mg
Group B	range 1.7–10 mg;	mean 6 mg.

Analysis of the results for the first 12 months (Table II) showed that nine patients in group B (half dose) relapsed (one at 2 months, three at 3 months, and one at each of 4, 8, 9, 10, and 12 months), compared with three patients in the group A (full dose) (one at each of 4, 6, and 9 months); $P<0.05$ (chi-squared with Yates' correction, $\chi^2=4.11$).

In the survivors of the first 12 months, there were no differences in mental state. The scores on the CGI and BPRS were equal for all subscales and individual items. On the Social Scale the mean total fell during the 12 months in both groups, but the improvement for group B (half dose) was slightly greater (NS). In general this group showed a greater capacity for improvement, with more items showing

TABLE II
Mean total scores 0 months to 18 months

	0 months	*3 months*	*6 months*	*9 months*	*12 months*	*15 months*	*18 months*
Group A (full dose) (n = 31)							
Relapses*	0	0	2	1	0	1	0
BPRS	1.5	1.3	1.5	1.1	0.9	0.9	1.0
Psychiatrist's global assessments	0.452	0.323	0.379	0.310	0.143	0.269	0.231
Nurse's global assessment	0.323	0.355	0.276	0.276	0.071	0.308	0.231
EPRS	0.81	0.68	0.66	0.79	0.75	0.50	0.42
AIMS	1.87	1.68	2.48	1.83	1.79	1.31	1.04*
Stevens' Social Scale	2.87	1.83	1.83	1.76	1.25	1.54	1.77
Group B (half dose) (n = 28)							
Relapses*	0	4	1	2	2	1	2
BPRS	1.0	0.6	1.5	1.0	0.9	0.4	0.6
Psychiatrist's global assessment	0.250	0.292	0.304	0.182	0.211	0.111	0.067
Nurse's global assessment	0.143	0.250	0.217	0.091	0.158	0.056	0.133
EPRS	0.93	0.58	0.61	0.55	0.32	0.33	0.38
AIMS	1.75	1.50	1.91	1.18	1.16	0.94	0.40
Stevens' Social Scale	3.64	2.25*	2.22	1.59	1.05	0.83	1.07

*$P<0.05$ (Wilcoxon's Matched Pairs Signed Ranks and the Mann–Whitney *U* Test).

a reduction in score. Only the item 'relatives' anxiety' showed a significant difference, with the group B (half dose) having more improvement at 6 months and 9 months (mean change 0.24 *v.* 0.52 and 0.24 *v.* 0.55; $P<0.05$), but this group had slightly higher scores on entry (0.26 *v.* 0.50; $P=0.06$).

There were no significant differences in the first 12 months in the risks for extrapyramidal symptoms, tardive dyskinesia (TD) or other measures of side-effects (Table II). On the AIMS the mean total scores and individual item scores tended to fall in both groups. In group A (full dose), the number of patients with clinical TD remained constant at seven, but a reduction occurred in the group B (half dose) from nine to seven patients. On the EPRS the mean total score fell in group B (half dose) but remained constant in group A (full dose) (NS). Akathesia scores fell in both groups but tremor (0.23–0.36 *v.* 0.25–0.05), rigidity (0.00–0.14 *v.* 0.11–0.05) and hypokinesia (0.019–0.25 *v.* 0.21–0.11) increased in group A (full dose) and fell in group B (half dose) (NS). On the side-effects checklist both groups showed a reduction in total incidents (group A (full dose) 55%); (group B (half dose) 65%), with a considerable variation over time, but the patterns of individual side-effects were similar in both groups.

A comparison of the first 6 months on reduced dose for each group (group A, 12 months to 18 months; group B, 0 months to 6 months) revealed no significant differences between the groups in mental state, relapses, or extrapyramidal symptoms. However, in group A, who had been on a full dose 12 months longer than group B, there was a significant reduction ($P<0.05$) on the AIMS, compared with an initial reduction at 3 months (NS) followed by a slight increase at 6 months in group B (Table II). Significant differences were also noted in social function. Group A showed a slight trend for the total score to increase, with a particular increase in item 'dissatisfaction felt by next of kin' (mean change 0.269, $P<0.05$). In contrast, group B improved, with reduced scores at both 3 months and 6 months ($P<0.05$). On individual items, this group had a significant fall in 'relatives' anxiety' (mean change 0.52, $P<0.001$). The true significance of the differences in social function, particularly at the 5% level, must be questioned, since group A had a lower total score on entry (NS) and therefore a reduced capacity for improvement.

During the principal study, from 0 months to 18 months, when the full assessments were carried out, no correlations were found between dose and absolute score or change in score for mental state, social functions, or extrapyramidal side-effects in either group. For group A (full dose) from 0 months to 12 months, the AIMS scores correlated negatively with dose ($0.01<P<0.05$). This implies that the patients on lower doses had higher reductions in scores. From 12 months to 18 months (i.e. following dose reductions), the AIMS total scores for this group correlated positively with dose ($0.001<P<0.01$). This suggests that patients on the higher-dose regimens experienced the greatest reduction in symptoms. Collectively these changes suggest that the patients originally on the highest doses benefited most from having their doses halved.

There were no differences in the additional drugs prescribed to the two groups from 0 months to 18 months (anticholinergic drugs 9 *v.* 7; benzodiazepines 5 *v.* 4; anti-depressants 1 *v.* 1).

Ten patients had three or more assessments before relapse. There was a slight trend for the total BPRS scores to increase prior to relapse, with a general increase in the items of affect. The numbers of patients who experienced change with any one item was small, with depression (four patients) showing the most frequent change.

From 18 months to 36 months, assessments were restricted to the CGI and BPRS every 6 months, together with a review of all notes on completion of the study. Therefore it was possible during this period for patients to experience an adverse change in their mental state, requiring a change in treatment, without meeting the full criteria of relapse, since partial or complete remission may have taken place before the next review. At 36 months, group A (with 25 patients completing follow-up) had been on a reduced dose from 12 months to 36 months, and group B (with 23 patients completing follow-up) from 0 months to 36 months.

It was found that 56% of group A and 70% of group B had met the full criteria of relapse, and 76–78% of all patients had been prescribed an increase in their neuroleptic medication to at least the level of their former full dose.

Discussion

The principal comparison is the 12-month double-blind prospective comparison of a standard dose *v.* half-dose reduction. Even though this dose reduction was less than in previous studies, these results confirm that it is not possible to make a substantial dose reduction in maintenance therapy without a significant increase in the risk of relapse (10% *v.* 32% at 12 months). The longer prospective follow-up with slightly less strict monitoring reports a relapse rate of 56–70% after 2–3 years on the reduced dose, with three-quarters of the patients resuming their original full dose. The evaluations for group B at 18 months and for both groups at 36 months confirm the observations of Marder *et al* (1985) that 12 months' is too short a period of assessment for the real risk of increased relapse to be known. It is particularly important to note that the sample studied was of patients in a stable mental state with low BPRS scores and good social function on a moderate dose of medication.

It has been suggested that, despite an increased risk of relapse, there may be advantages for the use of low-dose maintenance therapy. Both Kane *et al* (1983) and Marder *et al* (1985) speculated that relapses might be milder and more readily controlled. In this study a prodromal development was suggested in both groups over 6 months, but it is unlikely that acute relapses can ever be regarded as 'mild', since the consequences of an acute relapse, particularly for stable, good-prognostic patients such as this sample, are more substantial than commonly recognised.

The previous studies noted either a lower prevalence of tardive dyskinesia or a lower rate of symptom emergence in the low-dose group. These results are also suggested by this study. Although there were no statistical differences in abnormal movement scores for over the period 0 months to 12 months in group B (half-dose), there was a slight reduction of TD at 3 months, followed by a slight increase at 6 months. In group A there was a significant fall in TD during the 6 months following half-dose reduction ($P<0.05$), with no evidence of a withdrawal rebound.

Suggestions have been made that patients may either have improved levels of social function or feel more comfortable on low-dose regimens. The results of the present study are complex and do not indicate a clear advantage with either dose prescription in social function among survivors. Side-effects, including extrapyramidal side-effects, were less on a reduced dose, but no analysis showed changes of statistical significance.

Within the dose ranges used, both at the standard and reduced dose level, no correlation was found between actual dose prescribed and any measurement of outcome or unwanted effects.

The results of this study demonstrate once again that low-dose strategies, even a modest 50% reduction, have an unacceptably high risk of relapse for schizophrenic patients in general. However, it is recognised that individual patients can occasionally survive on very low dose prescriptions. The only suggested advantage for a 50% reduction in dose was the possibility of a reduced risk of tardive dyskinesia. Perhaps patients who either already have TD, or have been identified as being particularly at risk, might be placed on low-dose prescriptions, provided that they are fully aware of all the considerations. At this level of dose reduction, the previously claimed advantages of improved patient comfort and function were not evident.

Previous studies have suggested that for stable out-patients the standard dose of fluphenazine decanoate (Kane *et al*, 1983; Marder *et al*, 1985) is in the range 12.5–25 mg every 2 weeks (or equivalent). This study suggests that, for similar patients, 20–40 mg flupenthixol decanoate every 2 weeks (or equivalent) is the likely range.

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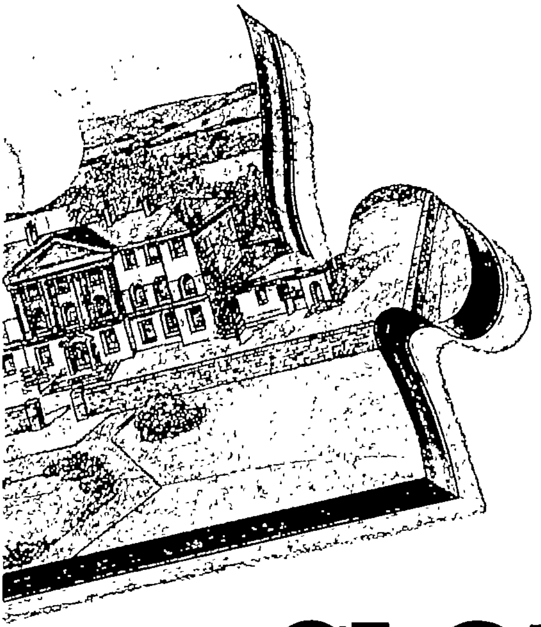

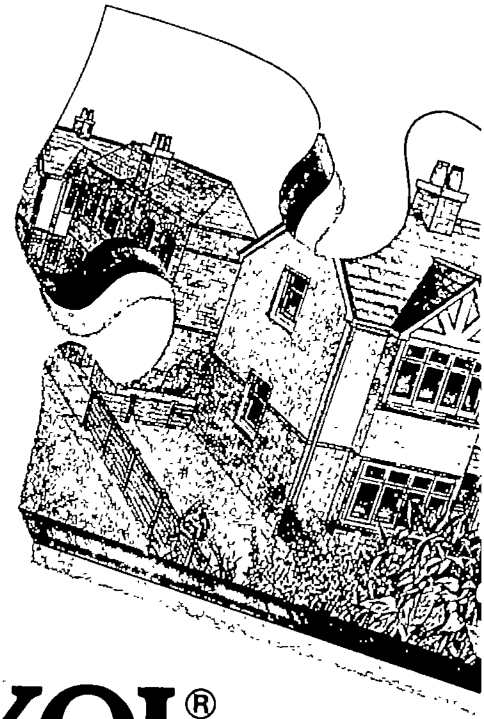

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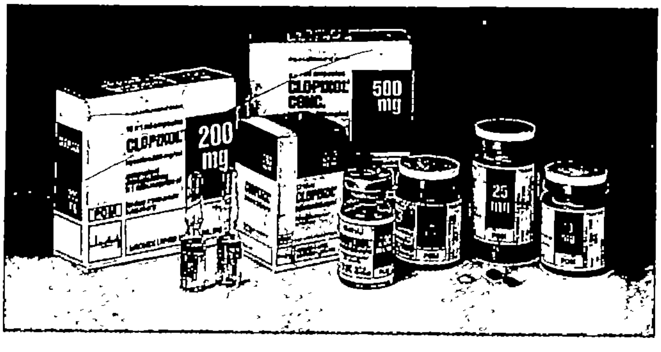

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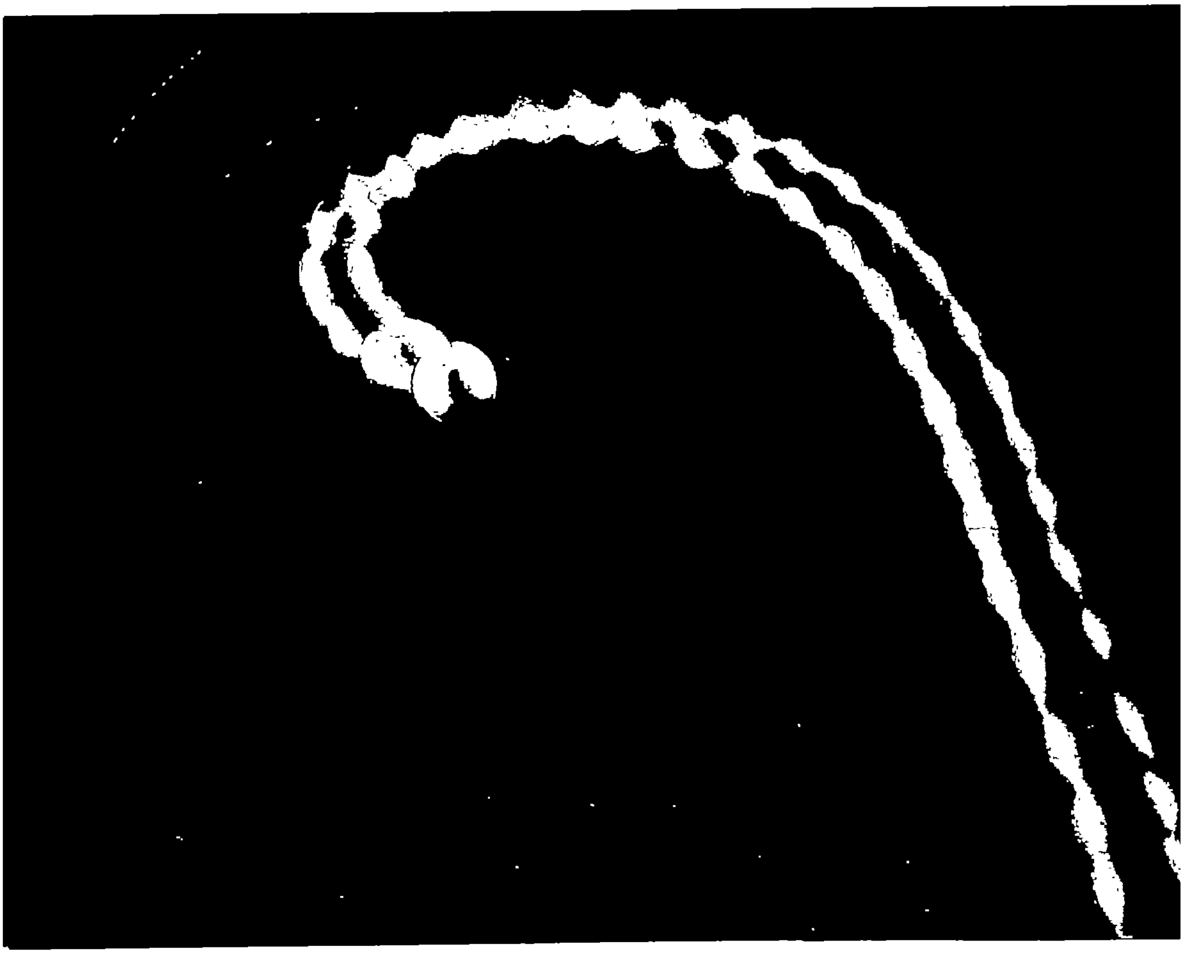

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Double-Blind Study of Imipramine versus Phenelzine in Melancholias and Dysthymic Disorders

J. VALLEJO, C. GASTO, R. CATALAN and M. SALAMERO

In a 6-week double-blind trial, the efficacy of imipramine and high dose phenelzine were compared in the treatment of Major Depression with Melancholia, and Dysthymic Disorder (DSM-III). Both drugs were found to be equally efficacious in the treatment of 32 patients suffering from Major Depression. In 32 Dysthymic patients, phenelzine in high doses was found to be superior to imipramine. The clinical implications of these findings are discussed.

In the treatment of the endogenous depressions, the efficacy of the monoaminoxidase inhibitors (MAOIs) (Rees & Davies, 1961; Hamilton, 1982; Medical Research Council, 1965) has been less well established than that of the tricyclic antidepressants (TADs) (Klein & Davies, 1969; Morris & Beck, 1974).

In the case of the so-called neurotic and atypical depressions, the efficacy of pharmacotherapy is less clear. Earlier evidence in favour of the MAOIs (West & Daly, 1959; Robinson *et al*, 1973) has been qualified by more recent work showing that comparative results can be obtained with TADs (Nies & Robinson, 1981; Rowan *et al*, 1982). In one study, however, phenelzine was shown to be superior to imipramine (Liebowiz *et al*, 1984).

This paper reports a double-blind trial comparing imipramine and phenelzine in the treatment of DSM-III-diagnosed Major Depressions with Melancholia, and Dysthymic Disorders.

Method

Subjects

Selection of 83 subjects from a population of 116 unmedicated out-patients was made by two psychiatrists according to the following entry criteria: (a) age range 18–63; (b) diagnosis of Major Depression Episode with Melancholia, or Dysthymic Disorder (DSM-III); (c) absence of severe physical disease, on-going medical treatment and pregnancy; (d) absence of psychopathic/sociopathic disorder; Briquet's syndrome; alcohol/drug abuse; psychotic illness and bipolar, obsessive–compulsive, somatoform, panic, eating, and phobic disorder (DSM-III); (e) score of >16 in the Hamilton Rating Scale for Depression; and (f) ability to give real consent.

The 83 patients were assessed further and given placebo medication for a week. Ten improved and were excluded; the remaining 73 (melancholia, 34; dysthymic disorder, 39) entered the study. Each clinical group was then randomly allocated to imipramine or phenelzine in a double-blind 6-week design. Medication and placebo was given in tablets of identical appearance.

The following daily drug protocol was used: first week, imipramine 100 mg or phenelzine 30 mg; second week, imipramine 150 mg or phenelzine 45 mg; third week, imipramine 200 mg or phenelzine 60 mg; fourth to sixth weeks, imipramine 250 mg or phenelzine 75 mg.

All patients were asked to observe the MAOIs food precautions. Medication was given at breakfast and lunch time. No patient was allowed to take other medication except ten who required diazepam (5 mg *nocte*) for sleep disorder.

The patients were rated weekly for side-effects and compliance. During these contacts their medication was dispensed in seven small boxes. At the end of the trial, all patients were retested.

Instruments

The following rating scales were used: (a) Hamilton Rating Scale for Depression (HRSD); (b) Zung Self-Rating Depression Scale (in the Spanish translation by Conde) (ZDS); (c) Eysenck Personality Inventory (EPI-B).

Neuro-endocrinological tests

Dexamethasone suppression test (DST) was performed on all patients. Plasma cortisol was estimated at 4 p.m. after the administration of dexamethasone (1 mg) the night before at 11.30 p.m.; exclusion criteria as specified by Carroll *et al* (1981) were used.

Statistical analysis

Data were analysed by means of Student's *t*-test, chi-squared or Fisher's Exact Test. Patients in each clinical group were randomly subdivided and the coefficients of variability (Snedecor's Test) were compared to ascertain that there were no significant differences.

Results

Of the 73 patients who entered the drug trial, nine dropped out – seven from the Dysthymic Disorder group (four on

TABLE I
The two clinical groups compared

Characteristics	*Melancholic group (n = 32)*	*Dysthymic group (n = 32)*	*t or χ^2*	*Significance*
Sex				
Women	24 (75.10%)	28 (87.5%)		
Men	8 (25.0%)	4 (12.5%)	$\chi^2 = 1.641$	NS
Mean age: years	44.3 (s.d. 10.3)	40.2 (s.d. 11.5)	$t = 1.506$	NS
Socio-economic status				
Lowest	22 (68.8%)	24 (75.0%)		
Middle	8 (25.0%)	8 (25.0%)		
Highest	2 (6.3%)	0 (0.0%)	$\chi^2 = 0.309$	NS
Mean scores				
HRSD	25.1 (s.d. 5.2)	20.5 (s.d. 4.0)	$t = 3.937$	0.01
ZDS	57.4 (s.d. 9.0)	57.5 (s.d. 6.4)	$t = 0.046$	NS
EPI neuroticism	15.9 (s.d. 3.1)	15.7 (s.d. 4.3)	$t = 0.233$	NS
EPI extroversion	8.5 (s.d. 2.8)	6.8 (s.d. 3.4)	$t = 2.149$	0.05
Duration of disorder: months	8.0 (s.d. 1.8)	36.6 (s.d. 4.1)	$t = 36.131$	0.001
DST				
Suppression	19 (59.4%)	31 (96.9%)		
Non-suppressors	13 (40.6%)	1 (3.1%)		$P = 0.0002$

imipramine and three on phenelzine) and two from the Major Depression with Melancholia group (one on imipramine and one on phenelzine). The reasons were that one Major Depression with Melancholia patient (on imipramine) developed a delusional syndrome and had to be admitted for ECT; and the other eight patients abandoned the trial on account of side-effects (mainly hypotension). No significant differences were found on any variable between the drop-outs and the patients completing the study. The final sample consisted of 32 patients with Major Depression with Melancholia and 32 with Dysthymic Disorder.

The melancholic and dysthymic groups could not be differentiated in terms of sex, age, social class, ZDS or EPI neuroticism scale (Table I). The melancholic sample, however, showed higher mean scores on the HRSD (25.06/20.53) and the EPI extroversion scale (8.5/6.8). It also contained more DST non-suppressors (40.6%/3.1%). However, the duration of current episodes was longer for the Dysthymic Disorder group (36.6/8.0 months).

The groups differed significantly on ten HRSD items: depressive mood, early awakening, work and activities, retardation, agitation, gastrointestinal somatic symptoms, loss of weight, insight, diurnal variation of mood, and obsessive and compulsive symptoms. They also differed on two ZDS items: improvement in the evening and loss of appetite.

Imipramine versus phenelzine in Major Depression with Melancholia

For the Major Depression with Melancholia patients, comparison between those on imipramine (16) and those on phenelzine (16) showed no difference on any variable except sex ($P < 0.018$) (there was only one male in the imipramine group as against seven in the phenelzine group). Likewise the two groups showed no difference on final assessment (Table II). The variance of HRSD scores in the imipramine group was significantly smaller than in the phenelzine group ($F = 2.555$, $P < 0.05$). There was a tendency for the HRSD scores to be lower in the imipramine group than in the phenelzine group, but the difference did not achieve statistical significance.

The effect of imipramine was more marked on the following HRSD items: suicide ($P < 0.03$), retardation ($P < 0.005$), hypochondriasis ($P < 0.03$) and diurnal variation of mood ($P < 0.03$).

Imipramine versus Phenelzine in Dysthymic Disorder

Among the Dysthymic Disorder patients, no significant difference was found between those on phenelzine (16) and those on imipramine (16). Significant differences were found on final assessment on all rating scales except EPI extroversion (Table III): the phenelzine group had lower scores on the HRSD ($P < 0.05$), ZDS ($P < 0.01$) and EPI (neuroticism scale) ($P < 0.02$). HRSD item analysis showed imipramine to improve depressive mood ($P < 0.02$) and phenelzine to enhance work and activities ($P < 0.07$).

Discussion

Our data may help to solve earlier controversies. While the satisfactory response to both antidepressant drugs shown by the melancholic group is not surprising, it is of interest that the dysthymic group behaved to a certain extent in the same way.

TABLE II
Major Depression with Melancholia patients: drug subgroups compared (post-treatment values)

Variables	*Imipramine subgroup*		*Phenelzine subgroup*		*t*	*Significance*
	Mean	*s.d.*	*Mean*	*s.d.*		
HRSD	7.06	5.48	10.63	8.76	1.382	NS
ZDS	35.69	9.65	38.69	11.85	0.785	NS
EPI neuroticism	12.94	2.77	13.75	3.53	0.722	NS
EPI extroversion	10.31	3.40	10.19	3.02	0.106	NS

TABLE III
Dysthymic Disorder patients: drug subgroups compared (post-treatment values)

Variables	*Imipramine subgroup*		*Phenelzine subgroup*		*t*	*Significance*
	Mean	*s.d.*	*Mean*	*s.d.*		
HRSD	10.44	5.10	7.25	3.17	2.125	0.05
ZDS	43.06	9.02	34.88	7.40	2.804	0.01
EPI neuroticism	15.38	4.11	11.75	3.53	2.680	0.02
EPI extroversion	7.75	3.55	10.63	4.61	1.980	NS

It could be argued that this similar response by the two clinical groups reflects the fact that our sample of Dysthymic Disorder was 'overpurified' by the exclusion of subjects suffering from sociopathy, obsessive-compulsive disorders, anxiety, phobia and hysteria. This view can be accepted without reducing the value of our findings, and it can be concluded that our results apply only to a specific subpopulation of Dysthymic Disorder.

A technical objection to our study may be that it includes no placebo group. The decision to do so was taken on three grounds. First, imipramine and phenelzine are well-tried antidepressants. Secondly, the objective of the trial was not to show that these drugs were superior to placebo. Thirdly, all patients had already received a placebo during the selection week. Furthermore, compliance was strictly controlled.

The Major Depression with Melancholia group

The Major Depression with Melancholia patients responded equally well to both drugs. HRSD symptom reduction in the imipramine group was 71.4% and in the phenelzine group 58.2%, and ZDS symptom reduction was 37.7% and 31.8% respectively. The smaller difference detected by the ZDS (5.9% as opposed to 13.2% in the HRSD) suggests that it is a less sensitive instrument than the HRSD; on the other hand, it may simply result from the fact that ZDS scores depend upon patient self-scrutiny and not, like the HRSD, on objective clinical assessment. The standard deviation for the HRSD scores was smaller in the imipramine group; this reduced scatter may reflect a more homogeneous action by this drug.

The difference in terms of phenelzine efficacy between the present trial and the Medical Research Council trial (1965) is explained by the fact that our sample included neither patients with bipolar disorders nor psychotic depressives, and by the fact that our patients received higher drug dosage (75 mg daily) than those in the MRC trial (60 mg); in this respect our results coincide with those of Rees & Davies (1961) and Hamilton (1982), who used high dosage.

It can be concluded that Major Depression with Melancholia without psychotic symptoms responds equally well to imipramine and phenelzine, provided that high doses of the latter are used. Phenelzine should be considered as a good alternative to tricyclic antidepressants in cases when these drugs are contraindicated, cause major side-effects, or prove ineffective.

The Dysthymic Disorder group

In relation to Dysthymic Disorder patients, previous studies have tended to show MAOIs to be less effective (Young *et al*, 1979) or at best to have similar therapeutic action to TADs (e.g. amitriptyline *v*. phenelzine) (Nies & Robinson, 1981; Rowan *et al*, 1982; Paykel *et al*, 1982).

Our study shows phenelzine to be significantly superior. The standard deviation for the HRSD scores was smaller in the phenelzine group; this reduced scatter may reflect a more homogeneous action by this drug. However, the fact that imipramine also reduced symptom scores (50.3% in HRSD and 25.3% in ZDS) suggests that TADs are an adequate alternative when MAOIs prove ineffective or cause major side-effects. It is also of some interest to note the effect of phenelzine on the neuroticism (19.1%) and extraversion scores (47.2%) of the EPI. This suggests that these scores are contaminated by mental state.

The clear-cut nature of the results obtained in this study can be explained, firstly, by the fact that patient selection was strict (this also may explain the divergence between our results and those of Paykel *et al* (1982)); and secondly by the fact that our patients received a high dose of phenelzine. Higher doses may also explain the positive findings of Liebowitz *et al* (1984) (in panic disorder and hysterical dysphoria) when compared with those of Nies & Robinson (1981), who used lower doses.

It can be concluded, therefore, that in the treatment of Dysthymic Disorders, differences in favour of phenelzine appear only when high doses are employed, and care is taken to exclude psychopathic and hysterical personalities.

HRSD item analysis shows imipramine to have more effect on depressive mood and phenelzine more on work activity. In contrast to other studies (Nies & Robinson, 1981; Rowan *et al*, 1982; Paykel *et al*, 1982), we did not find any specific action of phenelzine on anxiety, although the scores on the EPI may show some trend in this direction. It is possible that the use of specific anxiety scales might have provided us with better data on this point.

The final conclusion of this study must be, therefore, that imipramine and phenelzine are effective in Major Depression with Melancholia and Dysthymic Disorder; and that phenelzine is more effective in the latter.

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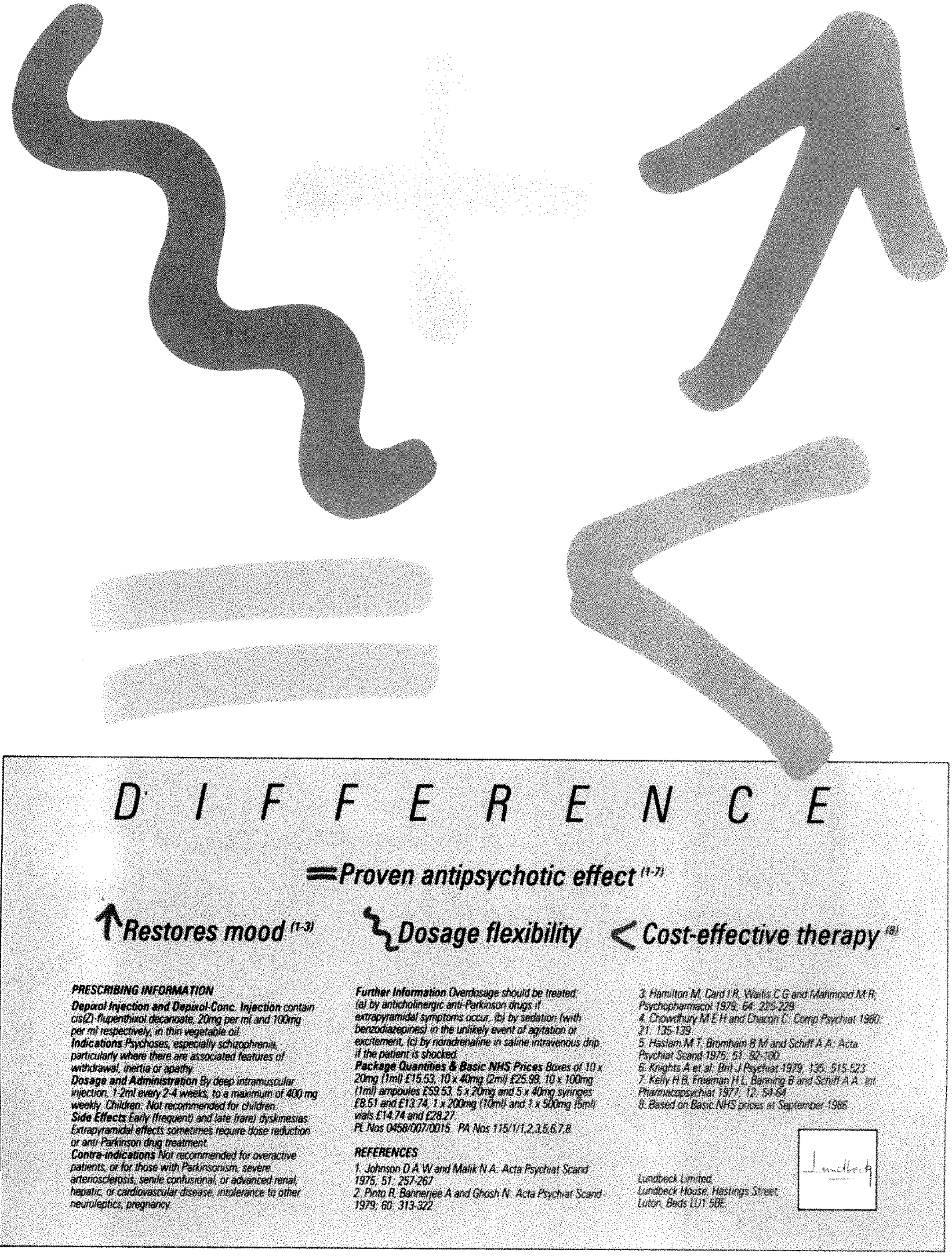

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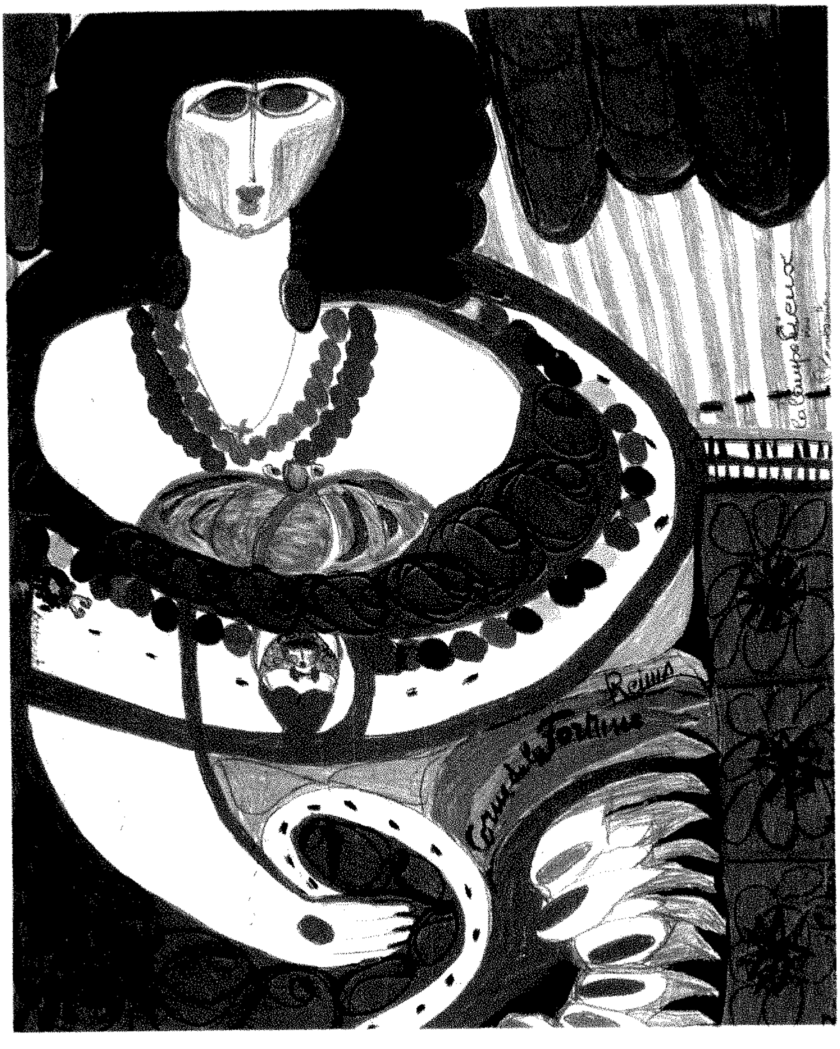

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Self-Appraisal, Anxiety and Depression in Women

A Prospective Enquiry

J. G. INGHAM, N. B. KREITMAN, P. McC. MILLER, S. P. SASHIDHARAN and P. G. SURTEES

Depressive illness is known to be associated with low self-evaluation, but it has been suggested that there may be a reciprocal connection as well, such that low self-appraisal (in the absence of illness) makes the subsequent onset of depression more likely. A prospective study, using a community sample of 376 women, provided data about clinical state over a period of 18 months, and self-appraisal questionnaire scores were determined on two occasions separated by 6 months. There was no evidence that low self-evaluation predicted future episodes of depressive illness, except in women who reported previous psychological episodes for which they had sought medical help, and, even for those with previous episodes, much of the predictive power of low self-esteem was accounted for by individuals who were subsequently recognised to have been in the early stages of illness. Conversely, there was little evidence that prior episodes predicted future illness in people with high self-esteem. One explanation of the findings is that recurrent episodes of illness cause progressive impairment of self-appraisal, but other possibilities are also considered. Women who had recovered from illnesses detected at the first interview still had significantly less self-confidence 6 months later than those who were well throughout.

The link between negative self-appraisal and affective disorder is well known and documented. What is not clear, however, is the extent to which negative self-appraisals such as low self-esteem play any causal role in the genesis of illness. A recent study of self-esteem and self-confidence in women presented evidence of the influence of some social factors on self-esteem in the absence of illness (Ingham *et al*, 1986). These factors included the lack of a close confidant, and early separation from the mother, both of which are among the 'vulnerability' factors postulated by Brown & Harris (1978) as predisposing towards depressive illness. These findings may be taken as a partial confirmation of the latter authors' suggestion that impaired self-evaluation was the common intervening variable between their vulnerability factors and depression. However, although this cross-sectional study provided some support for the Brown & Harris hypothesis, the effect of the social variables on self-esteem was very small when compared with the effect of major depression itself.

Moreover, the association between self-esteem and vulnerability factors may have nothing to do with the causation of depression. There is a link missing from the chain of reasoning. The theory clearly states that low self-esteem existing beforehand will lead to the onset of illness, given other appropriate circumstances. It is thus necessary to establish that low self-esteem has some predictive power. There has been little previous work on self-esteem as a predictor, and what has been done is not very encouraging (Lewinsohn *et al*, 1981).

The theory also implies that low self-regard persists in depressed patients after recovery from illness. The main weight of evidence indicates a return to normal levels (Mayo, 1967; Caine, 1970; Hamilton & Abramson, 1983) unless the follow-up occurred soon after recovery (Foulds, 1976).

While analysing the cross-sectional data we noticed that reports were appearing indicating that it is the presence of negative self-evaluation that is linked with vulnerability to depression, rather than the absence of positive self-evaluation (Brown & Bifulco, 1985; Warr & Jackson, 1983; Parry, personal communication). The usefulness of the distinction was confirmed in our own cross-sectional study, and has been maintained in the present enquiry.

Aims

The aim of the present study was to test predictions from the Brown & Harris theory by a follow-up of cases and non-cases derived from a general population survey. Initial assessments were obtained for all those who were well to see whether those who subsequently became ill had significantly lower self-appraisal beforehand. Similarly, those who had been cases at the outset were re-assessed to determine whether they maintained their relatively low self-evaluation even after recovery.

There was also an obvious opportunity to confirm previous evidence, already strong, that new onsets of either anxiety or depression result in a decline in self-evaluation.

Thus the hypotheses to be formally tested were as follows:

Hypothesis 1. Women who are not cases but who are low in self-appraisal are more likely to become depressed subsequently.

Hypothesis 2. Women who have recovered from an affective disorder (depressive illness or anxiety state) have lower self-appraisal than those who have not been ill.

Hypothesis 3. Women who become ill with anxiety or depression suffer a decrease in self-appraisal.

For testing hypothesis 1, two self-appraisal scales (see below) were used and the same strict criterion was at first applied as was used in the cross-sectional study (Ingham *et al*, 1986). This was that both scales should show the predicted difference between means at the 5% confidence level. As will be seen, the criterion was later relaxed for further exploration of the data. The standard scales, and the sub-scales comprising negative and positive items, were considered separately. For testing hypotheses 2 and 3, only one scale was available and the usual 5% criterion was applied.

Method

Design

As already indicated, the study is a follow-up of a sub-sample of the women who had taken part in a cross-sectional survey of affective disorders reported in several earlier publications (Dean *et al*, 1983; Surtees *et al*, 1983; Ingham *et al*, 1986). The sample selected for follow-up comprised:

(a) all those who had been diagnosed as cases on either the Research Diagnostic Criteria or Present State Examination criteria for current psychiatric disorder
(b) all those who had one or more of a set of factors that were thought to be possible predictors of vulnerability to affective disorder
(c) a 40% random sample of the remaining women who were neither ill nor had any of the vulnerability factors.

The vulnerability factors were lack of close social support, lack of diffuse social support, poor relationship with one or more household members, living with three or more children aged under 14, living alone, loss of a parent before the age of 11, unemployment of spouse or cohabitee, previous contact with health services for 'nerves', and low self-confidence.

An important component of the research was the identification of women who were not 'cases' at the first assessment but who became ill at any time during the follow-up, which always extended for 6 months and generally for a full year after the initial assessment. A total of 415 women (cases and non-cases) were selected at the start of the study. At the 6-month point, 376 (91%) were successfully re-interviewed, and of these all but 43 were seen at 12 months as well. Details of the initial sampling are contained in the earlier publications. Data obtained at each interview were sometimes incomplete, and for this reason the tabulations show slight differences in sample size depending on which variables are being analysed. For the same reason there may also be slight discrepancies between the numbers reported here and those in other published tables concerning different aspects of the same survey. Similarly, other analyses, not reported here, have included nine women interviewed at the second follow-up but not at the first.

Interview

All the interviews were conducted by a team of interviewers specially selected and trained to undertake the initial survey (for further information see Surtees *et al*, 1983; Ingham *et al*, 1986). There was a further short period of training before the first follow-up interview. As far as possible interviewers followed up respondents they had seen before, but this was not always practicable and approximately 40% of the respondents were seen by a different interviewer.

Psychiatric assessment schedule

The psychiatric assessment schedule comprised the first 40 items of the Present State Examination (PSE) and additional questions from the Schedule of Affective Disorders and Schizophrenia (SADS) so that CATEGO and Research Diagnostic Criteria (RDC) could be applied (Wing *et al*, 1974; Spitzer *et al*, 1978). Where appropriate, and in particular when there was evidence of anxiety or depression, a standard procedure was used to determine the times of onset and remission of symptoms as accurately as possible. For determining current psychiatric status at each interview the normal PSE procedure was used, enquiring over the past month, but in addition enquiries were made which covered the 6 months preceding each interview (Surtees *et al*, 1986*b*). A final assessment, using all three schedules (in some cases only two were available), was then made in which dates of onset and remission were determined for each episode of illness throughout the period of study, together with its diagnosis. For each interview point, therefore, it was possible to define psychiatric status either as it was seen at that time or, with hindsight, as finally revised. The two were not always identical and we have used them both in ways that will be indicated. Other choices could have been made but we do not believe our conclusions would have been altered materially, except in the one instance where both analyses have been presented.

Self-ratings of anxiety and depression

For self-rating of anxiety and depression, visual analogue scales were used requiring each subject to mark her position on a 10 cm line representing severity. They have been described more fully by Miller & Ingham (1979).

Assessment of self-appraisal

The first interview included two widely used scales of self-appraisal, the Rosenberg Low Self-Esteem Scale (Rosenberg, 1965) and the Foulds and Bedford Lack of Self-Confidence Scale (Bedford & Foulds, 1978). A modification was introduced into the Rosenberg scale, to increase the likelihood that it would measure a relatively long-term characteristic rather than be over-influenced by short-term mood states. To this end the words "most of my life" were inserted into each of the statements in the scale, making it consistent with the Lack of Self-Confidence Scale.

The overall score from each scale was calculated as prescribed by its originator. Rosenberg used combinations of items that he claimed formed a Guttman scale; we used a simple sum of these combined items. The scales have been described in more detail by Ingham *et al* (1986).

Each scale had an equal number of positively and negatively worded items and it was a simple matter to split them into separate scales. An example of a positively worded item from the Rosenberg scale is

Most of my life, on the whole, I have been satisfied with myself

Strongly agree Agree Disagree Strongly disagree

A negatively worded item is

At times during most of my life I have thought I was no good at all

Strongly agree Agree Disagree Strongly disagree

Items were allotted scores of 1 to 4. The positive score was the sum of all five positive items, giving a range of 5 to 20, and similarly for the negative scale. Since a high score on the positive scale indicates low self-esteem, the scores on the negative scale were reflected for comparability.

An example of a positively worded item from the Foulds–Bedford scale is

Most of my life I have felt as capable as other people

Never Seldom Often Nearly always

A negatively worded item is

Most of my life I have been very unsure of myself

Never Seldom Often Nearly always

Again scores of 1 to 4 were allotted; each scale comprised three items, giving a range of 3 to 12. In this case a high score on the negative scale indicates low self-confidence, so the positive scale was reflected. Thus a high score on any of the scales tabulated indicates low self-appraisal.

After the first interview a decision was taken to shorten subsequent interviews, so the Rosenberg scale was dropped. As mentioned above, this meant that only one scale was available for testing hypothesis 2 and 3, and the usual 5% criterion was applied. The main reason for retaining the Foulds–Bedford scale in preference to the self-esteem scale was for comparability with other Edinburgh studies.

Results

Hypothesis 1

"Women who are not cases but who are low in self-appraisal are more likely to become depressed subsequently."

This is a prospective hypothesis predicting from information available at the first interview.

An initial problem arose concerning the status of a group of 11 individuals not initially recognised as cases who were subsequently reclassified, using information obtained at later interviews, as cases having onsets before the first interview. In nine of these there was evidence at the first interview that anxiety or depression was present during the previous month but symptoms were insufficient, in number, severity or duration, to meet the case criteria; in the other two, symptoms were not elicited at the time but the later data were judged to be more dependable. A strictly prospective position requires us to use a classification based only upon information available initially in selecting the sample of non-cases on which to make predictions. It can also be argued, however, that the 11 doubtful individuals should be excluded from the sample of non-cases on the grounds that they were subsequently shown to have been ill. This matter has been referred to in two earlier publications dealing with different aspects of the same study (Surtees *et al*, 1986*a*,*b*). We shall first adopt the strictly prospective position and then demonstrate the effect of taking the alternative viewpoint.

Table I gives the means and standard deviations, on the self-assessment scales at the time of the first interview, of respondents who were not then recognised as 'cases'. It compares those who became cases during the 12-month follow-up (including the 11 'doubtfuls'), subdivided by RDC diagnosis, with those who did not. There is a clear tendency for those who became cases to have had lower self-esteem or self-confidence at the time of the first interview. Analysis of variance produced significant *F*-ratios for the Rosenberg scale (*P*-values are shown in the table), but by the *a priori* criterion (which requires significant differences on both the Rosenberg and Foulds–Bedford scales), hypothesis 1 is not verified, either for combined or for separate sub-scales.

The analysis could end at this point but it is unwise to dismiss the hypothesis entirely. As all the mean differences are directionally consistent with the hypothesis, and the *F*-ratios for the Rosenberg standard and negative forms are statistically significant, further exploration of the data is suggested. Deviant scores were most marked for those who became major depressives, and separate *t*-tests showed that even on the two Rosenberg scales, it was only they who differed significantly from the women who remained free from illness. There were no significant differences in the scales based on positively worded items, but the future major depressives had larger variances on all six scales. *F*-ratios showed that the variances on each measure were significantly larger for this group than for those with no new episodes. This is a complicating factor in the analysis. If it is taken into account, the significant *t*-values for Rosenberg standard and negative scales are reduced, and remain significant with only a 1-tail test. However, the

TABLE I

Self-assessment scores at first interview, excluding people who were cases at first interview

(a) Rosenberg scale

	Standard form			*Negative items*			*Positive items*		
	n	*Mean*	*s.d.*	*n*	*Mean*	*s.d.*	*n*	*Mean*	*s.d.*
No new episode	266[1]	1.18	1.45	267	10.02	2.59	266	9.82	1.79
New case of anxiety or minor depression	20	1.50	1.54	20	10.65	2.35	20	9.60	1.90
New case of major depression	23	2.04	2.03	23	11.48	4.03	23	10.30	2.38
F-ratio		3.7			3.4			0.9	
P		0.03			0.03			NS	

(b) Foulds–Bedford scale

	Standard form			*Negative items*			*Positive items*		
	n	*Mean*	*s.d.*	*n*	*Mean*	*s.d.*	*n*	*Mean*	*s.d.*
No new episode	267	10.82	2.61	268	5.84	1.57	267	4.98	1.49
New case of anxiety or minor depression	20	10.65	2.06	20	5.95	1.28	20	4.70	1.08
New case of major depression	23	11.96	3.83	23	6.39	2.21	23	5.57	2.17
F-ratio		2.0			1.3			2.0	
P		NS			NS			NS	

1. Slight variations in sample size result from missing values.

difference in variance is itself an interesting finding, which tends to support the hypothesis.

Thus overall the evidence so far is of some limited support which falls short of our preset level of statistical significance for hypothesis 1. Negative self-appraisal does appear to contribute something to the onset of depressive illness, although it is not a spectacularly successful predictor. In so far as it is operative at all, it remains to be determined whether negative self-regard might be better viewed as a reflection of some other predictive variable.

Emotional state at first interview

It is possible to hold a continuum concept of psychiatric illness. From this point of view, some of the people who were not recognised as cases at interview 1 may nevertheless have had an increased level of distress placing them some way towards the arbitrary threshold discriminating cases from non-cases. The visual analogue rating scales of anxiety and depression provided some evidence on this point. On both scales, the mean scores of those who were subsequently to become cases were significantly elevated. If marked negative self-appraisal is only found in women who are already on the way to becoming diagnosable cases, the meaning of hypothesis 1 is markedly altered. However, analysis of covariance, adjusting for variations in both anxiety and depression scales, did not alter the findings materially and the significant variations shown in part (a) of Table I remained.

So far, the 11 individuals who were not recognised as cases initially but were later reclassified have been included among the new onsets. The data were next re-analysed from the alternative viewpoint, excluding the 11, leaving 299 of the 310 respondents. The differences in standard and negative Rosenberg scales then became non-significant. The 11 doubtful individuals can be regarded as sub-clinical at the first interview, and their self-appraisal at that time could be part of the incipient illness.

Effect of prior medical contacts

'Vulnerability', as defined by the presence of any one of the seven factors stated, had been incorporated into the design to increase the likelihood of detecting new-onset cases. In the event, this was largely unsuccessful, as the 'vulnerable' sample did not produce a significantly higher proportion of new cases than the remainder (Surtees *et al*, 1986*b*). However, one group was selected because of previous contact with health services for 'nerves', and there was a strong indication that they did have a higher proportion of new onsets. The implications of this for the interpretation of Table I were examined by logistic regression analysis (Baker & Nelder, 1978), a technique which permits examination of the separate components in a multivariate analysis when the outcome is classified in simple categories. It was only possible to consider outcome (the response variate) in binary form, and since the link between self-appraisal and onset was clearly established only for major depression (Table I), the division was fixed accordingly to separate new onsets of major depression from the rest of the sample. Self-appraisal was taken as one predictor and previous contact with the health services for 'nerves' was another. The results are shown in Table II and also for Rosenberg standard and negative scales in Table III with the 11 sub-clinical cases excluded. In the column headed 'unadjusted' are scaled deviances of new

TABLE II

Prediction of 23 new onsets of major depression in 310 subjects not recognised as cases at first interview

	Scaled deviance (d.f. in brackets)		
	Unadjusted	*Adjusted*	*Interaction with health service contact*
Health service contact	4.0(2)	2.3(2)	—
Rosenberg scale			
Standard form	5.6(1)**	3.9(1)*	1.9(2)
Negative items	5.6(1)**	3.9(1)*	8.6(2)**
Positive items	1.5(1)	0.8(1)	0.3(2)
Foulds–Bedford scale			
Standard form	3.5(1)	2.4(1)	1.2(2)
Negative items	2.4(1)	1.4(1)	1.8(2)
Positive items	3.1(1)	2.4(1)	1.0(2)

*$P<0.05$; **$P<0.02$

TABLE III

Prediction of 16 new onsets of major depression in 299 subjects not recognised as cases at first interview (excluding 11 who were sub-clinical)

	Scaled deviance (d.f. in brackets)		
	Unadjusted	*Adjusted*	*Interaction with health service contact*
Health service contact	8.0(2)**	6.3(2)*	—
Rosenberg scale			
Standard form	2.8(1)	1.3(1)	0.0(2)
Negative items	3.5(1)	1.8(1)	5.2(2)

*$P<0.05$; **$P<0.02$

onsets accounted for by each factor taken separately and ignoring all other factors. In the next column, previous contact has been adjusted for Rosenberg negative scale; reciprocally each of the self-appraisal measures has been adjusted for previous contact. The scaled deviances are distributed as χ^2 with degrees of freedom shown in the tables.

The results in Table II confirm those in Table I and show, furthermore, that the standard and negative Rosenberg scales retain their predictive power even after adjustment for previous contact. For the negative scale, it is the adjusted interaction term that is most striking. Table III shows again the lack of a significant self-appraisal effect with the 11 excluded, and the predictive power of prior health service contact shows up very clearly. The nature of the effects that underlie the pattern shown in Tables II and III is further demonstrated in Tables IV and V, in which the Rosenberg negative scale has been divided arbitrarily into three categories. Negative self-appraisal is associated with previous medical consultations for nervous disorders ($\chi^2 = 8.86$, d.f. 2, $P<0.02$). The significant interaction effect with respect to onset of illness shown in Table II is evident from Table IV, indicating that the negative scale predicts new onsets in those who have had previous consultations but does not do so for those with no prior treated episodes. Similarly, prior contact predicts illness only in those tending to low self-esteem. The effect is reduced, however, when the 11 sub-clinical cases are excluded from the sample.

In all these analyses we have distinguished three categories of previous health service contact, as shown in the tables, but it appears that it is previous psychiatric consultation which is the more powerful predictor of new episodes. If the 11 sub-clinical cases are excluded, as in Table V, it is the only significant predictor, and the effect of GP contact becomes non-significant. As a *post hoc* exercise, the analysis in Table II was repeated, using only two categories for prior contact – those with and without a history of consultation with a psychiatrist – and it was then found that the effect of negative self-esteem was significant irrespective of treatment status (the interaction term became non-significant). This result should be interpreted with extreme caution pending replication, and it remains the case that there is no evidence that low self-esteem predicts new onsets

TABLE IV

New onsets of major depression (23 in 310 subjects) in different categories of self-appraisal (Rosenberg negative items) and health service contact for 'nerves'

Health service contact	*Rosenberg negative items*			
	Most negative (scores 13–20)	*Intermediate (scores 11–12)*	*Least negative (scores 5–10)*	*Subjects*
No contact	2/26 (7.7%)	2/49 (4.1%)	8/120 (6.7%)	195
GP contact	4/16 (25.0%)	0/25 (0.0%)	1/38 (2.6%)	79
Psychiatric contact	3/9 (33.3%)	2/13 (15.4%)	1/14 (7.1%)	36

TABLE V

New onsets of major depression (16 in 299 subjects) in different categories of self-appraisal (Rosenberg negative items) and health service contact for 'nerves' (11 sub-clinical cases excluded)

Health service contact	*Rosenberg negative items*			
	Most negative (scores 13–20)	*Intermediate (scores 11–12)*	*Least negative (scores 5–10)*	*Subjects*
No contact	1/24 (4.2%)	2/49 (4.1%)	5/116 (4.3%)	189
GP contact	1/13 (7.7%)	0/25 (0.0%)	1/37 (2.7%)	75
Psychiatric contact	3/8 (37.5%)	2/13 (15.4%)	1/14 (7.1%)	35

in women who have not sought medical help for psychological problems.

Summary for hypothesis 1

The findings have not verified hypothesis 1. There is, however, some indication that negative self-esteem may help to predict new episodes of major depression in women who have already had prior health service consultation for psychological symptoms. (An alternative possibility, to be discussed later, is that prior contact only operates as a predictor in women with low self-esteem.) The predictive power of low self-esteem is reduced when women in a sub-clinical stage of illness are eliminated from the sample, but the failure to reach significance may result from smaller numbers. These distinctions will be considered further in the discussion section.

Hypothesis 2

"Women who have recovered from an affective disorder (depressive illness or anxiety state) have lower self-appraisal than those who have not been ill."

At follow-up, to keep the interviews as short as possible, the Rosenberg scale and other parts of the schedule had been discarded. Women who had been recognised initially as cases and who had recovered before the second interview tended to show lower scores on the Foulds–Bedford indices (greater self-confidence) on the second occasion, but still had significantly higher mean scores than people who remained non-cases over the 6-month period. Table VI shows means and standard deviations for all those who had been cases at any time during the period, distinguishing those who had recovered and those who had not. Both have significantly higher mean scores than non-cases, as shown by the F-ratios from separate analyses of variance. Thus hypothesis 2 was supported: women who have recovered from affective illnesses lack self-confidence when compared with others who have not been recent cases. A covariance adjustment for anxiety and depression scales at the time of the second interview reduced the differences between groups slightly, but they remained significant for standard and positive scales. A further analysis showed no evidence that the scores of recovered cases were affected by either the duration of the episode or time since recovery.

Hypothesis 3

"Women who become ill with anxiety or depression suffer a decrease in self-appraisal."

As expected, women who became cases after the first interview showed a fall in self-confidence on the Foulds–Bedford measure (statistically significant for standard and positive scales) when compared with non-cases, who tended to change in the opposite direction. Hypothesis 3 is thus also supported and, as a corollary, unrecovered or relapsed cases had very significantly greater mean scores (lower self-confidence) than non-cases at the follow-up (Table VI). This time, in contrast with recovered cases (see previous paragraph), a covariance adjustment for anxiety and depression scales did eliminate all three significant differences.

Additional findings

Our initial interest in self-appraisal was as a possible link between vulnerability factors and illness (Ingham *et al*, 1986). Two of Brown & Harris's vulnerability factors (absence of confidant and childhood separation from mother) were associated with low self-appraisal in a sample of people who were not ill at the first interview, but neither of these factors was a statistically significant predictor of new onsets in the present study.

The number of children under 14 in the home predicted new onsets significantly when onsets were defined to include any disorder of anxiety or depression (major or minor, see Table VII). It became non-significant, in a logistic regression analysis, after adjusting for low self-appraisal. However, the confidence level was changed only marginally by the adjustment ($P<0.10$), and when the analysis was repeated with number of children represented as a dichotomy (two or more/less than two), its effect remained significant even after adjusting for low self-appraisal. The latter cannot be claimed to be an important mediating element.

Marital status was also a predictor of new onsets, but retained its predictive power when similarly adjusted. Unemployment of husband, after making the necessary adjustments for marital status, was also a significant predictor of new onsets, and also remained so after self-esteem was controlled. Table VII summarises the findings. Hence,, whatever the mechanism by which marital status and unemployment of the husband conduce to new illnesses, self-esteem does not appear to have an important role.

TABLE VI
Scores at second interview

	Standard form			*Foulds–Bedford scale* *Negative items*			*Positive items*		
	n	*Mean*	*s.d.*	*n*	*Mean*	*s.d.*	*n*	*Mean*	*s.d.*
Non-cases	270	10.21	2.30	271	5.45	1.41	272	4.75	1.40
Cases recovered at interview 2[1]									
Anxiety and minor depression	23	11.26	2.40	23	6.13	1.63	23	5.13	1.29
Major depression	21	11.71	3.42	22	6.05	1.68	22	5.77	2.05
F (d.f. = 2)		5.5			3.8			5.5	
P		0.004			0.023			0.005	
Cases remaining or relapsed at interview 2[1]									
Anxiety and minor depression	27	11.44	2.89	27	6.11	1.53	28	5.36	1.73
Major depression	26	13.08	4.04	26	7.04	1.97	26	6.04	2.36
F (d.f. = 2)		17.1			15.4			9.7	
P		<0.001			<0.001			<0.001	

[1] Cases include the 11 who were 'sub-clinical' at interview 1. Diagnoses are based on all information, including that obtained at interview 3 when it was available.

TABLE VII
Prediction of new onsets, including anxiety, minor depression and major depression (n = 310)

	Scaled deviance (d.f. in brackets) *Unadjusted*	*Adjusted for Rosenberg negative items*
Number of children under 14	7.9 (3)*	6.9 (3)
Marital status	8.9 (2)**	7.7 (2)*
Unemployment of husband (adjusted for marital status)	6.0 (2)*	6.2 (2)*

$^*P<0.05$; $^{**}P<0.02$

Discussion

Our intention has been to discern whether indicators of low self-regard can predict subsequent depressive and other illness episodes. We have not set out to explore a full interaction model by including data on life stresses and adversity, but rather have confined ourselves to testing the three hypotheses set out earlier in the paper.

The conclusions to be drawn concerning the role of self-appraisal must hinge on the adequacy of the measures used, especially those assessing self-appraisal *per se*. The two instruments employed have all the limitations of standard questionnaires. Moreover, a little reflection suggests that self-esteem is a complex concept which must involve a number of components with possibly discrete psychological meanings. On the other hand, the instruments used are well established and extensively researched. Both scales distinguish normal subjects from those with depression at high levels of discrimination, and are also able to reflect differences in social background and early environment among normal individuals. Details have been given elsewhere (Ingham *et al*, 1986). For the present, the scales appear adequate for exploratory purposes.

The "most of my life" phrase was inserted into the Rosenberg statements to reduce the likelihood that the scale would be susceptible to short-term fluctuations. Such a device does not seem to be completely successful, as present results with the Foulds–Bedford scale have shown. Without it the scales may well have reflected prodromal depressive states to a greater degree, but it is unlikely that other results would have altered materially.

Using the Rosenberg scale we have found, as have previous writers (e.g. Brown & Bifulco, 1985), that it is people's tendency to make negative statements about themselves that is associated with depression. There was no such link with the absence of positive statements. Warr & Jackson (1983), who used a scale similar to our own, including some Rosenberg items, showed the same differential link (between positively and negatively worded items) with unemployment, which is also associated with depression (Surtees *et al*, 1983).

The three Foulds–Bedford scales, however, show a different pattern. Neither the standard form, nor either of its components, is a predictor of new onsets. Furthermore, it was the positive scale that showed a significant lowering of self-confidence at the second interview in new-onset cases. There is little point in speculating on the reason for this anomaly, but in comparing the scales there are two obvious differences that may be relevant. The first is that the Foulds–Bedford sub-scales contain only three items each and may consequently be less reliable than the Rosenberg sub-scales. Secondly, the difference in wording of possible responses between the two scales may be significant. It should be noted, however, that most of the differences between group means are in the predicted direction.

The findings prompt some general speculation concerning the relationship between depression and low self-esteem. Like all other investigators, we found a major fall in self-esteem with the onset of depressive illness, especially major depressive illness, whether the levels are compared with the individual's own pre-morbid level or with general population norms. It appears that the magnitude of the drop is closely related to the intensity of the depressive or anxious mood change. In this context, then, low self-esteem is intimately related to the illness itself. The temporal relationship between the drop in self-esteem and the onset of symptoms is not clear, but the results from our 11 'sub-clinical' cases suggest that the fall occurs at an early stage. It might well be one of the earliest manifestations of depression, and one of the last to disappear. But in addition it is possible that low self-esteem, generally of less severity than is found during a phase of illness, can be a more enduring characteristic. As discussed in our earlier paper (Ingham *et al*, 1986), the principal evidence against this view is that recovered depressives are generally reported to regain normal levels of self-esteem. However, most investigations of this issue have been based on hospital samples, which are known to be atypical in many respects. In the present survey, recovered cases could still be distinguished on their self-esteem scores from those who had been well throughout. Moreover, this difference persisted, even adjusting for current mood – a necessary procedure since recovered individuals may well have had some residual symptoms. Nor was there any evidence among recovered cases that self-esteem was influenced by the time since recovery or the duration of the recent illness, insofar as we could determine the relevant dates. Thus several findings point to the same conclusion – that individuals liable to depressive illness, as witnessed by their history, are distinguished by chronic self-disparagement which is not explained by their anxious or depressive mood. The mechanism underlying the persistence of low self-esteem, if confirmed, still remains to be clarified.

Our present concern, however, is essentially with the predictive power of low self-esteem (hypothesis 1) in the population at large. In exploring this topic it became clear that low self-esteem predicted future illness only among those women with a prior history of a treated episode. The strength of this association varied somewhat according to how the 11 sub-clinical individuals were considered, but the general pattern was as shown in Tables IV and V. These may be read as showing that low self-esteem is predictive only among those with a prior history, or conversely, that a prior history is only predictive among those with low self-esteem. It is well established that a prior history is a potent predictor of new episodes, and the present data suggest that low self-esteem moderates this relationship in some way. It is unlikely that self-disparagement *per se* induces depression (since as already explained it has no predictive power for women without a previous history). Alternatively, it could be that recurrent episodes of illness leave their mark, as it were, by a progressive impairment of self-confidence which can be recognised between spells of illness, an effect which might be non-specific for any kind of recurrent disorder. More speculatively, low self-esteem might be little more than a correlate of other factors which determine recurrence of illness, although perhaps this possibility is less likely. Resolution of these alternatives must await closer study of the broader questions already adumbrated. Meanwhile, we conclude that for the majority of women, namely those with no known history of illness, self-concept is no pointer to psychiatric illness in the near future.

Finally, we briefly consider the role of chronic social adversity or vulnerability factors insofar as they relate to new onsets. The question is important, since in recovered cases it is possible that the persistence of low self-esteem after recovery (see above) might be due to the continuation of the adverse social stresses which triggered the illness itself; among those with no prior history, adversity might predispose to illness by first causing a loss of self-regard. Our numbers do not make detailed analysis possible, but, for the total sample, the results do not support the notion that the putative vulnerability factors described in the earlier cross-sectional study (Ingham *et al*, 1986) reduce the threshold to depressive illness by reducing self-esteem. The factors that were most associated with low self-esteem were not the ones that best predicted new onsets; and, conversely, two of those that did predict new onsets continued to do so after adjustment for self-esteem.

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Marital Problems and Treatment Outcome in Depressed Women
A Clinical Trial of Social Work Intervention

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In a clinical trial investigating the effectiveness of social work intervention with depressed women patients in general practice, 80 women were randomly allocated to an experimental group for referral to attached social workers or to a control group for routine treatment by their GPs. They were reassessed at 6 and 12 months. The results indicated that women who had major marital problems were more likely to be depressed at follow-up than those with good relationships. However, patients with marital difficulties in the experimental group made more improvement than the controls. Women initially assessed as suffering from 'acute on chronic' depression and having major marital difficulties were found to benefit most from social work intervention.

Marital disharmony and breakdown have a number of damaging ramifications both for the adults concerned and the children. It brings with it a higher risk of suicide, behavioural problems in children, and deterioration in physical health (Home Office, 1979; Dominian, 1980).

Depression and marital problems are also closely linked, although there is controversy as to their sequence (Briscoe & Smith, 1973; Brown & Harris, 1978). Paykel *et al* (1969) found that increases in marital difficulties and disputes were the most frequent 'life events' reported by depressed women in the 6 months prior to seeking treatment. In another study, Weissman & Paykel (1974) assessed the social adjustment of depressed female outpatients and compared them with a normal female population. They found that the depressed women had much more marital dysfunctioning than the normal women, characterised by poor communication, friction, disengagement, and sexual problems. In addition, these marked impairments persisted long after the women recovered clinically (Bothwell & Weissman, 1977). Other studies have suggested that women who lack close confiding relationships with their husbands or cohabitees are particularly vulnerable to episodes of depression (Miller & Ingham, 1976; Brown & Harris, 1978).

Research has indicated that more people approach their general practitioners with their marital difficulties than any other professional (Chester, 1971; Home Office, 1979; Mitchell, 1981; Brannen & Collard, 1982), presenting their problems either overtly or covertly, with symptoms of physical or mental ill-health. It has been advocated that general practitioners and the primary care team should be strengthened by the attachments of other professionals to deal specifically with problems such as these (WHO, 1973; Shepherd, 1974), and in recent years an increasing number of different professions have been attached, including social workers, clinical psychologists, community psychiatric nurses, and counsellors (Kincey, 1974; Hicks, 1976).

While there has been a considerable number of reports and subjective accounts on the advantages that these various schemes confer (e.g. Clare & Corney, 1982), there has been a lack of independent objective assessments of whether these schemes actually benefit the patients referred.

The aim of the clinical trial described in this paper is to investigate whether the attachment of a social worker to general practice can benefit depressed women of child-bearing age. As previous studies have found that clinical improvement is often directly related to marital improvement, this suggests that the treatment of depression should also include social intervention aimed at reducing marital difficulties and strife (Rounsaville *et al*, 1980). This paper, therefore, specifically focuses on the subgroup of women with marital difficulties, investigating their clinical outcome after 6 months and one year, and the effects of treatment.

Method

Following a pilot study to test feasibility, the main study was initiated involving the participation of six doctors. Five of these were based in a health centre with a social work attachment scheme involving four part-time social workers. This scheme had been in operation for 2 years when the study started and was well established. The sixth doctor was from a single-handed practice and had been involved in previous studies with this unit.

The doctors were asked to refer women aged 18–45 years presenting with 'acute' or 'acute on chronic' depression, whose symptoms of depression had appeared (in the case of the former group) or intensified (in the case of the latter group) in the previous 3 months. Women suffering from major physical ill health and those already seeing a social worker were excluded.

All referrals were initially interviewed by a psychiatrist, using the Clinical Interview Schedule (CIS; Goldberg *et al*, 1970). This schedule was developed specifically for use in community surveys and has been found to be acceptable to individuals who do not see themselves as psychiatrically disturbed. It includes a detailed enquiry into the severity and frequency of ten common symptoms (e.g. irritability, depression, anxiety) which are each rated on a five-point scale. The total symptom score is a composite rating obtained by adding these ten scores together. At the end of the interview, the psychiatrist rates the patient on the presence of 12 'manifest abnormalities' observed during the interview. These are each rated on a five-point scale and the scores are added together to form a composite 'manifest abnormality score'. A total score for the interview is obtained by adding the symptom score to twice the manifest abnormality rating. This method of scoring has been found to have the best agreement with clinical judgement. A rating of overall severity is also made. A score of 0 is not a case, 1 is subclinical, 2 a psychiatric case of mild degree (disturbance is just clinically significant), 3 a case of moderate degree, and 4 a case of severe degree.

Patients ascertained as obtaining a rating of overall severity of 2 or more who also fulfilled the age and chronicity criteria were referred to a social research worker using the Social Maladjustment Schedule (Clare & Cairns, 1978). This instrument includes a detailed marital assessment in which married and cohabiting women were rated on four items: shared interests and activities, sources of conflict, responsibilities and decision-making, and sexual compatibility. For women on their own the rating concerned the woman's relationship with her boyfriend or her attitudes towards the opposite sex and her opportunities for meeting men.

Patients were then randomly allocated to the experimental or control group by a research assistant who was not involved in the assessments. In order that the two groups were matched in certain characteristics, patients were allocated into one of eight sets according to two categories of age, married or single status, and whether they were suffering from an 'acute' or 'acute on chronic' depression. The first patient in any of these sets was assigned to the experimental or control group by the toss of a coin, the second patient was automatically assigned to the alternative group, the third patient randomly allocated and so on. Those allocated to the experimental group were referred to one of four attached social workers for treatment, while the controls were referred back to their doctors for routine treatment.

After 6 months, the women were reassessed by means of the same instruments. The research staff involved in these assessments were unaware of any intervening treatment. Additional information was obtained by placing a card in the general practitioner's notes. The date of any clinical improvement or deterioration, from the initial psychiatric assessment to one year later, was recorded, as well as the number of visits the patient made to the doctor and any psychotropic drug prescription. Details were also collected from the attached social workers, who completed specially designed forms regarding each client referred to them. These forms recorded the initial assessment of the social worker and the problems presented, and also details of the social worker's contacts with the client, the types of intervention given, and the reason for closure.

The results were analysed by means of univariate and multivariate analyses of covariance, taking into account the three matching variables and also the initial scores as covariates.

Results

Characteristics of cohort

There were 116 women referred to the study, of whom 104 attended for the first interview. Of these, 91 were assessed as suitable for inclusion in the study. Of the 13 women who were ineligible, nine were considered to be chronically depressed, two had depressions of insufficient severity, and two were already seeing a social worker.

Of the 91 eligible women, four refused the social interview and seven were unwilling to participate in the follow-up

TABLE I

Summary of initial characteristics of experimental and control groups

	Experimental group (n=41)	*Control group (n=39)*
Marriage		
Married women and cohabiting women: %	76	69
Women with major problems in their marital or sexual relationships: %	63	59
Work		
In paid employment: %	61	64
Social class		
Social class I & II: %	17	10
Social class III: %	68	79
Social class IV & V: %	15	10
Ethnic origin		
British by birth: %	88	90
Duration of illness		
'Acutely' depressed: %	39	49
'Acute on chronic': %	61	51
Age		
Mean age: years	31.0	28.6

TABLE II
Presence or absence of major 'marital' problems: patients improved 6 and 12 months after referral

	No or minor problems		*Marked problems*	
	6 months (n=31)	*12 months (n=29)*	*6 months (n=49)*	*12 months (n=40)*
Percentage of patients improved (severity rating 0 or 1)	87	72	51	47.5
Percentage of patients not improved (severity rating 2 or 3)	13	28	49	52.5

TABLE III
Presence or absence of major 'marital' problems: patients improved in the experimental and control groups

	No or minor problems				*Marked problems*			
	Experimental		*Control*		*Experimental*		*Control*	
	Number	*%*	*Number*	*%*	*Number*	*%*	*Number*	*%*
Patients improved	11	73	16	100	17	65	8	35
Patients not improved	4	27	0	0	9	35	15	65
Total	15	100	16	100	26	100	23	100

interviews or had moved out of the area. Therefore, 80 patients were included in the study, 41 being allocated to the experimental group (existing services plus allocation to the social worker) and 39 to the control group (existing services only).

At the time of the initial assessment, the experimental and control groups were closely similar in respect of their demographic characteristics, and psychiatric, physical ill health and social ratings, and there were no statistically significant differences between them (Table I). Over two-thirds of both groups were married or cohabiting, and high proportions had major problems in their relationships with their husbands, cohabitees, or boyfriends (hereinafter called major 'marital' problems). Approximately 80% of each group were taking psychotropic drugs. Table I shows some of the initial characteristics of both groups.

Follow-up scores

When the women were re-interviewed 6 months later, approximately two-thirds were assessed by the psychiatrist as improved (i.e. given a severity rating of 0 or 1 on the CIS). However, the majority of women who had no major 'marital' problems had improved at follow-up, in comparison to only half of those with major problems in this area (Table II).

This difference was statistically significant at below the 1% level for the four follow-up composite clinical ratings (symptom score, manifest abnormality score, severity rating, and total score) using univariate analyses of covariance. The medical notes also revealed that women with no or minor 'marital' problems improved in health much quicker than those with 'marital' difficulties ($P<0.01$). The former were also less likely to be depressed one year after referral (Table II). However, this information was only available for 69 women, as 11 patients had moved and changed practices.

Although the quality of the patient's relationship with her sexual partner affected the clinical outcome, it was found to have no statistically significant influence on the improvement of social scores, on psychotropic drug prescription by the doctor, or on the patient's attendances at the surgery, as recorded by the general practitioner.

Effects of treatment

There was very little difference in outcome between the experimental and control groups after 6 months or one year according to the clinical data collected or the social or medical criteria.

Over 80% of both groups had taken psychotropic drugs between the two assessments, and there were no major differences between groups on the length of time patients were taking these drugs. Although at the time of follow-up assessment 22% of the experimental group were taking a psychotropic drug in comparison with 31% of the controls, this difference was not statistically significant.

Women with major 'marital' problems who received social work help made much more improvement than the controls (Table III). This interaction between treatment and quality of the patients' 'marital' relationship was found to be significant when a multivariate analysis of variance was performed on all the follow-up clinical scores ($P<0.05$), and for three out of four of the follow-up composite clinical ratings using univariate analysis of covariance ($P<0.05$; Corney, 1981*a*, 1985). However, there were no significant interactions on the social scores indicating improvement, or on psychotropic drug prescription or patients' attendances at the surgery.

TABLE IV

Proportion of patients improved after 6 and 12 months for 'acute' and 'acute on chronic' patients according to quality of 'marital' relationship

	Group 1 (acute on chronic; no or minor problems)		*Group 2 (acute on chronic; marked problems)*		*Group 3 (acute; no or minor problems)*		*Group 4 (acute; marked problems)*	
	Proportion	*(n)*[1]	*Proportion*	*(n)*	*Proportion*	*(n)*	*Proportion*	*(n)*
Proportion improved at 6 months (severity rating 0 or 1)								
Group E[2]	0.6	(10)	0.8	(15)	1.0	(5)	0.5	(11)
Group C[3]	1.0	(4)	0.3	(16)	1.0	(12)	0.4	(7)
Proportion improved at 12 months (according to medical notes)								
Group E	0.6	(9)	0.6	(14)	0.6	(5)	0.6	(10)
Group C	0.5	(4)	0.2	(11)	1.0	(11)	0.6	(5)

[1] Numbers in brackets represent total in each subgroup.
[2] Group E = experimental group.
[3] Group C = control group.

Further analysis of the data indicated that the chronicity of the patient's depression was another important variable in determining outcome. An important distinction between the patients included in the study is that some were 'acutely' depressed, having had symptoms of depression for 3 months or less, while others were assessed as 'acute on chronic', having had symptoms for a longer period. The results indicated that a much higher proportion of 'acute on chronic' patients improved if they were referred to a social worker. For 'acute' patients the reverse was true, fewer patients improving when referred to the social work service. This interaction between treatment and chronicity was also found to be statistically significant in a multivariate analysis of covariance ($P < 0.05$), and in the univariate analyses of three out of four of the composite clinical scores ($P < 0.05$).

Chronicity of illness, presence of marital problems, and treatment

As the analyses revealed that chronicity of the patient's depression and the quality of her 'marital' relationship were both important, further analyses of three-way interactions of these two variables and treatment were performed. These were statistically significant for univariate analyses of three out of four of the follow-up composite clinical scores ($P < 0.05$).

These findings suggested that one subgroup of patients benefited more from the social workers' interventions than the three other groups (Table IV). These were women who were assessed initially as having major 'marital' problems and who were suffering from 'acute on chronic' depression (group 2). With this particular group of women, 80% of the experimental group were assessed as clinically improved at follow-up in comparison with only 31% of the controls. This difference still held one year after referral when the medical notes were examined. Of the experimental group, 57% were judged by the doctor to be well, in comparison with 18% of the controls. In contrast, women assessed as acute on chronic who had no or minor 'marital' problems (group 1) fared less well in the experimental group than the controls, but there was no difference in outcome one year after the initial assessment.

For the acutely ill patients, there were fewer differences in outcome between the experimental and control groups. All those with no or minor 'marital' problems improved (group 3), while over half of those with a poor relationship failed to get better.

The majority of women who had improved clinically also made some improvement in their social adjustment; thus 'acute on chronic' depressed patients with major 'marital' difficulties who were referred to a social worker made more improvement in their social adjustment than the controls in this category. However, the three-way interactions for social work intervention were not statistically significant, either for social scores in general or for those scores measuring marital adjustment (Corney, 1981*a*).

Social contacts

The women were also asked about the number of social contacts they had. The women with major 'marital' problems also tended to have poorer social contacts. Over 40% of the women with 'marital' problems were rated as having poor social contacts in comparison with only 16% of the women who had no or minor problems. The results suggest that those women with poor social contacts benefited more from the additional help from a social worker, as more clinical improvement was made by patients with poor social contacts in the experimental group than those allocated to the control group. This interaction was significant, however, for only two of the four composite clinical ratings made at follow-up, and a multivariate analysis of variance was not possible. However, all five subjects in group 2 ('acute on chronic', major 'marital' problems) with poor social contacts improved in the experimental group, by contrast with only two out of eight of the controls.

TABLE V

Who helped most: numbers improved at 6 months for the four groups of patients studied

Who helped	*Group 1 (acute on chronic; no or minor problems)* Number improved	(n)[1]	*Group 2 (acute on chronic; marked problems)* Number improved	(n)	*Group 3 (acute; no or minor problems)* Number improved	(n)	*Group 4 (acute; marked problems)* Number improved	(n)
Experimental group								
No one	3	(6)	3	(4)	1	(1)	4	(6)
Social worker	2	(3)	7	(8)	3	(3)	0	(2)
Other	1	(1)	2	(3)	1	(1)	1	(3)
Total	6	(10)	12	(15)	5	(5)	5	(11)
Control group								
No one	2	(2)	3	(11)	6	(6)	1	(3)
Other	2	(2)	2	(5)	6	(6)	2	(2)
Total	4	(4)	5	(16)	12	(12)	3	(7)

1. Numbers in brackets represent total in each subgroup.

Initial and follow-up social scores

When the initial scores of the four categories of patients outlined in Table IV were examined, there was no evidence to suggest that the differences in outcome between the patients in the experimental and control groups for these categories were due to the initial social scores (Corney, 1981*a*).

The social workers' contacts with their patients and the nature of the intervention

As the previous results suggest that social work intervention was more beneficial to patients suffering from 'acute on chronic' depression, especially those with marked 'marital' problems, the social workers' activities were analysed, taking into account the four groups of patients outlined in Table IV.

The social workers also recorded in detail their activities performed either with the client or on behalf of the client. The social workers worked in a number of ways with these clients and were not given any specific guidelines. This was because the needs of the clients were remarkably varied: some women had few social problems while others had many. The social workers were asked, however, to try to see their clients on a regular basis, to make a contract with the client specifying frequency of interviews, and to agree on a plan of action regarding their work in the following months. They were also asked to limit their time of contact to the 6 months between assessments.

Communications with the clients were classified by the social workers according to the typology developed by Hollis (1964), which the social workers were familiar with using. The social workers used sustaining and exploring techniques with all the clients they interviewed, investigating the reasons why the patient was depressed as well as offering support and reassurance. The social workers also considered that in 90% of the cases they tried to develop their client's awareness and understanding of the dynamics of her personal situation, behaviour, and attitudes. In addition, the social workers tried to help some of their clients using behavioural techniques; for example, helping the client manage her depression by setting her a number of tasks to perform each day or devising a contract between husband and wife to increase co-operation between them.

In addition to the interventions, the social workers also gave some form of help concerning practical matters to over 40% of their clients. Of the 35 patients they saw, they gave information on practical issues to 15, they acted as an advocate or referred to other agencies for 8, and they gave substantial help to 2 women in the form of financial aid, holidays, day nursery placements, and council accommodation.

Although similar proportions of patients in all four groups received the different kinds of counselling identified, a higher proportion of patients in groups 2 and 3 received practical help.

Number of interviews

There was no general relationship between clinical improvement and the number of interviews given, but in many cases only a few interviews were given as the patient improved and further interviews were considered unnecessary.

Just over 60% of the patients seen by the social workers were interviewed alone. This was not usually due to decisions made by the social worker but due to the spouses of the patients often resisting involvement, or the patients' reluctance for their spouses to be interviewed. However, clients who were seen with their spouses did not improve more on ratings measuring marital adjustment or on clinical scores that those clients seen alone.

Motivation

At the end of their involvement with each client, the social workers were asked to rate the clients' motivation to be helped by them. The social workers assessed nearly 60% of the women in group 2 (Table IV) as highly motivated, in comparison with only 15% of the women in the other groups. This term was used by social workers when clients were willing not only to accept help but also to alter their behaviour or situation accordingly. The highest rate of improvement was found among the clients who were highly motivated; over 80% of this group had improved at follow-up.

Clients' assessment of help obtained

At the end of the second social assessment, the patients were asked if anyone had helped them, and they were given time to spontaneously mention one or more individuals. They were then asked who had helped most (Table V), and were questioned in further detail about whether they received help from their general practitioner or from a social worker.

Nearly 70% of the experimental group spontaneously mentioned one or more people as helpful (including spouse, friends) in comparison with only 45% of the controls. Two-thirds of those who saw a social worker spontaneously mentioned that she had helped them, especially those in groups 2 and 3.

When asked who had helped most, over 45% of the 35 patients who saw a social worker indicated that she had, particularly patients in groups 2 and 3 (Table V). The doctor, husband, friends, and relatives were mentioned by the control group as being the most helpful more often than by patients in the experimental group.

When the patients were asked specifically whether they had seen a social worker and whether it had been helpful, a further six patients thought she had been helpful. All but one of the 35 patients who saw a social worker had considered that the social worker had been understanding, and only two patients had found difficulty in talking to her. Generally, patients appreciated the help they received, with 80% mentioning the support given by the social worker. A third mentioned practical help or information, and the same proportion indicated that the social worker had helped them either "manage their depression" or increased "their understanding of themselves" or "helped in their relationships" (Corney, 1981*a*).

Discussion

The results suggest that the quality of the patient's marital relationship is a crucial factor affecting clinical outcome: women with marital difficulties were much more likely to be still depressed 6 months or one year after referral than women without these problems. These findings are in accordance with those found by Weissman and her colleagues in their study of depressed female out-patients (Rounsaville *et al*, 1980).

The results of this investigation suggest, however, that women with 'acute on chronic' depression who have marital difficulties may benefit from the help of a social worker. Compared with the controls, more patients in this subgroup who saw a social worker were not depressed at follow-up. The findings of other studies in this area are mixed. Weissman and her colleagues found that individual psychotherapy did not bring about any more substantial social, marital, or clinical improvement among individuals assessed as having "initial marital disputes", leading the authors to suggest that conjoint therapy might have been more appropriate (Rounsaville *et al*, 1979). Although some of the women in this present study received help together with their husbands, this was not built into the design of the trial and depended very much on the patient's wishes or those of her husband. There was no evidence to suggest, however, that conjoint therapy brought about additional improvement in clinical ratings or those scores measuring marital adjustment.

A study of task-centred social work conducted on self-poisoners who had recently been admitted into casualty with an overdose found that one of the major areas of improvement in the experimental group was the patient's marital and interpersonal relationships (Gibbons *et al*, 1978). Although no differences were found between the experimental and control groups in respect of repeated self-poisoning or of clinical criteria, the experimental group showed significantly more change than the controls in their social problems when assessed at 4 and 18 months after entry into the study.

Why did the social worker help patients in group 2 more than the others? Further analysis of the data of the present study, including details on the patients' social contacts and who they felt had helped, suggested that many of the patients with a poor marital relationship also had poor social contacts. Thus in many cases the patient not only could not talk to her husband but also had no one else in whom she could confide. This may be why this group of women so readily accepted the emotional support given by a social worker. The reason why the control patients in group 2 did so badly could have been due to their lack of support from others: over 70% felt no-one had helped them. In contrast, only one-quarter of the experimental patients in group 2 felt no-one had helped, and over half felt the social worker had helped most. Possibly, the lack of a difference between the outcome of the experimental and control groups in the other groups could have been due to the support the control patients received from relatives, husbands, and friends.

The results also indicated that the patients who gained most benefit were those who had been depressed for some time. The results, especially when considered in conjunction with a previous study of general practice patients, suggest that doctors should refer to social workers patients with more long-standing depressions and neuroses, as these will be helped more than those whose symptoms have a very recent onset (Cooper *et al*, 1975; Shepherd *et al*, 1979). When the patient's depression becomes acute on chronic, there is less chance of spontaneous recovery (Cooper *et al*, 1969; Kedward, 1969), and help from an outside agency may be necessary to bring about change. The patient may also be more likely to accept such help and act upon it.

Patients with acute depression appear more likely to recover without outside help. The additional help of the social worker may be unnecessary or even harmful, the social worker interfering with the individual's own mechanisms of coping or the support received from others. The patient may also be less motivated to receive the help offered, having become depressed only very recently, and may not be inclined to accept outside help or regard it as necessary. It is possible that very early intervention may therefore be inefficient, inappropriate or even harmful.

In this study the client's motivation to be helped by a social worker appears to be related to the length of time she has been ill and the help she receives from others. However, it is possible that the nature of the patient's social problems is another important factor affecting motivation. Patients in group 2 had more social problems than the others; many of these were practical matters, which may have been why so many of this group were eager for help.

The social workers carried out much more practical help on behalf of the clients in group 2 and contacted more agencies on their behalf. In the earlier study on chronic neurotic patients, where substantial differences were found between the experimental and control groups, the majority of the social work carried out was practical in nature (Shepherd *et al*, 1979). Although many social workers tend to stress their skills in counselling and underrate the practical help they can give (Wootton, 1978), the arrangement or provision of practical assistance by social workers may be of great value to general practice patients, including those who are depressed. Studies obtaining the clients' views of social work support this, clients appreciating most the combination of practical help with emotional support (Butrym, 1968; Mayer & Timms, 1970; Sainsbury, 1975; Corney, 1981*b*). Certainly the social workers' expertise and knowledge of practical resources are what specifically distinguish them from their other colleagues in general practice and can be used to argue their case for inclusion into the primary care term (Clare & Corney, 1982).

From the results of this study it seems reasonable to suggest that a further study should concentrate specifically on depressed women with marital problems using an 'attached' social worker with experience in conjoint marital therapy. The tendency of women with marital problems to become chronically depressed (Rounsaville, 1980) indicates the need for the development of effective treatment measures, resulting in both marital and clinical improvement.

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Caring and Its Burdens

A Study of the Spouses of Depressed Patients

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Spouses of 24 patients suffering from persistent depression were interviewed to find out how they were affected by the patient's illness. These effects were marked, although mainly endured without complaint, and included restrictions in social and leisure activities, a fall in family income, and a considerable strain on marital relationships. Some of the behaviour shown by patients was hard to bear, and 'negative' symptoms such as misery, withdrawal, and worrying commonly caused problems. Few relatives, however, know how to deal with difficult behaviour. Despite this, the majority remained committed to staying with the patient. Spouses had a variety of complaints about the way they were handled by hospital staff, particularly about being deprived of information and advice. These results have considerable implications for the way in which relatives should be dealt with as part of the overall management of persistent depression.

Over a period of 30 years there have been several studies documenting the problems of relatives living with those who suffer from psychiatric illness. We have reviewed these studies and their impact elsewhere (Fadden *et al*, 1987). The general conclusion must be that relatives bear considerable burdens, often without complaint and at some detriment to their own mental health. They are used as sources of information but their own questions are left unanswered by the psychiatric team. They are rarely offered any service or advice and consequently have little understanding of the patient's illness. They frequently receive the impression that they are held responsible for the patient's state, without being given any idea of how they could modify aspects of their behaviour that might contribute to the patient's condition. This situation will not remedy itself, and is likely to worsen in the face of the continuing move towards community care.

Studies of the problems resulting from specific conditions have so far been concerned with the relatives of those with schizophrenia (Brown *et al*, 1966; Creer & Wing, 1974; Wing, 1982). The current study arose from a concern that the relatives of depressed patients might be exposed to similar difficulties. No previous investigation has focused on this group. Even the professional who intends well towards such relatives is constrained by ignorance of the problems they may face. We therefore sought to establish a typology of the burdens of living with depressed patients. Although the data do not purport to describe the absolute extent of burden, they do indicate the kinds of problems that might be experienced by these relatives, and thus increase the awareness of a clinical team willing to help them. In order to obtain the widest view of these problems, we chose to investigate separate groups of bipolar patients, unipolar 'endogenous' depressives, and neurotic depressives. As a result, clinical course varied from persistent misery to relatively discrete but more florid episodes.

The study is for the most part descriptive, and had a number of aims. It sought to portray the burden that results when a patient is unable to carry out expected roles because of a depressive condition. It aimed to document the types of behaviour and symptoms exhibited by the patient that are most difficult for the relative to deal with, and the ways relatives have of coping with such behaviour. The relatives' own psychological adjustment was assessed. The knowledge that people had about the nature, origin and possible outcome of their depressed relative's condition was recorded, and their opinion was sought of the service they themselves had received from the psychiatric team.

Method

Patients were obtained from the emergency clinic and out-patient clinics of the Maudsley Hospital. In-patients were only admitted to the study if admission was recent or they were still spending some time every week at home. The following selection criteria were used:

(a) living currently and at least for the two previous years with a relative

(b) onset of illness at least three years ago
(c) no organic disorder, forensic problems, or alcohol abuse
(d) at least three episodes of illness or persistent symptoms of a level likely to exceed the requirements of the Index of Definition threshold more than 50% of the time since first onset.

Twenty-four relatives, all of whom were patient's spouses, were interviewed. Originally it had been planned to interview any adult with whom the patient spent most time. However, as the first 16 patients who met all the other criteria for selection were in fact living with spouses, it was decided to select thereafter only those patients who were living with a spouse. It seems a reasonable assumption that depressed patients who live with someone are most likely to be living with a spouse. It is improbable that selection bias could account for the preponderance seen in this study.

Thirteen cases who were in scope (35%) were excluded because they, their spouse, or their consultant refused permission for the interview.

Patients were allocated to one of three groups according to their measures on the Present State Examination (PSE) (Wing *et al*, 1974; Wing & Sturt, 1978). The Syndrome Checklist facility was used where appropriate, and the clinical data were processed by the CATEGO and ID computer programs. The three groups were:

(a) 5 females and 3 males with bipolar disorder
(b) 3 females and 5 males with unipolar episodes falling into CATEGO class R or D
(c) 4 females and 4 males with neurotic depression (CATEGO class N); these had symptoms more than 50% of the time but did not meet the criteria for the first two groups; in fact they had numerous symptoms most of the time.

This grouping was deliberately chosen to permit the study of cases with significant recurrent or persistent depressive symptoms. In terms of the problems they are likely to pose for relatives, if not necessarily in terms of the level of symptoms, they are therefore those towards the more severe end of the spectrum. In only one case from the bipolar group was the patient ill at the time the spouse was interviewed. In the other two groups the patient was ill when the relative was seen in half of the cases. Bipolar patients tended, as might be expected, to have distinct episodes, and were also more likely to be prescribed prophylactic medication. The clinical characteristics of the patients are given in detail in Table I.

TABLE I
Clinical characteristics of patient sample

	Total sample (n = 24)	*Bi-polars (n = 8)*	*Uni-polars (n = 8)*	*Neurotics (n = 8)*
Time since onset of initial illness				
3–5 years	5	1	1	3
5–10 years	6	1	3	2
10–20 years	6	2	2	2
More than 20 years	7	4	2	1
Length of stay in hospital over last 5 years				
None	7	1	1	5
2–4 weeks	5	—	3	2
2–3 months	3	2	1	—
3–6 months	5	3	1	1
6–12 months	1	—	1	—
More than 1 year	3	2	1	—
Form of illness				
Symptom-free between episodes	6	3	3	—
Mild symptoms between episodes	5	4	1	—
Quite severe symptoms between episodes	5	1	4	—
Symptoms all the time, with occasional worsening	8	—	—	8
Hospital status at time of interview				
In-patient	4	1	2	1
Out-patient	19	7	5	7
Day-patient	1	—	1	—
PSE CATEGO class				
R and M	3	3	—	—
N and M	4	4	—	—
D and M	1	1	—	—
R	7	—	7	—
D	1	—	1	—
N	8	—	—	8

Patients were of representative social class, but 15 were either unemployed or retired. Overall mean age was 49, with the bipolar group averaging six years younger than this (Table II).

The allocation of relatives to the three groups was determined by the type of illness seen in the patient. The relatives' characteristics are given in Table III.

As no measures were available which served the specific aims of this study, a new schedule was devised* which was developed on the basis of the Social Behaviour Assessment Schedule (SBAS) (Platt *et al*, 1983), the shortened version of the Camberwell Family Interview (Rutter & Brown, 1966; Vaughn & Leff, 1976), an instrument used in the current Camberwell High Contact Survey, and the schedule used by Paykel *et al* (1982). The schedule thus constructed assessed role function and allocation, difficult behaviours, and the relative's knowledge about the patient's condition and opinion of the service received. Coping was assessed with a 17-item questionnaire based on 14 items used by Folkman & Lazarus (1980) and a further 3 items described by Pearlin & Schooler (1978). Most of the information was gathered by the interviewer asking questions within a

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*Available from the authors.

TABLE II
Sociodemographic characteristics of patient sample

	Total sample (n = 24)	*Bi-polars (n = 8)*	*Uni-polars (n = 8)*	*Neurotics (n = 8)*
Sex				
Female	12	5	3	4
Male	12	3	5	4
Age				
Mean	48.58	42.88	53.75	49.13
s.d.	11.72	11.86	10.38	11.64
Employment status				
Working full-time	6	1	4	1
In charge of household	3	1	1	1
Unemployed	9	4	1	4
Retired	6	2	2	2
Social class[1]				
Professional/executive	2	1	1	—
Managerial	2	1	—	1
Skilled (non-manual)	6	2	1	3
Skilled (manual)	7	2	4	1
Semi-skilled	5	1	2	2
Unskilled	2	1	—	1

1. Social class was determined by husband's occupation according to the socioeconomic groups (SEG) of the Office of Population Census and Surveys (1970)

TABLE III
Characteristics of spouse sample

	Total sample (n = 24)	*Bi-polars (n = 8)*	*Uni-polars (n = 8)*	*Neurotics (n = 8)*
Sex				
Female	12	3	5	4
Male	12	5	3	4
Age				
Mean	47.46	42.00	51.63	48.75
s.d.	12.45	12.87	11.56	12.42
Employment status				
Working full-time	14	6	4	4
Working part-time	1	—	1	—
Retired	3	1	1	1
In charge of household	6	1	2	3
Number of years living with patient				
Mean	19.50	13.38	26.38	18.75
s.d.	13.18	15.35	7.98	13.12
Number of hours in contact with patient each week				
Mean	64.58	61.25	63.63	68.88
s.d.	17.31	18.10	16.95	18.30

structured framework and rating the answers, after further probing if necessary, in accord with predetermined categories. The degree of perceived control over adverse situations was rated using a Likert scale. Relatives were also given the PSE.

Initial contact was made with patients through their consultants, and if they met the criteria listed above they were interviewed with the PSE as part of the allocation to the appropriate group. Contact was then made with the relatives. Most interviews were carried out at home, and in a quarter of cases it was not possible to interview the relative in the absence of the patient. This happened more often with bipolar patients. It could be argued that the more optimistic assessments of those married to bipolar patients arose partly because of these circumstances of the interview, but it is also possible that bipolar patients tended to stay in the interview *because* they were well. The interview involved the administration of the instruments described above, and usually lasted 1½–2 hours.

Results

The practical consequences of the patient's depression for the spouse

There were 83% of male and 42% of female relatives at work. Very few had had to make major changes in work routine because of the patient's illness, but nearly half said that they found work a strain as a result of their responsibilities towards the patient. Two women had been required to take up full-time employment because the husband's bipolar illness prevented him supporting the family.

There were 41% of relatives who said that the state of their finances was much worse since the patient became ill. Most loss of income came about because the patient was unable to work, and in the case of four male patients the drop in family income was over 50%. Families dealt with this by cutting down on leisure activities and holidays, and in a minority of cases by borrowing or owing money, breaking into savings, or cashing insurance policies. Nearly half of the patients' spouses had had to take over total responsibility for finances.

The social and leisure activities of relatives suffered considerably. There were 71% of spouses who had experienced at least some reduction in the number of their social activities as a result of the patient's illness. Nearly half said they never went out with the patient socially, although only one of the spouses of bipolar patients said this. This constriction of social activity was particularly marked when the patient was male. Spouses of unipolar and neurotic depressives also reported that the patient's depression meant fewer friends called to the house. In their view, friends had been put off by the behaviour of patients when ill, particularly by their silence or conversations limited to distressing or gloomy topics. A quarter of relatives now felt they had no-one to turn to for help or support, and half did not know of anyone in a position similar to themselves. Many were embarrassed and reluctant to tell people about the spouse's depression, being conscious of stigma.

Social isolation went hand in hand with the long stretches of time relatives spent in face-to-face contact with the patient, a mean of 65 hours a week, consistent across the three patient groups. There appear to be no normative data for comparison, but this mean exceeds what would be arrived at if the couple spent every evening and all weekend in the same room. Indeed, a third of relatives said they shared all their activities with the patient, although the proportion was smaller for bipolar patients and larger for neurotically depressed patients. Employment predictably reduced the amount of face-to-face contact.

Spouses were asked to assess overall changes in their marital relationship since the onset of depression. Such assessments clearly depend on a number of factors, including the duration, persistence and intrusiveness of symptoms and the spouse's capacity for coping with them. Nearly half the sample mentioned actual difficulties in their relationship (Table IV). Where difficulties were mentioned, the spouses of bipolar patients tended to rate them as less severe. Indeed, a minority of this group actually felt the illness had brought them closer together. The most likely reason for their relatives' tolerance is that only one bipolar patient was ill at the time of the study, although it could also have resulted from the intermittent course. Wives were more likely than husbands to perceive a deterioration of the relationship.

TABLE IV

Reported effects of depression on the marital relationship

	Total sample (n=24)	*Bi-polars (n=8)*	*Uni-polars (n=8)*	*Neurotics (n=8)*
More arguments	13	4	6	3
Spouse no longer talks about own worries	13	4	5	4
Less affection shown by patient	12	3	4	5
Difficulties in relationship	11	2	3	6
Difficulties in sexual relationships	10	5	3	2
No longer have sexual relationships	8	2	3	3
Have separated for a time	5	3	2	0

Spouses outlined the ways in which the relationship had changed: over half disclosed that they no longer felt able to talk to the patient about their own problems. This was because they didn't want to worry the patient, because their own worries appeared insignificant by comparison, or because they felt the patient would in any case be unable to offer support. More than half of the spouses, particularly wives, felt that they received less affection from their partners than before the illness. A third said that they had ceased sexual relationships as a result of the depression, and a further 42% reported difficulties in this area. Although a third of all spouses said they had difficulty keeping the relationship going, none of these was married to a patient with bipolar disorder. Nearly half the sample had thought of leaving at some stage, and it is of interest that these were mainly married to bipolar patients. Four husbands and one wife had actually separated from their spouses for a time.

TABLE V

Patient behaviours which spouses report as being problematic

Behaviour	*Spouses describing behaviour as a problem* *n*	*%*
Social withdrawal/complaining behaviour		
Misery	20	83
Underactivity	18	75
Worrying	17	71
Fearfulness/anxiety	17	71
Irritability	17	71
Withdrawal	14	58
Slowness	11	46
Overdependence	11	46
Self-neglect	10	41
Indecisiveness	8	33
Forgetfulness	8	33
Attention-seeking	6	25
Nagging/grumbling	6	25
Somatic/florid behaviour		
Sleep disturbance	20	83
Unpredictability	11	46
Somatic complaints	10	42
Odd ideas	10	42
Appetite loss	9	38
Rudeness	9	38
Offensive behaviour	8	33
Suicide threat/attempt	7	29
Overactivity	7	29
Odd behaviour	6	25
Obsessionality	5	21
Elated mood	5	21
Destructive behaviour	4	17
Hallucinations	3	13
Violence towards people	2	8
Heavy drinking	2	8

Nearly half the couples had no children, and this was more likely if the patient was female. Where there were no children, in around half of cases this was attributed to eugenic considerations, to the patient's presumed inability to cope, to loss of libido, or to a combination of these factors. Only a quarter of couples had children who were still living at home, and it was therefore difficult to gain any coherent idea of how child care was affected.

Problem behaviour

Spouses were asked to indicate whether the patient exhibited any of 30 types of behaviour and whether these were a problem. The rating of a symptom as a problem depended on a combination of its persistence, duration, and intrusiveness but was probably also related to the spouse's ability to cope. Problem symptoms were of two broad types: social withdrawal and complaints; and somatic and florid types of behaviour. Apart from sleep disturbance, which 83% of spouses identified, the types of behaviour which were most often a problem fell into the first category (Table V).

The more florid features of depression were rarer and therefore less often seen as a problem. Not surprisingly, it was the spouses of bipolar patients who mentioned problematic florid symptoms. Many of the most commonly identified types of behaviour were also rated as severe in degree, but this was also true of the florid symptoms exhibited by bipolar patients.

As might be expected, the tendency to see behaviour as resulting from illness was greater for florid symptoms than for negative symptoms and complaints (Table VI). Appetite loss, odd and destructive behaviour, hallucinations, and violence towards others were not perceived by any relative as being under the patient's control, whereas other behaviour types were seen by a varying proportion of relatives as personality attributes.

TABLE VI
Relatives' causal attributions of patient's behaviour

Behaviour	*Due to illness: n*	*Under patient's control: n*
Worrying	10	13
Irritability	11	11
Indecisiveness	11	11
Attention-seeking	10	14
Nagging/grumbling	7	17
Obsessionality	7	17
Somatic/florid behaviours		
Sleep disturbance	18	5
Odd ideas	17	4
Appetite loss	24	—
Rudeness	19	5
Odd behaviour	24	—
Elated mood	19	2

Coping

Many spouses used what might be called cognitive coping strategies, such as "wish the situation would go away" (92%) or "look for the silver lining" (58%). A third of spouses criticised or lectured themselves, and 29% made themselves feel better by eating, drinking, or smoking more.

Almost half of the spouses interviewed (45%) felt that they had no idea what to do in a practical sense to deal with the mood disturbance of the patient and its consequences for their lives, and in the case of relatives of neurotics this figure rose to three-quarters. Two-thirds of the total sample felt that they were unable to see things from the patient's point of view. Among relatives of patients with bipolar affective disorder, this figure was only a quarter, but the large majority of relatives of neurotic and unipolar depressives felt this way. At the same time, three-quarters of those interviewed reported that they did not talk to anyone to find out more about the situation.

The spouses of bipolar patients frequently reported that things had been difficult during the first episode as they did not understand what was going on. At that time, they often had particularly bad experiences – through the wild expenditure of the patient, for instance. They did, however, learn through these experiences, both to spot earlier when the patient was becoming ill, and to take precautions like assuming charge of the cheque book when it was apparent the patient was relapsing. It appeared these spouses found it easier to cope because episodes were distinct, even though they felt as if they were living with a different person at those times: their husband or wife would 'return' between episodes.

Half the spouses reported that they were kept going by the hope that the patient would be "cured" one day and 29% felt that their spouse would be "OK any day now". A third of spouses said that they sometimes felt they could not cope with the situation any longer and would have to find a way out, and three reported that they felt like that all the time. Such feelings were considerably commoner in wives than in husbands.

Psychological strains and adjustments in relatives

Relatives were asked about the psychological adjustments they had to make as a result of the patient's depression. Such adjustment could be related to changes in the patients, to consequential changes in the spouses' circumstances, or to changes in themselves.

Two-thirds of the sample reported feeling that the patient was a completely changed person as a result of the depression. Over half experienced a sense of loss, as if they had lost the husband or wife they had once had. This was least marked among the relatives of bipolar patients and most prominent among those of neurotic depressive patients and is therefore probably the result of relatively unremitting symptoms. The vast majority (88%) of relatives felt that they wanted to stay with the patient in spite of all these changes, although for some this came from a feeling of duty. Dissatisfaction with the changes was strongest among the relatives of bipolar patients. Nearly half of the spouses felt that the patient had become like a child to them, someone who needed to be looked after.

Almost three-quarters of relatives, particularly those married to neurotically depressed patients, reported that some of their hopes and dreams for the marriage had remained unfulfilled because of the patient's illness. Despite this, only two spouses reacted bitterly. Grief, guilt and sorrow were commoner reactions, each experienced by a third of the total sample, although not by the relatives of bipolar patients. A sense of grief was much commoner in wives (58%) than husbands (8%), the only reaction in which there was an appreciable sex difference.

The relatives of bipolar patients rarely perceived that changes had been wrought in themselves, whereas this was almost universal in the rest of the sample. However, the reported changes were very varied, falling into three categories. Some changes made the situation easier to bear: arguing less, giving in more often to the patient's wishes, thinking more before saying things. Others represented some kind of personal growth: becoming more independent, assertive or mature, being able to understand other people's worries better. Some changes, however, involved deteriorations in physical and mental health.

Nine spouses (37.5%) scored above level 5 of the Index of Definition. Bebbington *et al* (1981) give values of 10.9% from a community sample in Camberwell. However, several of the families in the present report came from more affluent, outer London areas, and a lower rate than 10.9% might therefore have been expected (Table VII). However, none of the relatives of bipolar patient were cases themselves.

Knowledge about the nature and cause of the patient's condition

Most spouses attributed the patient's difficulties to something external - mental illness (42%), "nerves" (21%), or depression (12.5%). Three spouses felt that the patient had always been "that kind of person" and two could not say what was wrong with the patient. These five were all married to neurotically depressed patients. Three-quarters of the relatives of patients with bipolar affective disorder felt that their spouse was suffering from some form of mental illness, as compared with a quarter of relatives in the other two groups. Husbands were more prone than wives to use vague terms, like "trouble with nerves". The most common cause of the patients' difficulties given by relatives was that "things got too much for them" (42%). One-third of the sample had no idea what caused the patients' difficulties.

Relatives were asked whether they saw the patient or themselves as having any control over the problem. Nearly all the relatives thought that they themselves had little or no influence on the patient's condition, and two-thirds felt that the patient had little or no control over it either. Nevertheless, a small number in each group did think that the patient had some control.

Asked whether they could distinguish normal mood swings in the patient, or if changes in mood always meant that the patient was becoming unwell again, over half the sample (58%) reported that they were able to distinguish changes in mood which were not illness-related. However, among the spouses of unipolar depressives, 62% felt that changes in mood always meant the patient was becoming unwell again.

Services provided, services suggested

Relatives were asked about the amount of general information they had been given regarding the nature of the patient's difficulties, what had caused them, and what the outcome was likely to be.

Over half of the spouses reported that they could not remember having been provided with any information of this kind. Five spouses (21%) said they had received some information, three (12.5%) had received quite a bit, but only two spouses felt that they were given sufficient information to enable them to understand fully the patient's difficulties. The percentage of spouses who reported that they were provided with no information differed between the three patient groups. Thus, just over a third of the relatives of bipolar depressive patients, almost two-thirds of those of unipolar depressives, and three-quarters of those of neurotic depressives reported that they did not receive any information. Men were noticeably less likely to report that they had been provided with information about their spouses' problems.

Nearly all spouses said that they had never been told anything about the kind of behaviour to expect from the patient as a result of the illness, and the two (8%) who were provided with a little information in this regard were relatives of bipolar depressives. Nobody in the entire sample could remember ever having been given any advice on how to deal with patient behaviours they found difficult to cope with.

Half of the relatives interviewed did not feel that they could contact the hospital if they were worried or needed advice on matters relating to the patient. A further 42% felt that they could contact the hospital but had never actually done so. Only two relatives (8%) reported that they had actually contacted somebody at the hospital when they were worried about something. In contrast to this, almost everybody (92%) said that they had no doubts about being able to contact the hospital in times of crisis.

Only three relatives said that they were fully satisfied with the services they had received from the hospital. The remainder were either not satisfied at all with the service the hospital provided, or felt that there was much room for improvement.

TABLE VII
Relatives' CATEGO class (ID5+)

	Total sample		*Bipolars*		*Unipolars*		*Neurotics*	
	Number	*%*	*Number*	*%*	*Number*	*%*	*Number*	*%*
Neurotic depression	7	(29)	—	—	4	(50)	3	(38)
Anxiety state	2	(8)	—	—	2	(25)	—	—
Total	9	(38)	—	—	6	(75)	3	(38)

TABLE VIII
Relatives' suggestions for services

Services	*Relatives (n = 24)* *Number*	*%*
More information and consultation	20	(83)
Advice on how to deal with patients	8	(33)
Meetings with other relatives – group or individual	7	(29)
Leaflets/books/films	4	(17)
Earlier admissions	4	(17)
Reassurance that patient would get better	3	(12)
Practical help on entitlements, benefits etc.	3	(12)
To be seen promptly at appointed time in the out-patient department	3	(12)
Break or rest from the patient	2	(8)

The final question put to relatives was what they thought professionals should be providing for them, and what they would find helpful in their present situation. The replies are shown in Table VIII.

Discussion

This study is based on small numbers and its findings must be assessed within that limitation. However, its importance lies in describing the sorts of problems faced by those living with patients suffering from persistent depressive disorders. If the relative of a given person suffering from depression might share the sort of situation experienced by the spouses in our sample, the awareness of the mental health professional should be directed towards that fact. Humane and knowledgeable questioning will then establish details and provide the basis for any intervention necessary for the well-being of the patient and the relative. This being said, we would nevertheless be surprised if some degree of burden is not a commonplace among the relatives of depressive patients, at least where the disorder is of some duration and persistence.

Some of the relatives originally canvassed could not be interviewed because they, the patient, or the consultant withheld permission. This may have happened precisely because of particular difficulties between the patients and their spouses.

The patients selected were deliberately chosen because their illness had started at least two years previously. This means the findings here cannot be generalised to more short-lived and recent illnesses, and that there is no information about how relatives are affected by the onset of illness nor of how they change in their behaviour over time.

Our conclusions must be that relatives *are* affected and *do* bear burdens as a result of the patient's depressive illness. Although they rarely complain, these effects may be far-reaching. The enormous amount of time which relatives spent in the presence of the patients was probably both a cause and a consequence of the notable curtailment of leisure activities. Many spouses objected to this "smothering". Henderson *et al* (1981) pointed out the restricted social networks of those with affective disorders. The current study supports the claim of Nelson *et al* (1970) that this restriction also extends to the patients' spouses.

Marital and sexual relationships were badly affected. It is, of course, possible that marital difficulties may have been involved in the initiation of the depression, but the nature of the changes recorded and their apparent timing must lend some credence to the possibility that they were consequences of the depressive disorder. Spouses took over various roles the patient would normally be expected to carry out, and wives found this particularly difficult. These results confirm those of Collins *et al* (1971), who describe conflict over role functions in couples where the husband was depressed.

Relatives lost the facility of confiding, and commonly took decisions on their own. Their expectations of the relationship were generally reduced and many, particularly women, had a sense of bereavement as a result. There was an increased rate of minor affective disorder among relatives. This could have been due to assortative mating, but the evidence for this has been doubted (Kreitman *et al*, 1970; Merikangas, 1982). It has been claimed that the combination of stressful circumstances and the lack of someone to confide in is particularly detrimental (Brown & Harris, 1978). Our sample of relatives was fairly clear why they were depressed: as one husband said, "her depression is catching".

In contrast to the effects on the marital relationship and the situation at home, performance at work was relatively little affected. In fact, for some relatives, work provided their only break from being with the patient. However, the effect on finances could be very marked, especially where a male patient could no longer function as breadwinner.

The symptoms that relatives most frequently found burdensome were the 'negative' ones, such as worrying, irritability, and nagging. Because they overlapped with normal patterns of behaviour, these were frequently not seen by the relative as arising from the patient's depression. This is similar to findings in the relatives of those with schizophrenia (Creer & Wing, 1974), who complained less frequently; of florid than of negative symptoms. Florid symptoms also cause difficulties, but they are intermittent and less frequent, and the major proportion of the burden occasioned by symptoms seems to come from the negative ones. This may not be appreciated by those responsible for the care of the patient.

Although all groups of relatives were affected, those of bipolar patients were less so. This was partly because most bipolar patients were not disturbed at the time of the interviews, partly because the episodic nature of the condition meant that relatives obtained at least some relief, and partly because the symptoms were more easy to distinguish from ordinary behaviour – it was more obvious that something was wrong. There is no doubt that relatives were upset at times of acute disturbance. It is probable that psychiatrists find it easier to inform and advise the relatives of those with bipolar affective illness.

Female relatives tended to be more affected than males. They experienced an additional burden if they were faced with having to become the chief earner. They tended in any case to have fewer social contacts. Many expressed the strain involved in having to take over untraditional roles. Our findings are tentative, but such gender differences would seem a useful subject for future research.

Relatives' views of the service they received from those caring for the spouse require little additional comment. The main areas of complaint concerned deprivation of information or support, and difficulties over admission.

These findings, however qualified, have implications for services. The management of depression should always be designed to take account of the family setting, and people ought to be involved in decisions about care for their depressed relatives. Relatives could be offered the information and support they lack without major changes in existing services. However, more structured interventions could also be considered, like those used in studies of the families of schizophrenic patients (Falloon *et al*, 1982; Leff *et al*, 1982).

Our results argue for the provision of a service for the relatives of depressed patients on grounds of simple humanity. Such a service is likely to have the additional benefit of improving the lot, and possibly the prognosis, of the patients themselves.

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Neural Substrates of Violent Behaviour

A Preliminary Study with Positron Emission Tomography

NORA D. VOLKOW and LAURENCE TANCREDI

Brain function was evaluated in four psychiatric patients with a history of repetitive purposeless violent behaviour, using EEG, CT scan, and positron emission tomography (PET). Three patients showed spiking activity in left temporal regions, and two showed CT scan abnormalities characterised by generalised cortical atrophy. The PET scans for the four cases showed evidence of blood flow and metabolic abnormalities in the left temporal lobe. Two patients also had derangement in the frontal cortex. The patients showing the largest defects with the PET scans were those whose CT scans were reported as normal. This paper shows the utility of PET in investigating possible brain derangements that could lead to violent behaviour.

The distinction between violence and aggression is an important one. Aggression is a normal physiological behaviour that allows the organism to survive in its environment. Violence, on the other hand, is a form of aggression where the adaptive role has been lost, and which may reflect dysfunctions of the neural mechanisms associated with aggression (Williams, 1969; Yeudall, 1977; Monroe, 1981; Joest, 1983; Krieger, 1985).

Studies of violence have shown that subjects who commit repetitive acts of violence show higher incidence of abnormal brain function as evidenced by neuropsychological tests, electroencephalogram (EEG), and computerised tomography (CT) scan (Yeudall *et al*, 1982; Lorimer, 1972; Williams, 1969; Krieger, 1985). These abnormalities tend to be localised mainly in three brain areas: the frontal cortex, the amygdala, and the temporal cortex. These cerebral regions coincide with the areas implicated in the genesis of aggression in animal studies (Goldstein, 1974; Albert & Walsh, 1984; Mirsky & Harman, 1974).

Positron emission tomography (PET), a new imaging technique that allows direct investigation of brain function (Phelps *et al*, 1982; Volkow *et al*, 1985), may be of help in detecting cerebral abnormalities unrecognised by other imaging or electrophysiological techniques.

In this study we investigated the brain characteristics of four individuals with a history of recurrent violent behaviour, using PET, CT scan and EEG.

Method

We selected four patients from the in-patient psychiatric unit who had had at least three episodes of legal arrest because of violent behaviour. All of the patients were right-handed males and had an additional DSM-III (American Psychiatric Association, 1980) diagnosis for mental disorder. On admission, patients had a full psychiatric, neurological, and physical examination. All subjects had a CT scan with and without contrast, and an EEG performed with and without photic stimulation. All subjects were investigated with PET using ^{15}oxygen-labelled water for determination of cerebral blood flow and ^{18}fluorodeoxyglucose (FDG) for determination of glucose metabolism. The

TABLE I

Ratios left temporal/right temporal (LT/RT) and frontal/occipital (F/O) of the violent patients for FDG and CBF, and LT/RT and F/O for CBF of normal controls

	Patients				*Normals*	
Case	*LT/RT FDG*	*LT/RT CBF*	*F/O FDG*	*F/O CBF*	*LT/RT CBF*	*F/O CBF*
1	0.86	0.84	0.62	0.55	1.00	0.90
2	0.87	0.82	0.67	0.66	0.97	0.78
3	0.91	0.87	0.79	0.81	0.93	0.93
4	0.90	0.84	0.94	0.86	1.00	1.04
X	0.88	0.84	0.75	0.72	0.97	0.91

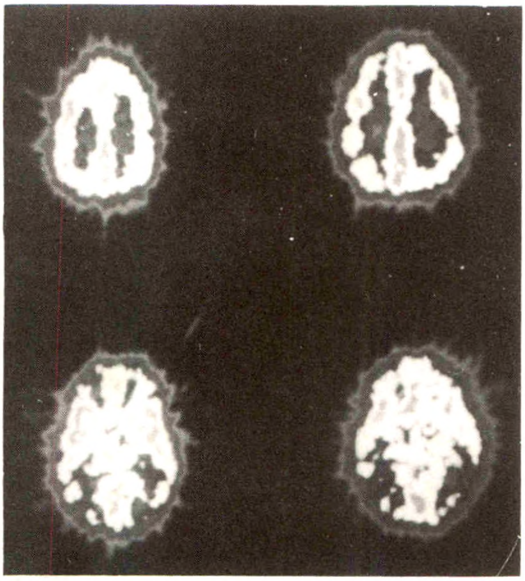


FIG. 1 Typical image for cerebral blood flows in a normal subject. The upper left and right images are at the level of the centrium semiovale; the lower left and right are at the level of the basal ganglia. All the images are axial planes. Note the high level of activity in the prefrontal cortex and the relative symmetry for left and right flow. (Left side of the image corresponds to left hemisphere.)

subjects were tested under baseline conditions (eyes open and ears unplugged). The studies were done using the TOFPET instrument (FWHM 1.2 cm) (Mullani *et al*, 1984). An initial attenuation image was obtained using a ring filled with ^{68}gallium, and a total of 200 million events were collected. The measurement of regional cerebral blood flow (CBF) was obtained after an intravenous bolus injection of 30-35 mCi of ^{15}oxygen-labelled water (Raichle *et al*, 1983). For this image, scanning was started immediately after injection of the label. The images were reconstructed by integrating the radioactivity detected from time of entry of the compound into the brain until peak counting was achieved (30-35). Measurement of glucose metabolism was obtained after an injection of 8-10 mCi of ^{18}FDG (Reivich *et al*, 1982). Scanning was started 40 minutes after injection and was continued for a total of 20 minutes.

PET images were analysed by comparing the left temporal versus the right temporal and the frontal versus the occipital cortex of the three slices centered at the thalamic level. The regions of interest were drawn directly on the PET images and the values obtained were an average of the total number of counts detected for the region in the three slices. Ratios were plotted both for the FDG and for CBF.

As a reference, a similar procedure was done in the CBF images obtained from four normal volunteers matched for age, sex, and handedness.

Patients

Case 1 was a 30-year-old white male, in hospital because of suicidal and homicidal ideation. The patient had a diagnosis of bipolar affective disorder and post-traumatic stress disorder. At the age of 25 he suffered a brain concussion, with loss of consciousness. Since then he had abused alcohol regularly. During intoxication he became extremely violent and had several incidents of physical assault, including homicide. The patient did not report guilt or repentance for his actions. He was treated unsuccessfully with various antidepressants, minor tranquillisers, and neuroleptics. The family history was positive for alcoholism in both parents, and for depression in the father.

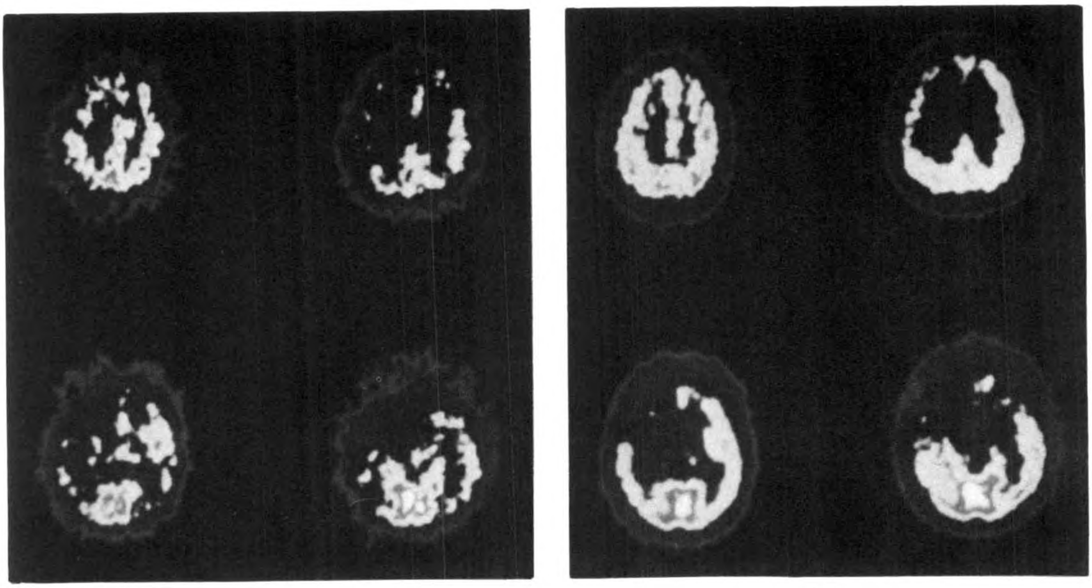
Case 2 was a 45-year-old black male in hospital for agitation and violent behaviour. Because of similar previous episodes, the patient had been misdiagnosed as chronic schizophrenic. He had been in prison for repeated incidents of violent, impulsive behaviour, including physical assault, arson, and rape of his sister. He did not seem to have an understanding of the ethical implications of his behaviour. There was no psychiatric history in the family. While in hospital, the patient's violent behaviour decreased with doses of neuroleptics.

Case 3 was a 21-year-old white man in hospital after a suicide attempt, with a diagnosis of borderline personality. At the age of 15 the patient began to exhibit outbursts of violent behaviour. During these episodes he experienced intense rage and lost control of his behaviour, and afterwards had no recall of the event. He also had a history of impulsive suicide attempts; by hanging and by drug overdose. The patient had a positive history of alcohol and drug abuse, mainly marijuana and phenobarbital. The family history was positive for paranoid schizophrenia and epilepsy in two of his three siblings. The patient was treated with Tegretol, with some resolution of the impulsive destructive behaviour.

Case 4 was a 39-year-old white male in hospital because of outbursts of uncontrollable violent behaviour. The patient had a past history of post-traumatic disorder and alcohol abuse. Since his return from Vietnam in 1968 he had had several episodes of uncontrollable violent attacks while intoxicated with alcohol. On one occasion, he shot a gun at his father; on another occasion he attempted to strangle a priest. He also made several suicide attempts. At the age of 30 the patient had a three-year history of generalised motor seizures, which were treated with Dilantin. He had been treated unsuccessfully with minor tranquillisers and antidepressants. There was a psychiatric history in the family. While in the hospital, the patient was treated with clonazepam, with resolution of the panic attacks and violent behaviour.

Results

Table I gives the left/right (L/R) and frontal/occipital (F/O) ratios for the FDG and CBF images of the patients and for the CBF images of the control subjects. All of the controls showed higher L/R values than the patients. Except for one of the control subjects, they also showed higher F/O CBF ratios than the patients. Of the patients, cases 1 and 2 had the lowest L/R and F/O ratios both for FDG and CBF. Figure 1 shows a typical image for CBF of a normal subject.



(a) (b)

FIG. 2 Case 1: images for (a) CBF and (b) FDG. Note the severe disruption in the frontal cortex, and left temporal area. Planes are similar to those of Fig. 1.

In case 1, the EEG revealed left anterior temporal sharp and slow wave activity, suggestive of an irritative lesion with epileptogenic potential. The CAT scan was reported as normal. The PET scan showed a defect of brain metabolism and blood flow on the left hemisphere, which was more accentuated for the left temporal cortex and frontal area (Fig. 2).

In case 2, the EEG and the CT scan were reported as negative. The PET scan showed decreased metabolism and blood flow on the left temporal cortex and the frontal cortex (Fig. 3).

In case 3, the EEG reported brief runs of left temporal sharp slowing consistent with an irritative lesion. The CT scan reported accentuated cortical atrophy. The PET scan showed a slight decrease in cerebral blood flow on the left temporal cortex (Fig. 4).

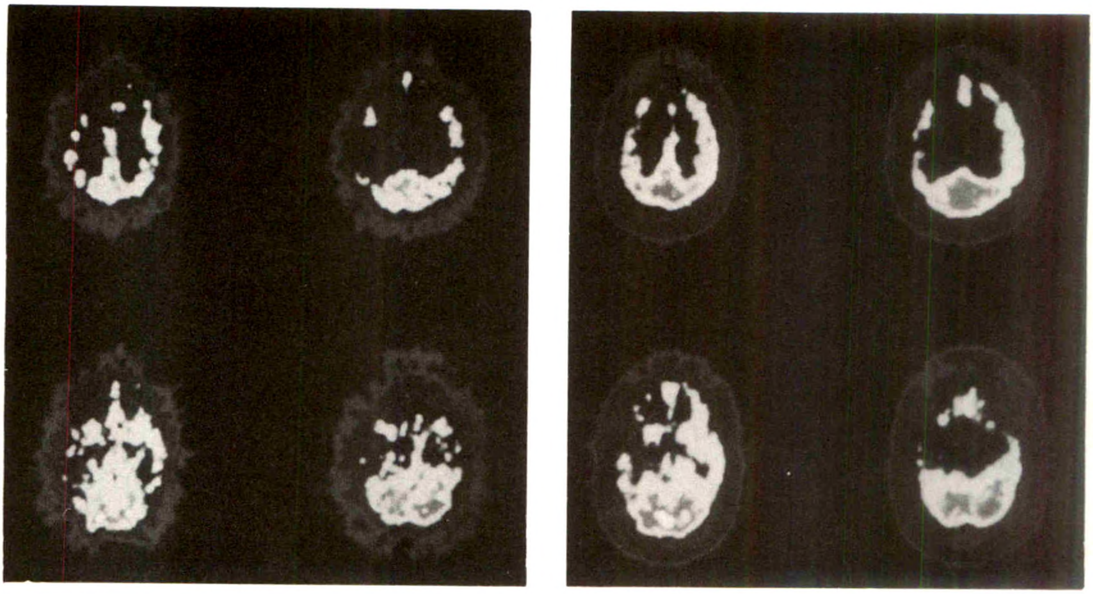
In case 4, the EEG reported left temporal slowing and sharp waves as well as excessive delta wave activity more marked for the posterior regions, suggestive of mild diffused disturbance of cerebral function. The CT scan showed atrophic changes out of proportion for age. The PET scan showed a mild decrease of blood flow for the left temporal area. The right temporal region showed an area of increased CBF (Fig 5).

Discussion

These four patients had in common a history of repetitive purposeless violent behaviour. Cases 1 and 4 showed violent behaviour mainly under the influence of alcohol. All of these patients showed temporal cortex asymmetry, with the left showing lower blood flow and metabolism than the right. Cases 1 and 2 also showed severe flow and metabolism disruption on the frontal cortex. Cases 1 and 2 appeared to show no remorse for their actions, whereas cases 3 and 4 regretted their violent behaviour after its occurrence. Cases 1 and 2 showed larger areas of defect on metabolism and CBF than cases 3 and 4, yet their CT scans were normal, whereas cases 3 and 4 showed minimal metabolic and CBF dysfunction and their CT scans revealed marked cortical atrophy. The EEGs of cases 1, 3 and 4 showed spiking activity in left temporal areas.

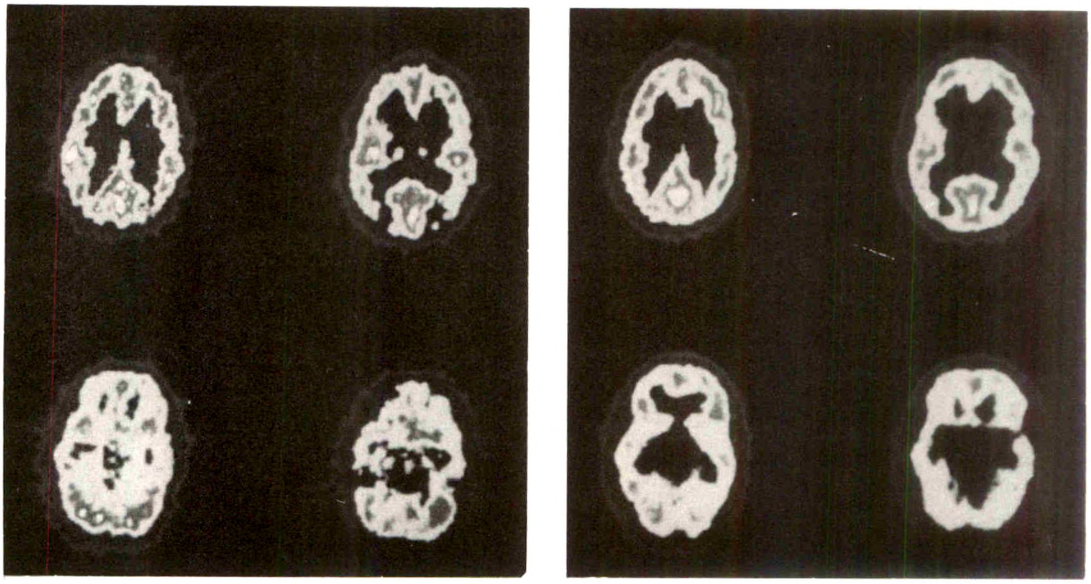
These cases illustrate the complexity of trying to understand cerebral derangements associated with violent behaviour. Rather than a particular focal brain lesion, these patients showed widespread defects in cerebral function, as evidenced by PET; all revealed evidence of left temporal dysfunction. These regional defects were not detected with the CT scan, which, if abnormal, showed only cortical diffuse atrophy. Cerebral dysfunction in this group of violent individuals, shown by the higher sensitivity of PET over CT scan, suggests that organic brain dysfunction in violent offenders may be more frequent than was previously recognised.

Analysis of the violent act is of importance in understanding possible neural system implication in



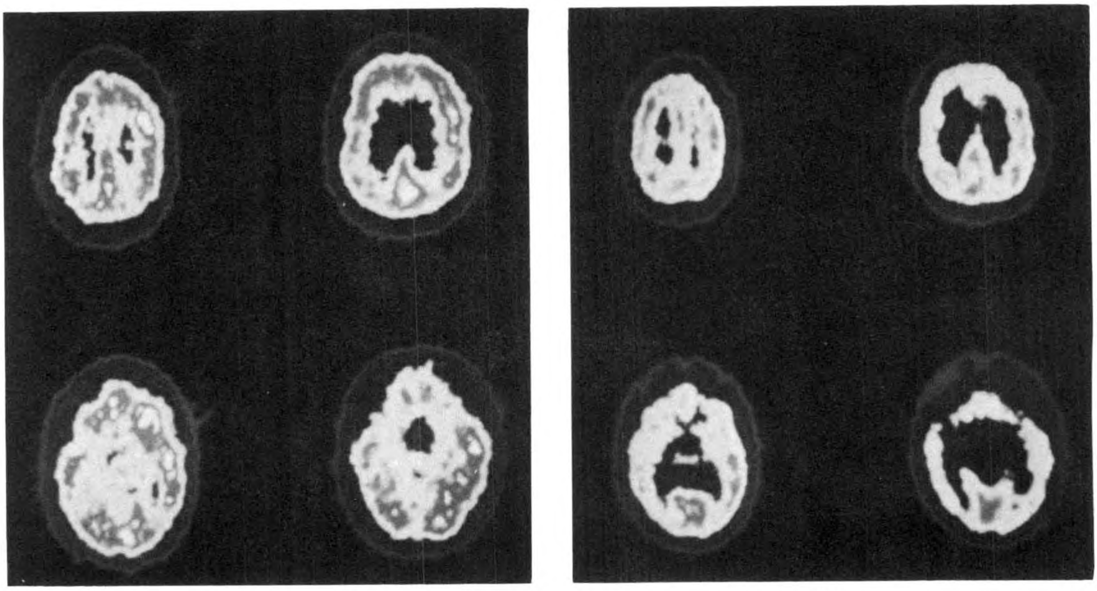
(a) (b)

FIG. 3 Case 2: images for (a) CBF and (b) FDG. Note the defects of perfusion and metabolism in the frontal areas and left temporal cortex. Planes are similar to those of Fig. 1.



(a) (b)

FIG. 4 Case 3: images for (a) CBF and (b) FDG. Note the decrease in blood flow in the left temporal area. Planes are similar to those of Fig. 1.



(a) (b)

FIG. 5 Case 4: images for (a) CBF and (b) FDG. Note the left/right asymmetry, with decreased left temporal activity. Planes are similar to those of Fig. 1.

its occurrence. At least three basic syndromes associated with neurological impairments have been described for the occurrence of violent behaviour. Firstly, defects in the prefrontal area, which is implicated in higher cognitive functions like consciousness and abstraction (Luria, 1966), could facilitate the occurrence of violent behaviour because of inability to understand concepts such as right or wrong or to appraise the consequences of the violent act. Secondly, abnormalities in the limbic areas (including medial temporal cortex), which are responsible for generation of affects and emotions, could elicit feelings that arise *in vacuo* (that is, independent of an evoking condition) (Blumer *et al*, 1974). This type of patient will tend to show random outbursts of rage and violence (Wieser, 1983), with very poor impulse control (Mungas, 1983). This subtype of violent individual appears to have a higher incidence of EEG abnormalities. Thirdly, abnormalities in the association areas of the brain which lead to derangements in perception would evoke assaultive behaviour when a stimulus is perceived as threating (Valzelli, 1981).

It is important to emphasise that we do not attribute the violent behaviour of these cases solely to brain abnormalities, but rather that the occurrence of the violent outbursts was facilitated by the cerebral dysfunction. Most of the violent behaviour seen in patients probably represents complex interactions between various neural systems, neurotransmitters, hormones, environmental stimulus, and learned responses (Mednick, 1982).

New imaging techniques such as PET allow us to detect cerebral pathology which was henceforth unrecognised by the EEG and the CT scan. PET, being designed to investigate cerebral function, is a very promising tool to help evaluate cerebral derangements in individuals with violent behaviour, and hence may help in the design of better preventive and therapeutic interventions.

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A Ten-Year Follow-Up of Men Discharged from Grendon Prison

GRAHAM ROBERTSON and JOHN GUNN

A ten-year follow-up on the criminal records of men discharged from Grendon Prison was carried out. These records were compared with those of a matched control group, and no differences were found regarding frequency or severity of post-discharge convictions. The validity of this exercise is questioned, and several case histories presented. An examination of subsequent offending within the Grendon group suggests that for some men, the better motivated and more intelligent, exposure to the Grendon regime may have contributed to a change in their criminal behaviour.

Grendon prison is an important psychotherapeutic unit within the prison system of England and Wales. It was originally proposed before the Second World War in a Home Office report (East & Hubert, 1939) and opened in July 1962. Its history and its therapeutic system have been described previously (Gunn *et al*, 1978), and its operation has recently been reviewed by a Home Office working party (Home Office, 1985). The regime in Grendon is a modified therapeutic community. It is not a hospital as defined by the Mental Health Act, thus it does not take offenders/patients given hospital orders under the Act. Offenders are not accepted direct from courts, but only after selection and preliminary investigation by medical officers in the prison service. Such prisoners must not be suffering from psychotic illness, must be willing to co-operate in the regime, and can apply to be returned to an 'ordinary' location if they wish. For most of its life the prison has been under the directorship of a medical director, but at time of writing and for the last few years has had a lay governor as its head.

Background

In the early 1970s, the Home Office commissioned the Forensic Psychiatry Section of the Institute of Psychiatry to evaluate the regime and its effects. The original study population comprised the intake to Grendon over a 12-month period, numbering 107 men. Most were in their middle-to-late twenties and had long criminal records. Many men had no home at all to return to after release, and there was no indication that the Grendon authorities were selecting men with a good prognosis in terms of their chances of being reconvicted.

In the original follow-up study 61 men were involved, and each was matched to a man in another prison who was not receiving psychiatric treatment. The Parole Index was used for matching. This is a systematic collection of demographic data on all convicted prisoners serving sentences of 18 months or more. Consequently, we were able to match on those variables of most importance in criminological prediction. Details are provided in Gunn *et al* (1978). Although well matched, the two groups also differed in some important respects. For example, when compared on their attitudes to treatment and to psychiatry, 88% of the Grendon men expressed some or a considerable desire for psychiatric help, compared with only 46% of men in the control group.

The ten-year follow-up study

The purpose of the present paper is to report the results of a ten-year criminological follow-up of the cohort previously studied. In our original report, we argued that reconviction rates were an inappropriate measure of Grendon's performance. Statistically, the variance contributing to reconviction in a given period is enormous, involving as it does the totality of the man's previous experiences, as well as huge error or random components, not the least of which is the difference between committing offences and being caught and convicted for those offences. It was argued that a fixed and limited period of time spent in prison X as opposed to prison Y constituted a tiny fraction of the variance which might be held to contribute to the criterion measure of reconviction, and that the measurement of its contribution was confounded by lack of control over these more potent contaminating variables.

Nonetheless, we were interested in finding out what had happened to the men we had interviewed in Grendon and to those men who comprised the control group for the Grendon cohort. Earlier efforts to keep in touch with these men when they left prison had proved very difficult. Six months after release, the response rate to the questionnaires we sent to them had fallen to 41%, and by 12 months the response rate was in single figures. We reluctantly therefore had to fall back on official records and, predominantly, criminal records.

Method

The criminal records of all men in the follow-up groups were obtained from the Criminal Records Office. The National Health Service Central Register supplied information about those men who had died, including the nature of any deaths that had occurred. These data were added to the information available from the original study, enabling post-discharge criminal variables to be correlated with the information we had obtained ten years earlier.

Results

The majority of men in both the Grendon and control groups had been reconvicted at some point during that ten-year period (92% and 85% respectively, $\chi^2=1.37$, d.f. 1, NS). In that time the Grendon group had made, on average, 5.3 appearances in court and the control group 4.5 ($t=0.80$, NS). Offences of serious violence (and we have included robbery in this category) were committed by 20% of the Grendon group and 17% of the control group. Between 60% and 70% of both samples had received a custodial sentence, and in this category we have included two men in the control group who had been sent to a special hospital under a hospital order.

The types of offence covered the full range of criminal behaviour. As would be expected, the most serious types of crime were rare (homicide, rape, grevious bodily harm and arson), and the only category of offence of numerical significance concerned acquisitive offending. In the Grendon group, theft represented the only type of crime committed by a majority of men. This was true also for the control group. Convictions for drug offences were significantly more common among the Grendon men, 13 (22%) of whom had received such a conviction compared with only one man (2%) in the control sample ($\chi^2=11.43$, d.f. 1, $P<0.001$). It is not surprising to find the two groups differing in this respect on follow-up, as despite exhaustive efforts to match these groups in the original study, the Grendon sample contained significantly more men with a known history of drug abuse prior to their participation in the study (19% of the Grendon men had convictions for drug-taking compared with 3% in the control group; $\chi^2=6.82$, $P<0.01$). The removal of all drug offenders from further analysis made no difference to the other results.

Post-discharge offending within the Grendon sample

As our main interest here is in Grendon and in the association between changes which had taken place there and post-release behaviour, only the Grendon population has been used for the remainder of the analysis. We examined the data to determine what variables were associated with recorded post-discharge criminality. For this purpose, two criteria were used: (a) the number of subsequent court appearances made (as a reflection of frequency of offending) and (b) the amount of imprisonment awarded by courts in that period (as a reflection of seriousness of re-offending).

TABLE I
Variables which correlated most highly with length of subsequent imprisonment in the Grendon sample

Variable	*r*	*P*	*n*
Criminal profile			
Our rating of violence	0.28	0.015	60
Our rating of drink-related offending	0.32	0.007	60
Personality characteristics			
History of disturbed personal relationships	0.29	0.012	60
Impulsiveness	0.29	0.012	60
Mental health assessment			
Alcohol problem	0.27	0.018	58
Depression	−0.31	0.009	58
Self-ratings			
Self-esteem	−0.25	0.039	51
Response to item 2 on the AC scale, "I like mixing with criminals"	−0.30	0.009	60

Table I presents the variables found to be associated with our measure of the seriousness of subsequent offending. The list has been restricted to items which correlated at greater than the $P=0.05$ level with the criterion variable. The variables in the table can be grouped into four categories: (a) alcohol abuse; (b) impulsiveness/violence; (c) low scores on the assessment of depression at mental state examination; and (d) low self-esteem. Item 2 of the Attitude to Criminality (AC) scale states, "I like mixing with criminals", and was negatively correlated with extent of subsequent imprisonment (those men who said they liked mixing with criminals were likely to have spent less time in prison). This is a rather unexpected result, but it may be that to assert a dislike for mixing with criminals is positively linked to self-esteem.

Table II presents the most statistically significant correlates of the number of court appearances made following discharge from Grendon. As with our measure of seriousness of reconviction, various indices of drinking behaviour were found to correlate significantly with the number of further convictions recorded. In the case of the frequency of post-discharge court appearances, variables such as vagrancy and low drive (defined in the study as lack of normal ambition and purpose) were associated with the criterion variable, rather than the violence and impulsiveness which correlated with length of subsequent imprisonment. At interview, we had assessed each man's propensity to lie,

TABLE II

Variables correlated highly with number of post-discharge court appearances in the Grendon sample

Variable	*r*	*P*	*n*
Personality characteristics			
Lying	0.32	0.006	60
Vagrancy	0.32	0.006	60
Low drive	0.41	0.001	60
Global rating for personality dysfunction as consequence of illness	0.27	0.017	60
MMPI scales			
Lie scale	0.27	0.03	52
K scale	0.29	0.017	52
Hostility	−0.26	0.034	52
Self-rating			
Total score on the 'Attitude to Psychiatry' scale	−0.38	0.001	60
Myself (semantic differential)	−0.25	0.022	52
Intelligence			
AH4 part 1 (verbal/ numerical)	−0.33	0.010	51
AH4 part 2 (spatial)	−0.48	0.0001	51
Previous criminality			
Total number of pre-Grendon court appearances	0.43	0.0001	60
Number of previous convictions for drink offences	0.28	0.016	60

and this rating was found to be associated with number of subsequent reconvictions. The Lie scale of the Minnesota Multiphasic Personality Inventory (MMPI) was also linked with reconviction, but to a smaller extent ($r = 0.27$, $P < 0.03$). One of the most statistically significant findings was the relationship between IQ and further convictions. The test used to measure intelligence, the AH4, is divided into two parts. The first part consists of verbal and numerical items, which in part reflect the educational level of the individuals completing the tests. The second part is much less dependent on education, consisting as it does of items requiring the manipulation of spatial material, and it is this part of the AH4 which correlates most highly with our criterion variable ($r = -0.48$). Attitude to psychiatry, as expressed on a rating scale, was also linked with frequency of reconviction, poor motivation being associated with reconvictions. It is noteworthy that mental state variables, including changes in Grendon, were not associated with post-discharge offending.

Relationship between attitudes towards psychiatric treatment and post-discharge criminality

In an earlier report (Robertson, 1974), it was stated that Grendon staff believed there to be a relationship between motivation and 'doing well' after release. In particular, it was supposed that men who were more motivated when they came to Grendon did well as a group; that those not well motivated did not do well; and that those who, although poorly motivated on arrival, improved in this respect did best of all.

In order to examine the first of these hypotheses, namely that men who came to Grendon with better motivation did better than those who were less well disposed to psychiatric treatment, the population was broken down into three groups on the basis of their Attitude to Psychiatry (AP) scale scores. Results are presented in Table III. Although not distinguished from one another in terms of the number of pre-Grendon court appearances made, nor indeed on any other of the major criminal variables, such as age at first conviction ($P < 0.71$) or the extent of their juvenile offending ($P < 0.83$), the group of men who had expressed a low opinion of psychiatry had made significantly more court appearances in the period following their discharge. This was supported by a related finding. In the original study, prison officers and other group leaders were asked to rate motivation for each man on a four-point scale; and the authors carried out a similar exercise after interviewing each man. Both sets of ratings correlated with frequency of court appearance after discharge, but not with subsequent length of sentences awarded by courts.

The hypothesis that men whose motivation had improved during their stay in Grendon would be those who did particularly well once they were released was tested by dividing the population on the basis of improvement in AP scale scores, but this produced no significant differences between groups as regards our outcome measures.

Case histories

The present report has the advantage over many reconviction studies of being able to consider behaviour over a long period of time. However, as the following three histories demonstrate, even with a ten-year follow-up period, official records remain an extremely crude and dubious measure of offending behaviour. In their separate ways, they also bring into question the issue of whether events which take place years after discharge from an institution can properly be linked to events within that establishment.

The first case was a man in his late thirties when admitted to Grendon, who was serving a long sentence for armed robbery. Being older than the majority of inmates, he felt that he had been of use to the younger men on his wing, and that for the first time in his life he had had something to contribute to others. He was realistic about his ability to go straight when he was released, and indeed, once discharged from Grendon, he received his first conviction within a few months, for stealing. This was followed some years later by convictions for burglary, for drunkenness, and for drug offences, making a total of four convictions altogether. None of these resulted in his being sent to prison, and in comparison with his previous record they represented a considerable improvement in his behaviour. Shortly before the follow-up period was due to elapse, he was involved in a drunken brawl which resulted in the death of the man

TABLE III

Criminal and other characteristics of the Grendon sample divided on the basis of their Attitude to Psychiatry scale scores

	Low AP score (18) (n = 20)		*Mid-range (19–20) (n = 11)*		*High AP score (21 +) (n = 29)*			
	Mean	*s.d.*	*Mean*	*s.d.*	*Mean*	*s.d.*	*F*	*P*
Court appearances								
Number of court appearances prior to Grendon	11.9	6.7	9.6	4.1	9.9	5.0	0.97	0.38
Number of court appearances after Grendon	7.7	8.1	4.3	2.7	3.9	4.3	2.86	0.06
Imprisonment								
Number of prison sentences prior to Grendon	5.8	4.3	4.4	2.7	4.0	3.0	1.58	0.21
Number of prison sentences after Grendon	2.8	3.8	1.7	1.5	1.8	1.9	1.04	0.36
Intelligence								
Total score on the AH4 test	65.0	27.6	79.4	12.9	78.9	18.7	2.40	0.10

he had fought with. Medical examination in his remand prison showed him to be terminally ill, and his prosecution was not proceeded with. He does not, therefore, figure as a homicide offender in our statistics. But even suppose that these exceptional circumstances had not pertained, and that he had been charged and found guilty of murder: what would it have meant? Ten years after he left Grendon he was involved in a drunken fight which culminated in tragedy. By that time the man was almost 50 years old and had spent a great deal of his life in all manner of institutions. In such circumstances, it is not possible or reasonable to hold Grendon attributable for that piece of behaviour.

The second inmate was a man in his mid-twenties when we first interviewed him. He had made his first court appearance at the age of 11 and had subsequently been convicted many times. On release from Grendon, he lived in a hostel and quickly returned to drug-taking and petty thieving – but was not caught. Within a year he had met a girl and they had settled down together. In the context of this relationship, and unusually for him, he found himself a job which offered security and prospects of promotion and which lasted some years. The break-up of his relationship with his girlfriend precipitated a general deterioration in his behaviour and ultimately led to a conviction for theft and to psychiatric treatment on an out-patient basis. The potent variable in determining his early success and later disintegration was an event over which Grendon had no control and for which it was not responsible.

The third man highlights the problems inherent in relying on conviction statistics alone. In his mid-twenties, his criminal career had started in the juvenile court and he had a considerable record by the time of his admission to Grendon. He had requested a transfer to Grendon because he wanted to do something about feelings of depression. When we conducted our final interview with him he said that he had benefited from his experience in Grendon, had come to terms with what he called his inferiority complex, and felt the place had made him more confident in himself. At the same time, he said he would probably return to stealing and indeed, once out, did so. But we know this only because of his report to us; he was not convicted for these offences. When his ten-year follow-up period ended he had a completely clean record. Judged by his former criminal record this event alone might have been hailed as little short of miraculous. Several months after the follow-up time had elapsed, he was convicted of possessing firearms and received a long prison sentence. By his own account he had continued to steal after leaving Grendon, and, rather than representing an improvement in his behaviour with regard to crime, his clean record in the period following discharge probably represented a marked improvement in his ability to avoid arrest. As it stands, a dismal pre-Grendon record contrasts with a completely clean sheet. Once again, the random or error variance in the equation makes statistical examination of a treatment effect totally inappropriate.

Discussion

Men who came to Grendon vary in their motivation for treatment and in their intelligence. The figures presented in Table III suggest the possibility that there is an interaction effect between motivation, intelligence, and Grendon, so that time spent there by the more intelligent and better motivated men may have made some difference to their later behaviour. Such a result would not be too surprising, given that the help offered in Grendon (group therapy) is dependent on the ability and willingness of the participants to talk. If this is indeed so, Grendon's role is best regarded as that of a catalyst, acting by giving men who want to change the opportunity of spending their time in prison constructively. We believe this to be a realistic description of what goes on there. To view the prison as 'treatment', analagous to the criterion variable in a physical experiment, is inappropriate and misleading.

Of more general criminological interest is the distinction to be made between the seriousness and frequency of reconvictions. These were found to have different correlates. For both measures, drinking behaviour was associated with poor outcome. But in the case of seriousness of reconviction, an association was also evident with violence and with various associated personality attributes such as impulsiveness and irritability ($r=0.26$, $P<0.023$). On the other hand, frequency of court appearances was associated with vagrancy, low drive, low intelligence, low self-esteem, and measures of motivation.

Conclusions

How should Grendon be assessed? It shares with all other penal establishments the aims of the prison service. Rule 1 of the Prison Rules (Homes Office, 1964) states that the aim is "To hold (securely) those people admitted to custody and to encourage and assist them to lead a good and useful life". Measured in these terms, Grendon has proved to be a success. In its 23 years of operation, only one man has ever escaped from it. Our earlier study described the efforts made in Grendon to encourage its inmates to lead a "good and useful life". For many men in the prison system, the main characteristic of a sentence is that it represents wasted time. Life on the outside ceases to exist, but the enforced isolation from normal society is not given over to contemplation and self-improvement but rather to tedium, discontent and further alienation. Because Grendon makes an effort to encourage inmates to change, it has tended to be compared with other prisons on whether or not it performs better in regard to the behaviour of its 'graduates'. The question which might better be asked is why all other prisons do not do as much as Grendon in this respect.

The question, "Does Grendon work?" is simplistic and naïve. Grendon is an institution which accepts individuals of widely differing opinions, motivation, and ability. For those who go there, it provides an opportunity to examine past behaviour and current problems. There is a fairly strong indication from this investigation that some such men, the more intelligent and positively motivated, may indeed use their experience to alter their lifestyles. But common sense would suggest that much more attention should be given to care in the community for these men if the potential for change is to be capitalised on.

Ten years after our original investigation of the prison, we remain convinced that it is a paradigm for good prison management, that it presents for all its inmates the opportunity not to waste time during their incarceration and that, for some, it may represent a watershed in their lives.

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A Ten-Year Follow-Up Study of Southampton Opiate Addicts

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A ten-year follow-up study of 74 opiate (most heroin) addicts referred to the Drug Dependence Clinic in Southampton showed that overall, little use was made of the treatment facilities available. Half of the patients on whom we were able to obtain follow-up data appeared to be no longer abusing drugs. An appreciable number of subjects had committed offences prior to abusing drugs and a large proportion offended during the follow-up period, adding support to the view that most addicts are not driven to crime because they are unable to obtain the help they need. The death rate of 15% is that expected in a long-term follow-up study. The results suggest that the natural history of many heroin addicts is uninfluenced by therapeutic intervention.

A Drug Dependence Clinic for Southampton and South West Hampshire was established in 1968 on the recommendation contained in the Second Report of the Inter-Departmental Committee on Drug Addiction. The functions of the clinic are described, together with the results of a ten-year follow-up study of 74 opiate addicts referred between September 1969 and February 1973. Relatively few addicts have been followed up over such a long period, and even fewer studies of opiate addicts have been carried out in provincial cities.

Size of the problem

The prevalence of drug abuse in the Southampton District is not known, but during the period of study the Psychiatric Case Register identified 80 patients given the diagnosis Drug Dependency, Morphine Type (ICD 304.0). This is an underestimate of the problem, as only data on patients referred to the service are recorded in the register. It is likely that the cohort described represented a significant proportion of the total population of Southampton opiate addicts during the early 1970s.

The service

Patients were referred by their GPs, other medical colleagues, social services, probation and after-care departments, the courts, solicitors and voluntary organisations. Most patients were seen as out-patients within a week, or within a day if necessary. Those thought to be dependent on opiates were offered immediate admission to hospital for withdrawal from drugs or to decide whether 'maintenance treatment' was indicated.

Those who refused rapid withdrawal had their drugs withdrawn gradually in the clinic, while a minority of patients, in whom all attempts at persuasion to abandon the drug habit (including imprisonment) had failed, were given maintenance treatment. Such patients attended the clinic daily, although some who did not live near were allowed to collect their drugs from a local chemist. In exceptional cases, patients who were unemployed or who were looking after their families while their spouses worked were given a week's supply of drugs.

Patients who had previously been maintained on diamorphine (heroin) were weaned off this and methadone, initially given intramuscularly and later orally, was substituted. Long-drawn-out arguments over the doses required were avoided, and instead patients were urged to participate in more constructive treatment. They were persuaded to accept rehabilitation in one of the units that have been established throughout the UK, although most who agreed to be rehabilitated were referred to Alpha House, a unit modelled along the lines of the Synanon, Daytop, and Phoenix House experiments in the USA. Addicts referred by the courts or solicitors were assessed by the staff at Alpha House, and if it was thought that they would participate in the rehabilitation programme it was recommended that they be remanded on bail for a 4–6 week trial period in the unit. If this was successful, it was requested that they be allowed to continue treatment under Section 3 of the Powers of the Criminal Court Act, 1973 (formerly Section 4 of the Criminal Justice Act, 1948). The court was also advised that those who failed to co-operate or left against advice should be brought back before the court and dealt with in any other way thought appropriate.

A Drug Abuse Advisory Service was established during the early 1970s to help those who were not prepared to take advantage of these facilities, and to provide advice for the families of drug abusers. This later became the Southampton Inter-Professional Drugs Liaison Committee. It provided the expertise of a social worker; probation officer; community psychiatric nurse; voluntary workers, including representatives from the Samaritans and Nightline (formerly run by Southampton University students, but later discontinued); a solicitor with experience of criminal law (representing the Law Society); a senior police officer; staff from the local authority's departments of education and health education; and a consultant psychiatrist (JGE). A telephone was installed and manned 24 hours a day by Nightline members. The telephone number, identified as a means of obtaining advice anonymously on problems related to drugs, and with no obvious connection with the 'establishment', was widely publicised for use by anyone in the district. Much advice was also given to general practitioners, casualty officers and others in their dealings with individual cases, while an informal telephone call was made to GPs when it was learnt that they were prescribing drugs of abuse, albeit in good faith.

Method

Patients

The cohort studied comprised 53 men (72%) and 21 women (28%). Their ages ranged from 16 to 35 years, with a mean of 21. The majority (92%) were in their late teens or early twenties. All but two patients were British. A large majority (97%) came from social classes III–V. At least four dropped out from school before the age of 16; only five received higher education. The majority of subjects (70%) were single, while a large proportion of those who had been married were separated. Most patients (88%) had unstable work records, having been sacked from several jobs or having had long periods of unemployment. Just over half (52%) lived at home with their parents, while the others lived with a spouse or cohabitee or lived alone. At least 18 patients (24%) had a family history of mental illness, nine (12%) of drug or alcohol abuse and eight (11%) of conflict with the law; some had a family history of more than one of these. At least 12 (16%) had a significant period of separation from their parents during early life, while at least 18 (24%) came from broken homes.

When first seen, the patients had been abusing drugs for periods ranging from two months to more than ten years. Only eight subjects (11%) had abused drugs for less than a year, while four (5%) had taken drugs for more than ten years. Practically all of the patients were poly-drug abusers. Fifty-nine (80%) had come into conflict with the law, at least eight of these prior to abusing drugs. Collectively they had committed 129 offences, mainly illegal possession of drugs, theft, and breaking and entering chemists' and other properties. They had been dealt with by the courts in various ways, including probation, treatment under sections of the Mental Health Act, and imprisonment.

Follow-up

Follow-up data were obtained from case records, and the patients were sent out-patient appointments. Visits were made to the last known address of those who did not turn up, and, if they had moved, attempts were made to trace them at their new addresses. Data were obtained from GPs, probation officers, Alpha House, other treatment centres, prisons and the Home Office files. Only 15 patients (20%) could be traced in person, so much reliance had to be placed on these other sources. Information gleaned from other drug abusers was not used as it was thought to be unreliable. Data suitable for analysis, although not always complete, were obtained on 64 patients (86% of the original cohort). Of these, 47 (73%) were men and 17 (27%) were women. The sex ratio is similar to that of the original cohort, but there is a bias in the age distribution, with a larger proportion of younger women among those followed up.

Results

Use of service

Sixteen patients (22%) received methadone 'maintenance treatment' during the follow-up period. Forty-one (55%) had been admitted to hospital, one patient as many as ten times. The total time spent in hospital (for all admissions) ranged from one day to 200 days, with a mean of 26 days. The average length of stay per admission was 11 days. At least nine patients had been admitted to hospital elsewhere, mainly in London, for periods ranging from 5 to 120 days. The more prolonged stays were for psychiatric illnesses (especially schizophrenia), which were diagnosed in a small minority. Four patients were admitted to hospital under Section 29 and four under Section 25 of the Mental Health Act 1959; all others were admitted informally. Twenty-nine patients (39%) were referred to Alpha House. Six were referred more than once, making a total of 37 referrals. The lengths of stay ranged from one day to two years. Four of the patients had been referred to other rehabilitation units.

Little use was made of the advisory service. Between December 1973 and March 1975 only eight telephone calls were received. Three of these were from students requesting information for sociological projects, one was from a woman asking for information concerning possible interactions between phenelzine and her oral contraceptive and one was a wrong number. Only three calls led to advice being offered on the telephone and in no case was this urgent.

Outcome of treatment

The outcome was assessed in the case of the 64 patients on whom we had follow-up data. Of the original 51 single patients, at least 19 (37%) had married, while of the 11 who were originally married, six (56%) had separated or been divorced. One of these had been divorced and remarried. More than half of the subjects (57%) were working full-time or were housewives, and another 10% were doing temporary or part-time work. A quarter of the patients lived with their spouses, while others lived with their parents, cohabitees or friends. None was homeless.

Forty-three subjects (58% of the original cohort of 74) had committed offences during the follow-up period. Of the 32 men who offended, six had previously been in trouble with the law; 10 of the 11 women who offended also had a history of previous conflict with the law. The main offences were illegal possession of drugs, obtaining drugs by deception, theft, and breaking and entering. These were dealt with by the courts in various ways, including probation in the case of 30 patients (41%) and prison in the case of 19 (26%).

Of the 64 on whom we obtained follow-up data, 32 (50%) claimed or were said to be no longer using drugs, while six (9%) were receiving maintenance treatment with methadone. We noted from the Home Office files that four (5%) of the original 74 addicts were in prison and 11 (15%) were dead.

Patients who appeared to be off drugs were more likely to have been living with their parents or spouses when first seen 10 years earlier ($\chi^2 = 10.540$, 1 d.f., $P<0.01$). There was no significant relationship between apparent abstinence and sex of patient, duration of drug abuse when first referred (five years or less versus more than five years), the provision of maintenance treatment, or whether the patients had remained at Alpha House for a week or longer during the follow-up period.

Discussion

For more than a decade, patients referred to the Drug Dependence Clinic in Southampton have been managed in much the same way as is currently recommended by the Medical Working Group on Drug Dependence of the Department of Health and Social Security. While it is not possible to draw general conclusions from the results of a single study carried out more than a decade ago in a city that could have an atypical drug-dependence problem, a measure of the outcome goes some way towards assessing the effectiveness of this management policy, adds to the sparse literature on the natural history of opiate addiction, and provides results with which those from other treatment programmes can be compared.

Proponents of our treatment have noted that every possible opportunity for help has been provided, and believe that it is not the fault of the service if addicts choose not to avail themselves of these. Critics, on the other hand, have argued that the policy is too rigid, and that insisting on addicts being admitted before providing maintenance treatment is unnecessary. Yet some youngsters who claimed they were addicted have been shown as in-patients not to be dependent on drugs. Had their claim been accepted and drugs prescribed, a non-addicted subject could easily have been given drugs to abuse or sell. In-patient assessment has also shown that maintenance doses of drugs smaller than those demanded usually suffice.

In the field of drug addiction one cannot win. If the treatment offered is not accepted, it is alleged that we are doing too little. If drugs are not readily prescribed, we are made the scapegoats for the problems that addicts bring upon themselves in their attempts to finance their illegal supplies. On the other hand, if we prescribe too readily, it is suggested that we are running clinics for 'kicks' and adding to the black market supply. No compromise on all these difficulties satisfies all people.

Research in this field is fraught with methodological hazards. There are difficulties in diagnosing dependence, difficulties in tracing patients, and difficulties in determining what is a good or bad outcome. Some believe that a good outcome is purely the abandonment of a drug habit; they may overlook the fact that the old addiction has been replaced by a new one, such as dependence on alcohol. Others place the emphasis on social adjustment or maturation of the personality. To a large extent, assessment of outcome is a value judgement that varies from individual to individual and from one professional group to another. Due to lack of financial and manpower resources, our measures of outcome were limited to the use of drugs, employment status, antisocial behaviour, and death.

Overall, little use was made of the service. Many patients did not keep their appointments and only turned up at a time of crisis or when in trouble with the law – sometimes hoping that this would be looked on favourably by the court. Even when addicts initially appeared motivated to abandon their drug habits, interviews often ended with a request for drugs. Not infrequently addicts agreed to admission but did not turn up, while a large proportion of those who did come into hospital discharged themselves against advice. Addicts often agreed to gradual withdrawal from drugs as out-patients but when steps were taken to decrease the dose claimed they were 'not ready'. Many patients were referred to Alpha House but they rarely remained there for long. Little use was made of the Drugs Advisory Service, even though there was no way in which it could have been seen as being associated with the 'establishment'.

In spite of this, the proportion of patients who at follow-up appeared to be no longer using drugs (50%) compares favourably with the results of other studies, in which the proportion of patients not known to be using drugs ranged from 16–45% over follow-up periods of 2–12 years (Bewley & Ben-Arie, 1968; Bewley *et al*, 1968; Zacune *et al*, 1969; Stimson & Ogborn, 1970; Boyd *et al*, 1971; Hitchins *et al*, 1971; Chapple *et al*, 1972; d'Orban, 1973; Ogborn & Stimson, 1975; Stimson *et al*, 1978; Wille, 1981; Cottrell *et al*, 1985). The high employment rate in those on whom we have follow-up data also compares favourably with the results of other follow-up studies, that show employment rates ranging from 21–70% over 1–7 year periods (Zacune *et al*, 1969; Stimson & Ogborne, 1970; Hitchins *et al*, 1971; Stimson *et al*, 1978; Oppenheimer *et al*, 1979; Hartnoll *et al*, 1980). These results subtract from the public image that all addicts end up as unemployed layabouts. The proportion of subjects that got married suggests a shift towards stability, but there is a high marriage rate within the age range of our patients, and the proportion of broken marriages has to be seen in relation to the high rate of marital breakdown in Britain as a whole.

The relationship between drug abuse and crime is complex. Drug abuse may be part and parcel of the chaotic lifestyle of antisocial personalities, while addicts may offend to obtain money for drugs. They often claim that they have been forced into crime by being refused drugs at treatment centres. Our results show that an appreciable number of subjects had committed offences prior to abusing drugs and 58% of them offended during their follow-up period. This is in keeping with the results of previous research, which has shown a higher rate of conviction before starting to abuse drugs than after starting (Kosviner *et al*, 1968; James & d'Orban, 1970; Stimson & Ogborn, 1970; Boyd *et al*, 1971; d'Orban, 1973), and a failure to reduce the rate of offending by prescribing methadone (Hitchins *et al*, 1971; Blumberg, 1976; Gordon, 1978), although in some studies a short follow-up period was compared with a long drug-taking career prior to attending the clinic. It appears, therefore, that addicts are not driven to crime because they are not given drugs, but are more often criminals to begin with.

A death rate of 15% is expected in a long-term follow-up study of this kind, previous studies having shown rates of 2–15.7% over follow-up periods of 1–11 years (James, 1967; Bewley & Ben-Arie, 1968; Bewley *et al*, 1968; Hitchins *et al*, 1971; Chapple *et al*, 1972; d'Orban, 1973; Ogborn & Stimson, 1975; Thorley *et al*, 1977; Gordon, 1978; Stimson *et al*, 1978; Willis & Osbourne, 1978; Oppenheimer *et al*, 1979; Hartnoll *et al*, 1980; Wille, 1981; Cottrell *et al*, 1985). There was little doubt about the connection between drug-taking and death in many subjects, but in others the link was tenuous.

It is difficult to make meaningful comparisons with the results of previous research, but the outcome in our patients does not appear worse and is in some ways better than that reported from areas where vastly more manpower and financial resources have been poured into the running of treatment centres. It is also difficult to generalise from a study of a population of addicts first seen over a decade ago, but, as little use was made of our treatment facilities, the results suggest that the natural history of those who do not die from their dangerous habits may not be influenced by therapeutic intervention to the extent that we may assume.

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The Prevalence of Bulimia Nervosa

A Replication Study

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There have been reports of a high prevalence of bulimic episodes and the syndromes of bulimia nervosa and DSM–III bulimia in community samples. A group of American authors recently compared the findings of a contemporary survey with those of a survey they had conducted previously and reported a three-fold increase in the prevalence of DSM–III bulimia. The present study replicates a community survey conducted four years ago in Britain. The prevalence of bulimic episodes, self-induced vomiting and bulimia nervosa found in the present survey was very similar to that found in the earlier study.

Since the specification of diagnostic criteria for bulimia nervosa (Russell, 1979) and bulimia (DSM–III; American Psychiatric Assocation, 1980), there have been numerous epidemiological surveys designed to provide an estimate of the prevalence of these disorders and their constituent components. As yet there has been no rigorous study of a large representative sample using sensitive standardised interview methods of assessment. Virtually all the studies have used simple self-report questionnaire measures on samples of convenience. Many of these studies have produced high prevalence rates. For example, among female American college students, reports of the prevalence of bulimic episodes have consistently been in the region of 60% (Halmi *et al*, 1981; Nagelberg *et al*, 1984; Hart & Ollendick, 1985). Remarkably high rates of the DSM–III syndrome of bulimia have also been reported. For example, a prevalence rate of 8% has been reported among female American high school students (Johnson *et al*, 1984); and among female American college students the rates have been even higher: 14% (Pope *et al*, 1984), 17% (Hart & Ollendick, 1985) and 19% (Halmi *et al*, 1981).

Against the background of these alarming estimates of prevalence, two reports from different continents, both published in 1983, produced significantly lower rates. Cooper & Fairburn (1983), in a questionnaire survey of women attending a family planning clinic in southern England, found that 20.9% reported bulimic episodes, defined as episodes of 'uncontrollable excessive eating'. They used three criteria to define the presence of probable bulimia nervosa: bulimic episodes; self-induced vomiting; and a 'morbid fear of fatness', defined as a response of at least 'often' to the question on the Eating Attitudes Test (Garner & Garfinkel, 1979) concerning being 'terrified about being overweight'. These criteria for 'probable bulimia nervosa' were fulfilled by 1.9%. A similar study was conducted in America (Pyle *et al*, 1983). A questionnaire based on the DSM–III criteria for bulimia was administered to a large group of college students. Among the women, 49.7% reported bulimic episodes, defined as having 'had the experience of rapidly eating a large amount of food at one time in a way that would be embarrassing if others saw you'. The DSM–III criteria for bulimia were fulfilled by 4.5% of the women, but this figure fell to 1% when a stricture was applied of at least weekly bulimic episodes and self-induced vomiting or laxative abuse.

The authors of the 1983 American study have recently reported a replication of their earlier work (Pyle *et al*, 1986). Using their conservative diagnostic criteria, they found the prevalence of DSM–III bulimia to be 3.2% among the female students; and they concluded that this constituted evidence for a three-fold increase in prevalence. The inference that the prevalence of bulimia is rising is questionable, given that the research methods employed in their two studies were very different. However, the difference in prevalence rates obtained by the two American studies suggested that a replication of the British family planning clinic survey would be worthwhile.

Method

The population studied consisted of attenders at two family planning clinics in Cambridge. Over the course of four weeks in January and February 1986, all attenders at these clinics were asked to complete a brief questionnaire. The questionnaire was similar to the one administered in the earlier study and was designed to elicit information on weight and on eating habits and attitudes.

Results

The questionnaire was given to 371 consecutive attenders at the clinics. Thirteen (3.5%) refused to complete it. While waiting to see the doctor, 276 (74.4%) were able to fill in the questionnaire; the remainder were given stamped addressed envelopes and asked to return it by post. Of the 82 women who took their questionnaires away, only 55 (67.1%) returned them.

An initial comparison was made of the demographic features and eating habits of the women who had completed their questionnaires in the clinic and those who had not done so. Since there were no differences between these two groups, the full sample of 331 was examined (i.e. 89% of the original 371 women approached).

TABLE I
Comparison of the 1981–1982 sample with the 1986 sample

	1981–1982 (n=369) %	*1986 (n=331)* %	χ^2	
Age (years)				
15–19	19.8	27.7		
20–24	40.8	36.0		
25–29	22.8	20.1	6.5	NS
30–34	10.1	10.1		
>35	6.5	6.1		
Weight (% MPMW[1])				
≤75	0.0	0.3		
75–85	7.4	11.4		
86–100	52.3	55.7	7.6	NS
101–115	33.7	26.8		
>115	6.6	5.8		
Eating habits				
Current bulimic episodes	20.9	27.1	3.50	NS
Current self-induced vomiting	2.9	2.4	0.09	NS
Current probable bulimia nervosa	1.9	1.8	0.04	NS

1. Matched Population Mean Weight.

Table I shows a comparison of the present sample with the earlier one in terms of age, weight and eating habits. As can be seen, there was no difference between the two groups in terms of age or weight distribution. The mean age of the earlier sample was 24.1, which is no different from that of the present sample (i.e. 23.8; $t=0.67$); and the current mean Matched Population Mean Weight (MPMW) of the earlier sample was 98.0, which is very similar to that of the present sample (i.e. 97.7; $t=0.42$). In terms of the frequency of bulimic episodes and self-induced vomiting, defined identically in the two studies, there was also no difference in prevalence between the two samples. The Eating Attitudes Test was not administered in the present study and a 'morbid fear of fatness' was defined as a response of 'markedly' to the question 'are you afraid of becoming fat?' This represented the most extreme response on a four-point rating scale. Out of the sample of 331, six women reported current bulimic episodes and self-induced vomiting and had a morbid fear of fatness. This represents a point prevalence rate of probable bulimia nervosa of 1.8%, which is almost identical to the estimate obtained in the earlier study.

Discussion

The present study constitutes a replication of an epidemiological survey conducted in Britain in 1981–1982 to determine the prevalence of bulimia nervosa. It was prompted by a report of a three-fold increase in the prevalence of DSM–III bulimia in recent years in America (Pyle *et al*, 1986). In the earlier British study the prevalence of probable bulimia nervosa was estimated to be 1.9%. In the present study, using similar measures on a sample likely to be similar in demographic features, the prevalence rates found for bulimic episodes, self-induced vomiting and bulimia nervosa were very similar to those found in the earlier study. It could be argued that a comparison of syndrome prevalence rates is illegitimate because the two studies used different definitions of a 'morbid fear of fatness'. Since in neither study did a case arise of an individual who reported the disturbed eating habits in the absence of a morbid fear of fatness, and since in both studies the class of those fulfilling the morbid fear of fatness criterion was much larger than the class fulfilling the other two criteria, it is most unlikely both on probabilistic and clinical grounds that the differing definitions had any influence on the syndrome prevalence figures.

It is unclear why the American and British epidemiological research on eating disorders continues to produce such different findings. One reason may be a genuine difference in the prevalence and incidence of disturbed eating behaviour in the two countries. Another possible reason is that different sets of diagnostic criteria are employed and different methods of assessment used. The uncertainty surrounding the epidemiology of eating disorders will remain until common diagnostic criteria are used in studies which employ rigorous and replicable research methods.

Russell (1985) has suggested that the 'cases' of bulimia nervosa identified in questionnaire surveys differ considerably from patients presenting at hospital clinics. Further examination of the 'cases' identified in the present study supports this speculation. Of the six probable cases, all of whom reported at least monthly bulimic episodes, only two reported at least weekly episodes and none reported such episodes occurring daily. In contrast, in a recent series of patients attending a psychiatric out-patient clinic, half had been experiencing bulimic episodes at least daily, and 17% at least twice daily (Fairburn & Cooper, 1984). Thus the prevalence figure of between 1% and 2% for bulimia nervosa obtained in the earlier British survey and confirmed by the present study should be regarded as a prevalence rate of mild disorders of uncertain clinical significance. The rate in the community of cases of bulimia nervosa of the severity seen in psychiatric out-patient clinics remains unknown.

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Brief Reports

Validity Study of the Hospital Anxiety and Depression Scale Among a Group of Saudi Patients

The Arabic version of the HAD scale was validated in a sample of 50 Saudi patients. The scale scores were assessed against the principal author's clinical evaluations. Spearman correlations of all items of the scale, except for one, were statistically significant. The non-significance of one item was probably related to the way it was translated into Arabic. The study furnished evidence that the Arabic version was a reliable instrument for detecting states of anxiety and depression in Saudi patients in a primary health care setting.

The Hospital Anxiety and Depression (HAD) scale (Zigmond & Snaith, 1983) was designed to be a reliable instrument for detecting states of anxiety and depression in the setting of hospital medical outpatient clinics. It is brief and limited to the two most common aspects of mood disorder presenting in hospital practice (i.e. anxiety and depression). It is composed of 14 items, 7 for anxiety and 7 for depression. Items representing symptoms of severe mood disorders (e.g. suicidal feelings) or bodily symptoms likely to occur in physical illness have been excluded.

The purpose of this study was to test the validity of the HAD scale in a sample of Saudi patients, as previous studies have only been carried out in the United Kingdom.

Method

No instrument measuring anxiety and depression disorders had been validated in Saudi Arabia before, and therefore it was decided to test the scale against the principal author's clinical judgement, after a formal interview and mental state assessment. The principal author is a British-trained psychiatrist, who has worked in the same geographical area for more than 10 years.

The study was conducted in a primary care health centre situated in the middle of Al-Khobar, Eastern Province, Saudi Arabia. Subjects were consecutive new Saudi patients of both sexes, between the ages of 15 and 65 years, who suffered from a variety of complaints and illnesses. The nature of the study was explained to each patient and it was made clear that there was no obligation to take part. Each patient was then given the Arabic version of the HAD scale to complete while waiting to see the physician. In the case of patients who were illiterates the physician read out the items and helped the patients to complete the scale.

After examination by the physician, every patient was interviewed by the principal author in a separate room, without any knowledge of the scores on the HAD scale. The severity of anxiety and severity of depression were assessed at a comprehensive and standardised interview. Careful enquiry was made about relevant psychosocial background. For anxiety and for depression, patients given ratings of 0 and 1 at this interview were considered to be non-cases, those given a rating of 2 to be doubtful cases, and those given ratings of 3 and 4 to be definite cases.

The HAD subscale scores were compared with the author's ratings for anxiety and depression; rank correlations were also calculated for each item of the HAD scale against the remaining six items in the appropriate subscale. Data from all 50 subjects were used in this analysis. Spearman rank correlation was also used to determine whether anxiety and depression subscales detected different aspects of mood disorder. For this objective, only the data collected from 14 subjects was used; that is, from only those who had a difference of 2 or more points between the anxiety and depression scores in the clinical assessment. The sensitivity and specificity of the HAD subscales were assessed in terms of false positive and false negative rates. The authors of the HAD scale recommend a borderline range for both anxiety and depression subscales; that is, scores of 7 or less as non-cases, 8–10 as borderline cases, and 11 or more as definite cases.

Results

Fifty subjects (25 males and 25 females) were included in the study. Following accumulation of data, statistical tests were done. Correlations between the scores on each of the 14 items and the total of the HAD scores on the other six items in the subscale and the scores of the clinical assessment were calculated.

The item–subscale correlations for all the items in both subscales were highly significant ($P<0.001$), except for one of the anxiety items, the English wording of which is, "I get a sort of frightened feeling like butterflies in the stomach." Similarly, all the items except this one had highly significant correlations ($P<0.001$) with the author's ratings of the respective mood disorder.

The overall total subscale scores correlated significantly with the author's rating: for anxiety, $r+0.88$, $P<0.001$, and for depression, $r=+0.86$, $P<0.001$.

The sensitivity and specificity of the HAD subscales are represented in Table I. This shows that for anxiety there were no false positives or false negatives; in the depression subscale there were no false positives but 2 out of 13 (15%) false negatives.

TABLE I
Distribution of 50 subjects according to total HAD scores and clinical assessment

	Clinical assessment, anxiety subscale			
	Non-cases (0–1)	*Doubtful cases (2)*	*Cases (3–4)*	*Total*
HAD anxiety score:				
0–7	19	8	0	27
8–10	1	4	1	6
11–20	0	3	14	17
Total	20	15	15	50

	Clinical assessment, depression subscale			
	Non-cases (0–1)	*Doubtful cases (2)*	*Cases (3–4)*	*Total*
HAD depression score:				
0–7	20	7	2	29
8–10	0	8	3	11
11–20	0	2	8	10
11–20	20	17	13	50

Data on the 14 patients earlier referred to were analysed to determine whether the HAD subscales differentiated between anxiety and depression. The anxiety subscale correlated significantly with anxiety ($r = +0.83$, $P < 0.001$) but had a negative correlation ($r = -0.41$) with depression. Similarly, the depression subscale correlated significantly with depression ($r = +0.82$, $P < 0.001$) but had a negative correlation ($r = -0.68$) with anxiety. These figures, although on a small subsample, strongly support the view that the HAD subscales are specific to the mood they are designed to measure.

Discussion

The finding that the HAD scale was not specific in pointing out doubtful cases does conform with clinical experience that mild emotional (or neurotic) disorders cannot be judged to be either present or absent with certainty. The crucial and relevant question of 'how much' emotional disorder is existent is resolved by the HAD scale as substantiated by previous results. The borderline range used appears appropriate, since the scale scores and the clinical evaluations proved consistent.

The fact that item 9 of the anxiety subscale, "I get a sort of frightened feeling like butterflies in the stomach", was the only insignificant item is probably due to difficulty in translation of this colloquial English expression. This one item does not affect the overall validity of the scale, but future users of the scale would be justified in substituting a different, although similar, expression referring to a sensation of fear centred in the epigastric region.

The Arabic translation of the words 'anxiety' and 'depression' is not agreed on with certainty among Arabic-speaking psychiatrists, and therefore different Arabic words which are occasionally used to translate them carry no specific and sharp boundaries between them, and some people may use them interchangably. This lack of specificity in Arabic words used to describe those two aspects of emotion give the Arabic version of the HAD scale special valuable weight, particularly when the process of case-finding is being done by somebody not experienced in psychiatry or not mastering the Arabic language. The scale items give clear description of certain emotional situations rather than using words vaguely.

A crucial point is that the essential basis of the depression subscale is anhedonia. Five of its seven items describe this experience in one form or another (Snaith, 1987). The scale, therefore, measures anhedonic depression which is taken to be the best indication of hypomelancholia. Kräupl-Taylor (1966) described the essential hypomelancholic symptoms as a decrease in mental and physical activity, in self-confidence and in enjoyment of pleasure, in addition to lack of energy and initiative in general. It is the view of many workers that anhedonia, if it is persistent and not adequately accounted for by the person's circumstances, is at present the nearest clinical guide to the biogenic depressive disorder. The suggestion needs to be put to the test of critical research (Snaith, 1987). At this stage it can be concluded that there is evidence that the HAD Scale is capable of detecting cases of depressive disorder, in particular mild ones, which are likely to respond to antidepressant drugs.

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A Possible Variant of the Neuroleptic Malignant Syndrome

A 20-year-old man developed generalised rigidity and signs of autonomic instability following the introduction of therapy with zuclopenthixol decanoate. A diagnosis of NMS was made, despite the absence of hyperthermia. He recovered on treatment with thiamine, dantrolene, and bromocriptine.

In their recent review of the neuroleptic malignant syndrome (NMS), Abbott & Loizou (1986) emphasised that the cardinal clinical features are the development of hyperthermia, muscle rigidity, and autonomic instability in a patient exposed to neuroleptics. I describe the case of a 20-year-old man who developed generalised rigidity and signs of autonomic instability following the introduction of therapy with zuclopenthixol decanoate. At no time did he develop a pyrexia of over 37°C.

Case report

At an early age the patient was diagnosed as mentally impaired and exhibited problem behaviour. Intermittent aggressive outbursts appeared by the age of 10, and from the age of 13 he showed episodes of bizarre behaviour such as talking to himself and barking like a dog. At this time he was admitted to a mental handicap hospital, but showed increasing levels of disturbance, with scratching, biting, kicking, bizarre talk, and self-mutilation. Diagnoses ranging from schizophreniform psychosis and manic–depressive psychosis to personality disorder were considered, in addition to his undoubted mental impairment. Treatments at various times included promazine, droperidol, sodium valproate, benperidol, sulthiame, lorazepam, carbamazepine, clopenthixol decanoate, and lithium carbonate. None made any appreciable difference. Attacks on staff escalated, and following an attempt to strangle a nurse he was admitted to Moss Side Special Hospital in August 1984 when aged 20.

His self-mutilation and violence towards others continued to cause great concern and a variety of different therapeutic measures, including several different drug regimes, was tried. By May 1986 his medication was carbamazepine (500 mg q.d.s.) (for one year), lithium carbonate (500 mg t.d.s.) (for eight months), procyclidine (5 mg b.d.) and diazepam (20 mg b.d.) (both since admission).

Despite the serum carbamazepine and lithium carbonate levels being within the therapeutic range, his behaviour remained difficult to control and zuclopenthixol decanoate (500 mg weekly) was added. A week later his serum lithium level was found to be 1.96 mmol/l and lithium was withheld. After a further week the level was 0.36 mmol/l and lithium at a lower dose of 500 mg b.d. was re-introduced. On the following day he was noted to be oversedated and ataxic and all drugs were stopped. Two days later he had two brief periods of absence accompanied by localised twitching of his jaw. He subsequently became very disturbed, indulged in head-banging, and the next day showed decreased level of consciousness, uncoordination of his upper limbs and generalised hyperreflexia.

He was transferred to a district general hospital where full blood count, serum multiple analysis, skull X-ray, cerebrospinal-fluid analysis, and computerised tomography scan were all normal. An electroencephalogram showed mild, generalised polyrhythmic abnormalities. No diagnosis was made and he was returned to Moss Side. The next day he was found to be comatose, just responding to painful stimuli, with blepharospasm and generalised extrapyramidal-type rigidity. He showed involuntary facial movements, a blood pressure of 150/90, siallorrhoea and dyspnoea, and was incontinent of urine. On transfer to a neurological unit a diagnosis of NMS was made, despite the absence of hyperthermia (temperature over 37.5°C).

Full blood count was normal, as was a chest X-ray. Serum multiple analysis revealed aspartate transanimase to be slightly raised at 45 i.u./l (normal below 40) and creatinine phosphokinase raised at 289 i.u./l (normal below 100). No traces of lithium or carbamazepine were detected on serum estimation. He required a nasogastric tube and oxygen; therapy with intravenous thiamine, oral dantrolene (25 mg t.d.s.) and oral bromocriptine (5 mg t.d.s.) was initiated. For the next 2–3 days he showed a fluctuating level of consciousness, he was frequently incontinent of urine, and his blood pressure varied from 110/70 to 150/90. The generalised rigidity and involuntary facial movements persisted. Over the next week the rigidity gradually resolved, his conscious level improved and ten days after admission he was ready for discharge.

Discussion

Many aspects of this case fulfil the criteria for diagnosis of the NMS. The patient developed dyskinesia, generalised extrapyramidal-type rigidity, fluctuating levels of consciousness and blood pressure, dyspnoea, siallorrhoea, and urinary incontinence approximately three weeks after the introduction of therapy with zuclopenthixol decanoate. Laboratory tests showed mild elevation of liver transaminases and creatinine phosphokinase, the EEG showed non-specific abnormalities, CSF examination and CT scan were normal. These results are to be expected in the NMS (Abbot & Loizou, 1986), although the levels of creatinine phosphokinase can be much higher than in this patient (Gibb & Lees, 1985), and indeed it might be argued that the raised level found could be accounted for by muscle trauma induced by head-banging rather than skeletal muscle necrosis as in the case of the NMS. Neurotoxicity has been reported when lithium is used with carbamazepine, despite both serum lithium and carbamazepine levels being within their accepted therapeutic ranges (Shukla *et al*, 1984). However, the symptoms reported in these cases were confusion, ataxia, horizontal nystagmus, hyperreflexia, and coarse tremors. None of these, apart from hyperreflexia, were present in this patient.

Despite the presence of so many diagnostic features of the NMS, at no stage was hyperthermia present. Hyperthermia has been considered an essential feature of the NMS, yet in an analysis of 120 case reports, Shalev & Munitz (1986) mention reports of "incomplete forms of NMS, i.e. forms without fever or muscle rigidity". In 1963, Price & Giannini reported a case of a man developing tachypnoea, tachycardia, dyskinesia, and hypertension following the initiation of chlorpromazine therapy and suggested that this represented a subtype of the NMS. I would suggest that this case too represents a variant of the NMS.

The pathogenesis of the NMS is unclear. Excessive dopamine receptor blockade may cause the extrapyramidal symptoms, and hypothalamic blockade may account for the disturbance of autonomic and thermoregulatory function (Szabadi, 1984). There have been suggestions that the extreme muscle spasm in the NMS is sufficient to generate a dramatic rise in body temperature and thus account for the hyperthermia. Evidence for this point of view comes from the effect of dantrolene, a drug acting peripherally on skeletal muscle, which produced a rapid relaxation in muscle tone and simultaneous fall in body temperature in some reported cases (Coons *et al*, 1982; Goulon *et al*, 1983). In this patient at least, the extreme rigidity occurring in the absence of hyperthermia would point to a central origin for the usually noted rise in body temperature.

It is worth noting the development of symptoms despite the previous uneventful use of clopenthixol decanoate. This phenomenon has been previously described, and indeed re-challenge with the original agent may not result in a recurrence of the NMS (Caroff, 1980; Shalev & Munitz, 1986; Abbott & Loizou, 1986). However, this patient had been on lithium therapy, and before the development of the NMS had shown signs of lithium toxicity. It has been suggested that concurrent administration of lithium with a potent neuroleptic may facilitate the development of the NMS due to inhibition by lithium of the compensatory proliferation of dopamine receptors when exposed to a neuroleptic (Szabadi, 1984).

The absence of a typically raised temperature may lead to a delay in diagnosis, as in this patient, and a subsequent delay in treatment. Shalev & Munitz (1986) estimated the mortality rate to be 22%, and Caroff (1980) reported that the risk increases when depot forms of neuroleptic are used. As there is now good evidence that specific treatment with dantrolene and dopamine agonists such as bromocriptine is effective (Shalev & Munitz, 1986), the recognition of *formes frustes* of the NMS is essential. Further research is needed to elucidate the pathogenesis of this puzzling condition.

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Self-Inflicted Eye-Injuries

Three cases of self-inflicted eye injuries are described and discussed. A review of the literature shows that several psychiatric diagnoses have been assigned to those who mutilate their eyes, and various unconscious mechanisms proposed to explain this unusual phenomenon.

Self-inflicted eye injuries are uncommon. Despite this they are widely mentioned in ancient literature and assume a prominent place in mythology. The Norse god Odin is said to have given one of his eyes for the right to drink a single draught from the Spring of Mimir, whose waters contained wisdom and understanding. Oedipus, the legendary King of Thebes, gouged out his eyes when he discovered the truth of his ancestry.

The notion of self-blinding for sinful thoughts comes from a variety of sources. St Matthew's gospel contains the exhortation: "And if thy right eye offend thee, pluck it out and cast it from thee; for it is profitable for thee that one of thy members should perish, and not that thy whole body should be cast into hell" (St Matthew ch. 5, v. 29). Self-blinding as a punishment for misdirected thoughts has endowed ophthalmology with three patron saints – Lucia of Syracuse, Triduana, and Medana (Stannard *et al*, 1984).

A comprehensive review of some of the older and less accessible literature showed that self-inflicted eye injuries may occur in a wide variety of situations (Tapper *et al*, 1979). Severe self-inflicted injury, including complete removal of the eye (self-enucleation) is usually associated with psychosis, or organic disorders such as epilepsy, encephalitis, and diabetes. LSD-induced psychosis has been particularly associated with self-injurious behaviour (Rosen & Hoffman, 1972).

Minor injuries may occur in the absence of any demonstrable psychiatric illness, when the motive is usually financial or to escape unpleasant duty. Self-mutilation occurring as a compulsive act is rare, but self-inflicted traumatic cataracts and retinal detachments have been described. A 30-year-old male with obsessive–compulsive neurosis virtually blinded himself by repeatedly hitting his eyes with his hands (Tapper *et al*, 1979).

Self-inflicted orbitocranial injury has been described in attempted suicide. A 44-year-old shopkeeper was admitted to a psychiatric ward following an overdose of barbiturates. Five days after admission he was found collapsed, with right-sided spasticity and a right periorbital haematoma. A diagnosis of cerebrovascular accident was made, but was revised when a straight skull X-ray revealed a ballpoint pen within the cranial cavity. The pen had been thrust through the right orbit, across the midline of the head, into the left tempero-occipital region. It was retrieved by a neurosurgeon, and two years later the patient had a right hemiparesis with some loss of visual fields in both eyes but otherwise normal vision. He described his action as a suicide attempt (Bowen, 1971, cited by Tapper *et al*, 1979).

In a wide-ranging review of the eye in psychiatry, Trevor-Roper (1980) reported a young Frenchman who proffered one of his eyes saying "It just fell out while I was playing cards". A tell-tale drop of blood was discovered on his fingernails. Trevor-Roper describes the eye as a "sounding-board for the travails of the whole persona" and emphasises the rôle of the eye in sexuality. To him the eye is "a sort of sexual surrogate, since it is such a convenient organ of displacement whenever fear, guilt, impotence or despair debar a more physiological outlet". Thus self-enucleation becomes "the final solution".

Case Report 1

A 67-year-old widow attended a casualty department with a history of having "poked" herself in the eye with a pair of scissors. Initially she said that it had been "a silly accident", but later volunteered that she had injured her eye deliberately. She was admitted to the ophthalmology ward where her minor injury was repaired.

Until the time of the incident the patient had been living with her son and daughter-in-law, despite having a house of her own nearby. Her son had tried to persuade her to return to her house, but she felt that she was too old to live on her own. Following her eye injury her son and daughter-in-law refused to allow her to return to their home. They described her behaviour as "manipulative".

The patient was examined by a psychiatric Senior Registrar, who found no evidence of psychiatric illness. In particular there were no depressive features and no cognitive impairment. She would not say why she had injured her eye.

A few days before she was due to be discharged the patient committed suicide by jumping from the fourth floor of the hospital.

Case Report 2

A 21-year-old unmarried NCO was invalided out of the services following the onset of a schizophrenic illness for which he received major tranquillisers. He suffered from auditory and somatic hallucinations and thought-broadcasting. He was convinced that one eye was of immense size and that his pituitary gland travelled out of his eye, damaging other people. In response to these delusions he fired an air pistol at his eye, the resulting damage requiring enucleation.

He continues intermittently to be deluded about his remaining eye, and is at risk of blinding himself during a future relapse.

Case Report 3

A 42-year-old single man with a history of paranoid schizophrenia was admitted to hospital in an agitated state. He had been acting strangely for several days, quoting from the Bible and singing hymns at work. He had absented himself from work for four days, and had returned wearing an eye-patch over his right eye. He refused to discuss the eye-patch, saying "It's a private matter between me and God."

Examination of his mental state revealed paranoid delusions and religiosity. He refused to allow his eye-patch to be removed or his eye to be examined. He made a good response to phenothiazines and later admitted that he had ruptured his eye by pressing with his fingers because "certain forces were tricking me. If thy eye offend thee, pluck it out. It's not as painful as you think."

One year later the remains of his eye were removed surgically. Five years later, during a relapse in his illness, he castrated himself using a razor blade.

Discussion

There have been several attempts over the years to fit self-mutilation of the eye into a single conceptual framework. A moral or religious model is often used by patients themselves, who feel they are carrying out the instructions of the Bible. In the earliest clinical case report the patient quotes from St Matthew's gospel, and a diagnosis of *'délire mystique'* is made (Bergmann, 1846, cited by Rosen & Hoffman, 1972). The case is similar to case report 3 above. The vast majority of Christians interpret the gospel in a symbolic and metaphorical way, but some psychotic individuals demonstrate 'concrete thinking' in their inability to make abstract generalizations.

Some psychoanalysts interpret Oedipus' self-blinding as the equivalent of self-castration, believing in an unconscious equating of the eyes with the genitals. One recent paper stated that "castration fears, failure to resolve Oedipal conflicts, repressed homosexual impulses and self-punishment are ubiquitous in self-enucleation" (MacLean & Robertson, 1976). A review of the literature shows this to be an unwarranted oversimplification (Tapper *et al*, 1979). An alternative psychodynamic paradigm is that the eye represents the symbolic condensation of the self, so that self-enucleation "is a substitute of a part of the body for the whole. It allows one to kill the devilish responsible self and yet live" (Rosen & Hoffman, 1972). This is the concept of "focal suicide". There are similarities between this psychodynamic interpretation and the exhortation in St Matthew's gospel.

Kell Yang *et al* (1981) emphasised neurochemical factors in self-inflicted injury. They noted that self-mutilation has been successfully treated with L-tryptophan and suggested that "biochemical assays of red blood cell purine metabolism should be done in patients with histories of self-inflicted mutilation of the eyes". This approach has received little support, although it may have a limited rôle in Lesch–Nyhan syndrome and Gilles de la Tourette syndrome, both characterised by self-mutilation.

The three cases reported here demonstrate that self-mutilation of the eye is not a single clinical entity. Two patients suffered from severe psychoses, while the third was a lonely woman with no demonstrable psychiatric illness. Ultimately, this form of behaviour may remain incomprehensible and impossible to anticipate. What can be said is that patients who damage their own eyes are likely to be suffering from a high level of distress, and should not be dismissed lightly.

The authors would be interested to hear of similar case reports from other clinicians.

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Paranoid Psychosis as the First Presentation of a Fulminating Lethal Case of AIDS

A 34-year-old heterosexual intravenous drug abuser presented with a paranoid psychosis and was subsequently found to have the Acquired Immune Deficiency Syndrome (AIDS). The patient died 14 days later of generalised septicaemia. The case is reported and the aetiology and pathogenesis of the organic psychosis are discussed. It is anticipated that similar psychiatric manifestations will occur as the AIDS epidemic increases.

The Human Immunodeficiency Virus (HIV) is the retrovirus thought to be the causal agent in Acquired Immune Deficiency Syndrome (AIDS). It infects and destroys T-helper/inducer (T4) lymphocytes, causing profound deficits in cell-mediated immunity. Consequently, the central nervous system is often affected by opportunistic infections and lymphomas (Snider *et al*, 1983). HIV is also neurotropic and may be the direct cause of dementia (Shaw *et al*, 1985; Navia *et al*, 1986), myelopathy (Petito *et al*, 1985) or neuropathy (Ho *et al*, 1985).

HIV was first isolated in France (Barre-Sinoussi *et al*, 1983) and in the USA (Gallo *et al*, 1984) from blood. It was known as Lymphadenopathy Associated Virus (LAV) and Human T-Cell Lymphotropic Virus (HTLV III) respectively.

The number of subjects exposed to HIV is increasing exponentially. The first case of AIDS reported in the United Kingdom was in 1981. By 1 August 1986, 465 cases of AIDS and 234 deaths from AIDS had been reported, and 3162 subjects had antibodies to HTLV III (figures compiled by Public Health Laboratory Service, Communicable Diseases Centre, London). As the epidemic increases, psychiatrists will be consulted more frequently to treat AIDS-related organic brain syndromes, reactions to the diagnosis or its psychosocial consequences.

The following case history is an example of a man who presented with a paranoid psychosis as the first feature of AIDS.

Case report

A 34-year-old married heterosexual plumber was referred to the out-patient department for treatment of heroin-dependency. He first started smoking heroin at the age of 29 years at the time of the break-up of his first marriage. Within two months he was injecting the heroin intravenously, up to 0.5 g/day. Over the next five years (except for a 12-month period of self-enforced abstinence), he continued to abuse heroin. At the same time he started to abuse benzodiazepines, mainly diazepam and chloridazepoxide (40–50 mg/day), which he obtained both from his GP and the black market.

During the eight months prior to his referral to our department he had developed a persistent sore throat and had lost 20 kg in weight. For three weeks he had abstained from heroin following the prescription of a reducing course of methadone. At interview he was an unshaven cachectic man who looked ill. His behaviour was inappropriate, he sucked a bright red lollipop throughout the interview and later pulled out a bottle of methadone and started to drink it.

He was orientated in time, place, and person but was not fully aware of the purpose of our meeting. He talked in a slow monotone and said that he felt anxious and physically ill. He looked perplexed. He believed that his parents' house was infested with bugs and that some of these were inside his stomach eating his methadone. He incorrectly believed that his father was watering down his methadone and selling the remainder. He admitted to seeing fleas on his bedspread a few days earlier and to hearing second-person auditory hallucinations. Cognitive testing revealed slightly reduced attention and concentration. He correctly registered a name and address but could only remember three out of six items at five minutes. His general information was fair.

After this initial consultation he was admitted to hospital. Shortly after admission he became more deluded, believing that he had sunstroke and that one part of his head was lower than the other.

Physical examination: apyrexial, pulse 80/min regular, BP 120/70, oral candidiasis, a cough producing thick green purulent sputum but no localising signs in the chest, and a maculopapular rash on his chest and arms. Investigations: Hb 11.8 g/dl; WBC 4.5×10^9/l with a lymphopenia of 0.23×10^9/l. The film showed anisocytosis, occasional hypochromia and pencil cells; ESR 60/min; total protein 88 g/l; albumen 32 g/l; globulin 56 g/l; liver enzymes normal. Anti Hb Core IgG positive (implies previous infection with hepatitis B); HTLV III antibody positive; sputum grew normal flora only; throat swab grew candida and a staphylococcus aureus.

Chest X-ray: pneumonic consolidation at the base of the right upper and left lower lobe. Electroencephalogram (EEG) showed slightly slow symmetrical alpha rhythm at 8–9 Hz, with even slower medium-voltage theta rhythms at 4–6 Hz in all areas. Computerised tomography (CT) scan showed dilated ventricles, cortical atrophy and enlarged sulci and a marked sinusitis. Lumbar puncture: clear sterile CSF, with normal protein and sugar concentrations.

He was treated with methadone (10 mg t.d.s.), diazepam (10 mg t.d.s.), and chlorpromazine (150 mg q.d.s.). Nystatin suspension and later amphotericin were prescribed for the candidiasis. His delusions resolved over the next five days but he was intermittently mildly confused (worse at night) and disorientated in time. On one occasion he got lost around the ward.

In view of his poor physical state and the good response of his psychosis to neuroleptic medication, it was decided to transfer him to the local infectious diseases hospital for further treatment of his opportunistic infections. He was counselled regarding his HTLV III status and his oral candidiasis, sinusitis, and pneumonia. He denied that there was anything wrong with himself and refused to be transferred. Later that day he absconded and went home. When at home he became more aroused and clearly psychotic. He thought the doctors were imposters and that the hospital was not a National Health Service hospital. He threatened his mother and others with physical harm if he was made to return to the hospital. He was placed on a Section 4 of the Mental Health Act (1983) and was returned to the hospital with the assistance of the police.

It was decided to treat his chest and sinus infection empirically with co-trimoxazole (960 mg b.d.). Twenty-four hours later he became septicaemic following a deterioration of his respiratory infection and was transferred to Intensive Care. A second X-ray showed radiological changes typical of pneumocystis carinii infection. He was ventilated for 72 hours, and died following a prolonged period of intractable hypotension.

Discussion

This 34-year-old heterosexual married man was referred for treatment of heroin dependency and found at interview to have a paranoid psychosis. There was no family or previous personal history of psychiatric illness. An organic aetiology for the psychosis was presumed and was supported by the impairment of consciousness, intermittent confusion, and disorientation (Lishman, 1978). He denied using amphetamines. No-one at this stage had suspected that he might have AIDS. It was only after admission to the psychiatric unit that opportunistic infections, lymphopenia and antibodies to HTLV III were identified meeting Centers for Disease Control criteria for a diagnosis of AIDS (Jaffe *et al*, 1981).

The most parsimonious explanation for the presenting paranoid psychosis is that it was part of the clinical picture of AIDS. There are different ways in which AIDS could have led to the psychiatric syndrome. The central nervous system (CNS) could have been affected directly by HIV infection or an opportunistic infection of the brain, or the psychosis could have been a toxic consequence of opportunistic infections (e.g. pneumonia, sinusitis, fungal stomatitis) outside the brain. Although a toxic state could not be excluded, the findings of clear sterile CSF, diffuse bilateral slowing of the EEG, and the CT scan changes of cerebral atrophy and dilated ventricles would be consistent with HIV infection of the brain (Shaw *et al*, 1985).

The course of the illness was unusually rapid, with death occurring two weeks after presentation, whereas in most reported cases the course of the illness was more protracted. Indeed, the most common psychiatric complication is a dementia developing over weeks or months. It is of interest that the CT scan showed generalised atrophy in our patient, indicating that probably there was a subclinical phase of the damage to the brain prior to the onset of symptoms.

HIV infection of the brain has been variously termed AIDS encephalopathy (Shaw *et al*, 1985; Sharer *et al*, 1986), subacute encephalitis (Snider *et al*, 1983; Nielsen *et al*, 1984) and AIDS dementia complex (Navia *et al*, 1986). It occurs in about one-third of patients with AIDS (Carne & Adler, 1986). The condition is characterised by subtle cognitive changes, confusion, malaise, lethargy, loss of libido, social withdrawal, depressed mood, personality change, and headache, progressing over weeks or months to profound dementia with fits and ataxia. Psychosis has also been described (Hoffman, 1984; Rundell *et al*, 1986). Kleihues *et al* (1985) report a 24-year-old woman who presented with symptoms resembling hebephrenic schizophrenia.

It is now recognised that in AIDS, CNS manifestations are common (Johnson & McArthur, 1986). In a post mortem study, 31 (78%) out of 40 patients with AIDS had significant CNS disease (Nielsen *et al*, 1984). It is important to realise that psychiatric rather than neurological manifestations may be the presenting feature of AIDS (Hoffman, 1984; Kleihues *et al*, 1985) and AIDS dementia can sometimes be the sole manifestation of HIV infection (Navia *et al*, 1986).

There are important practical conclusions to be drawn from this case. In every patient presenting with a psychosis of unknown origin and a history of intravenous drug abuse, AIDS should be suspected and the test for HTLV III antibodies be performed. Such patients can present great difficulties in management: the psychosis may require custodial treatment on a psychiatric ward, whereas the medical complications require urgent treatment on a general medical unit, preferably specialising in infectious diseases.

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Koro Syndrome Associated with Alcohol-Induced Systemic Disease in a Zulu

A case report is presented of the genital retraction syndrome, koro, associated with alcoholic hepatitis, avitaminosis and urinary tract infection, occurring in a Zulu male. Treatment of the physical conditions resulted in resolution of the koro symptomatology.

The nosological status of koro is discussed and it is proposed that the condition be regarded as a symptom-complex reaction to a variety of psychological or physical stressors rather than as a purely culture-bound syndrome.

Koro is the popular name given to the genital retraction syndrome originally described by Blonk (1895), characterised by the belief that the penis is shrinking into the abdomen and that its eventual disappearance will lead to death. The subjects are consequently anxious or frightened and have usually instituted some mechanical means to maintain the penis in its external position. Koro has been a periodic focus of interest since the original reports and is no longer regarded as a purely culture-bound syndrome restricted to the Chinese peoples of Southeast Asia as was once the case (Yap, 1965). The cases reported in non-Chinese subjects to date are reviewed by Berrios & Morley (1984), who contend that this group tend to exhibit partial or incomplete syndromes as opposed to the full symptom complex originally described in Chinese patients. Koro has been described as both a circumscribed entity subject to epidemic spread (Sachdev & Shukla, 1982), and as a phenomenon found in other better defined primary psychiatric conditions (Berrios & Morley, 1984). The following case demonstrates an association with systemic physical illness and is the first report of koro occurring in any race group in Southern Africa.

Case report

The patient was a 38-year-old married Zulu male with five children, employed as a casual labourer. He had been resident in Natal, South Africa, his whole life. He presented initially with dysuria, but later elaborated that his penis had been shrinking into his abdomen for the past three days and that he knew he would die when it disappeared completely. This belief was unshakeable despite reassurances to the contrary. He had no previous medical or psychiatric history of note. A brother had been in hospital for a transient psychiatric illness from which he had fully recovered; no further details were available. The patient's wife reported that he drank moderate amounts of alcohol on a daily basis and had done so for many years, with no previous ill effects. Systematic interrogation revealed dysuria with frequency and red-stained urine at times. There were no vegetative features of depression, reports of bizarre behaviour, or recent change in drinking pattern. On examination he was a moderately obese man of pyknic build with a low grade pyrexia (37.6) and symmetrical pellagrinous dermatitis on the face and arms. Central nervous system was grossly intact and all other systems were normal. The urine was dark brown with no macroscopic blood. Mental state examination revealed an awake, alert, fully co-operative man, able to give a logical sequential account. He was notably anxious with congruent affect. The belief concerning the shrinking penis was of delusional intensity: there were no other psychotic features. There were no physical signs of alcohol withdrawal. Laboratory investigations showed a hypochromic macrocytosis and uniformly elevated hepatic enzymes. Venereal disease reference laboratory (VDRL) test was negative; skull and chest X-rays were normal. Urine microscopy showed a low grade haematuria with five leucocytes per high-power field: *Staphylococcus Epidermis* was cultured. He was admitted with the diagnosis of alcohol-induced avitaminosis, alcoholic hepatitis, and urinary tract infection, and commenced on a course of ampicillin, diazepam and vitamin supplementation. The ideas concerning the penis abated after four days without any specific neuroleptic medication, and he gained full insight.

Discussion

This case supports the assertion that koro is not a culture-bound syndrome and suggests that the symptom complex can emerge as a reaction to a variety of non-specific medical or psychiatric stressors. Ang & Weller (1984) consider koro to be a symptom complex occurring in the presence of a hyperacute anxiety state complicating any psychotic illness, whereas Sachdev & Shukla (1982) propose that the term 'koro' be reserved for the acute, short-lasting, circumscribed illness as originally described and that 'koro symptom' should not be included in other psychiatric conditions. This latter view appears unnecessarily restrictive and is not supported by the case reported here or by most of the recent literature. Berrios & Morley (1984) include three cases in their review in which koro symptoms occurred in the presence of systemic physical disease. In his extensive review of the koro literature from an ethnological perspective, Edwards (1984) concludes that the category of genital retraction syndromes is of little nosological significance other than being of heuristic value in drawing together diverse sets of observations. The place of koro within current classification systems (i.e. ICD-9 (World Health Organization, 1978) and DSM-III (American Psychiatric Association, 1980)) remains uncertain. Traditionally regarded as a depersonalisation disorder under the emic transcultural perspective (Yap, 1965), the culture-bound syndrome would by definition be excluded from any nosological category implying psychosis, whereas from the etic transcultural viewpoint the belief that the penis is shrinking would be regarded as a delusion, best subsumed under the categories of Atypical Psychosis (DSM-III 298.90) (i.e. as a monosymptomatic delusion of bodily change) or Psychogenic Paranoid Psychosis (ICD-9 298.4). The presence of organic/physical factors as reported here leads quickly and unequivocally to the diagnosis of Organic Delusional Syndrome (DSM-III 293.81) if the appropriate diagnostic 'decision tree' is followed. Rin (1965) has offered a psychodynamic explanation for the development of the syndrome but this is directly related to the Chinese concept of illness and not to the model of any classical Western psychodynamic theory. Considering the obvious analogies to the Freudian concept of castration anxiety, it is surprising that no recognised authority from any of the established analytical schools has postulated a comprehensive dynamic explanation. Overall it appears that the full or partial koro syndrome manifests in a relatively small number of individuals, suitably predisposed for reasons yet to be determined, from disparate ethnic and cultural backgrounds, at times of psychological and/or physical stress. The nature of the symptomatology is probably a function of the relative importance of sexuality in both the personality of an affected individual and his society as a whole. Total resolution of koro episodes is the rule, but further study of this and related phenomena should provide insights into certain aspects of personality development and organisation that are ubiquitous and appear germane to all cultures.

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Ganser Syndrome and Its Management

The literature on the Ganser syndrome is reviewed, and a case history is described. A brief hospital admission is worthwhile for patients with this syndrome, but planning and persuasion are needed.

In 1897, Ganser described two prisoners who developed brief bouts of mental illness characterised by disturbed consciousness, hallucinations, sensory changes of a hysterical kind and characteristic answers to questions. These episodes terminated abruptly, with subsequent amnesia for the episode and complete recovery. He believed the condition to be hysterical and sufferers to be genuinely ill – not malingering. Many English textbooks followed Ganser in regarding the condition as hysterical (Whitlock, 1967).

However, there has been only modest agreement as to which features are necessary for the diagnosis. Mayer-Gross *et al* (Whitlock, 1967) stressed *vorbeireden* (approximate answers, e.g. How many legs has a horse? "3", and apparently foolish answers showing that the question was understood). Fish (Whitlock, 1967) also believed *vorbeireden* and paralogia were diagnostic, and stressed similarities with schizophrenic thought disorder. Whitlock (1967), however, thought that *vorbeireden* alone was insufficient for the diagnosis. He regarded the condition as essentially psychotic rather than hysterical and as one kind of clouded consciousness. Enoch & Trethowan (1979) listed the four essential diagnostic features as approximate answers, clouding of consciousness, somatic conversion, and visual and/or auditory hallucinations (pseudo-hallucinations). There is agreement, nevertheless, that the disorder is not confined to prisoners (Whitlock, 1967).

The aetiology of Ganser is diverse. Schizophrenia is commonly cited, but other causes have been listed, including neurosyphilis, alcoholism and Korsakoff's psychosis, cerebrovascular disease, depression, mental retardation, trauma and artefactual illness.

With regard to the differential diagnosis, Enoch & Trethowan (1979) listed malingering, simulated insanity, true dementia, affective states (pseudodementia), epileptic twilight states, buffoonery state, catatonia and oneirophrenia in schizophrenia. Kiloh (1961) contrasted Ganser with pseudo-dementia, Whitlock (1967) stressing disturbed consciousness in this regard as being characteristic of Ganser. Review of the relevant literature, however, suggests that clouded consciousness may be confined to organic cases (e.g. Whitlock, 1967). Ganser has been clearly distinguished from malingering; in the latter but not in the former the patient is aware of his motivation. Tsoi (1973) concluded from a major investigation that the disorder is best placed on a hysterical–malingering dimension rather than with psychosis. Although there is a consensus for a highly favourable outcome, it is reasonable to suppose that this largely depends on the resolution of the underlying problem or disorder.

The only large-scale epidemiological study appears to be that of Tsoi (1973), who surveyed 1200 admissions to a mental hospital serving the two million people of Singapore. He found 21 suspected and 10 typical cases of Ganser. All were unable to answer simple questions, add up or count (e.g. backwards from 20 to 1), and gave approximate answers (believed by Tsoi to be absolutely characteristic of Ganser). Tsoi concluded that these patients were trying to simulate psychotic illness or exaggerate psychotic experience, according to ideas derived from fellow mental-hospital inmates. The patients came from a wide variety of races and cultures. Most exhibited not all but only some of the features described by Ganser.

As Ganser is not a common disorder (Goldin & Macdonald, 1955) – with probably fewer than 50 cases described – and little is known of its correct management, it is felt that the following case history is interesting and instructive.

Case history

In April 1985, a 26-year-old male patient was remanded to prison for medical reports, having been charged with fraud and deception. Following his arrival there he began behaving in a bizarre way which necessitated his being assessed by one of the authors (MWPC). He claimed that he had served as a mercenary in the Vietnam War, and began demonstrating his skills at kung fu on fellow inmates and staff. He claimed to have alopecia and was seen regularly to pull hair from his head. He was disinhibited, spitting everywhere and smearing the cell with excreta. He was observed apparently replying to non-existent people but declined to explain his strange behaviour. He refused to leave his cell to be interviewed and had to be seen there. He was lying under a dirty blanket and the cell walls were faecally smeared. Once started, he spoke freely and no disorder of talk, behaviour, mood, affect or thought process was discernible. Cognitively he was intact, although there were obvious attempts to falsify his answers, apparently deliberately. Some of his responses were as follows (correct answers given in brackets):

Q. How old are you?
A. 25 [26]
Q. When were you born?
A. In 1962 [1960]
Q. What is the date today?
A. March 1986 [October 1985]
Q. Where do you come from?
A. Vietnam. My parents died when I was five in Vietnam. I married a local girl at 13 and she lives in Vietnam. I was a mercenary during the Vietnam War, serving in the Soviet Army.
Q. How many legs has a horse?
A. 5.
Q. How many legs has a dog?
A. 7.
Q. How many eyes has a cat?
A. Depends on how much he is using its claws.

Later he gave his correct age, and when the inaccuracies in his replies were pointed out, he did not appear concerned. He denied the charges, claimed that he was falsely imprisoned and denied that he was mentally ill. Throughout, there was no unequivocal evidence of hallucinations, delusions, or other psychotic features, and no evidence of treatable psychiatric disorder. No medical recommendation was made, therefore.

He was again assessed in Winchester Prison in April 1986, after he had been refused a place in a regional secure unit, and the Crown Court had ruled that a place be found for him in hospital under Section V(1) of the Criminal Procedure (Insanity Act) 1964. He had been transferred back to Winchester from another prison where he had been since the earlier assessment.

He appeared in a night-shirt and throughout the interview was blank and insouciant. He denied his true name but said he was "Alexander Peron", "on the military warden wing" and that his nationality was "Perronic English". He refused to say where he lived but later denied that he had ever known Harrow, his domicile. To many questions he answered "No" and to others he replied, "That is inconsequential". When asked his age, he replied "18" [26], claimed that he was born in 1960 [correct], but said the year was 1987 and that he was 97 years young. He then changed the year of birth to 1979.

When confronted with his name, he answered, "He is one of my men – an audio-staff sergeant." When asked his regiment, he said "RE, Royal Artillery, Battery A . . .". He refused to say how long he had been in the Army and denied ever serving in the SAS (a claim he had made to the prison authorities). He gave his whereabouts as a "military hospital – the hospital wing". He claimed to know a Gartwick Northerin [? an anagram of Northwick Park]. When asked to spell this he replied "NOPCORXTHEN".

He gave his occupation as "Doctor – a doctor surgical – I don't mean a surgeon – not a pathological – orthopaedics". He then added "Soldiering". When asked where he did this, he replied "Dollon – the inner city" [? anagram of London].

He gave the Queen's name as "Victoria" but did not know her husband's name. When asked for the names of her children, he replied, "I am not one of their army." When asked for the Prime Minister's name he answered, "No consequence – I am not involved in politics." He claimed to be married (untrue) and gave his nationality as "Perronic English". He claimed his wife was guarded by soldiers – "Perronic soldiers". He gave the capital of England as "Dollon", the capital of Scotland as "Aberdeen", of France as "Paris", and of the United States, as "Boston". When asked who was the President of the United States, he said "Farillo" (possibly he meant Ferraro – a name topical in the US at the time). However, he denied knowledge of Gadaffi. His favourite animal was the tiger. When asked how many legs has a horse, a tiger and a cat, he said, "That is a ridiculous question." He claimed to have university degrees and when asked which, he said "Edinburgh University, Pathological, Masonic, Bowie and Akaba", together with "Three fellows' degrees". When asked, "What is Akaba?" he had first replied "A language", then he said, "It is near the Red Sea". When asked, "What is the Red Sea? he said, "It is a lake near Israel".

He claimed to enjoy killing people – "Dead". He then added, "That is a pathological answer, isn't it?" He claimed the prison officers were his staff officers and that he was the platoon sergeant. He had "75 billion" men. When asked whether he heard voices he said, "Yes – somebody on the outside – on the end of a telephone". He then adopted a histrionic listening pose, pointed to his ear and said, "It is an ear lock." He claimed that he had three children and that he had seen them three years ago [untrue]. When asked about his parents, he said they were dead [untrue], and lived in Perronica.

He then added, "Whitehall is in Perronica." He claimed to be able to write Perronican English and obligingly scribbled a lot of nonsense on a piece of paper.

Throughout the interview he remained insolent and indifferent. He talked nonsense as exemplified above, but it was clear that he was in touch with what was happening. Thus he was acquainted with current events, gave several approximate answers when he obviously knew the correct responses, and gave anagrams of several correct replies. He was apparently aware of what he was doing and of talking deliberate rubbish. He otherwise showed no signs of schizophrenia, mania, or other forms of mental illness.

Thus, he appeared to be conveying the layman's idea of madness; that is, the Ganser syndrome. As the prison staff were clear that he had not exhibited any dangerous behaviour, arrangements were made to transfer him to a psychiatric unit.

Subsequently we pieced together the following history of the development of the present condition.

In August 1984 he was psychiatrically assessed at a police station following violence at home when he stuck a skewer into his mother's buttock. He was reassessed in October 1984 and the diagnosis of psychosis was again raised.

However, later that month he dishonestly obtained a British Airways ticket to Dubhai and subsequently made his way to Singapore, where he was admitted to a mental hospital with gross behavioural disturbance. At the request of the managers of the hospital, he was deported. After Christmas in Hong Kong, he reappeared in Bangkok, where he apparently tried to break into the British Embassy at midnight, only narrowly escaping being shot by the Gurkha guard. He was repatriated from Bangkok at a cost of £1519 to H.M. Government.

While awaiting a medical examination at Heathrow he absconded and broke into his parents' house. After three weeks in Aberdeen, he returned to Harrow where he was arrested, set fire to the police cell and was sent to Brixton. It transpired that he had managed to smash up a friend's house, allegedly tried to assault his father with a knife and defaecated outside the house.

His early history is unremarkable. Born in London, he was the fourth of five siblings. He had a normal upbringing in a caring family. His childhood was uneventful and he passed his City and Guilds examination for plumbing and heating with credits at 19 years of age. His premorbid personality was also unremarkable and, apart from a brief experience of amphetamines at the age of 13, apparently he did not abuse drugs or alcohol.

There was no previous history of any significant physical or mental illness and no family history suggestive of mental illness. He first came under psychiatric care in 1980 at the age of 20 when he was briefly admitted under Section 25 of the Mental Health Act. He presented with paranoid psychotic features and a three-month history of gradual change in his behaviour.

Following this he took several small overdoses of drugs. Although he was intermittently under psychiatric care for about five years, with diagnoses varying from a paranoid psychosis to psychopathic personality disorder, an element of deception emerged over the years, and a diagnosis of mental illness was regarded as increasingly unlikely.

Admitted to Northwick Park Hospital in June 1986, he at first continued to behave in a bizarre fashion, talked nonsense and used anagrams. Thus he called himself

"Skroob". After being stabilised on depot fluphenazine decanoate, he suddenly made a marked improvement, became symptom-free and has remained so. A comprehensive physical and neurological investigation was negative.

Discussion

This man presented with a recent history of deception, simulated illness, blatant fabrication and buffoonery. His outlandish claims and statements were transparently untrue. His grotesque behavioural disturbance was on a grand – not to say global – scale. His more bizarre untruths resembled Walter Mitty-isms rather than delusions. He talked and wrote utter nonsense with insouciance. His talk included much rubbish, many approximate and untrue answers, and anagrams of correct replies. His behaviour was sufficiently outlandish as to suggest that it was deliberately 'attention-seeking'. The motivation, however, was obscure. His demonstrations continued in prison but subsided soon after he reached hospital – typical of artefactual illness behaviour (Carney, 1979). He thus conformed to the classical Ganser pattern. However, his quasi-delusions suggested schizophrenia, and this diagnosis was indeed made in 1980, and in support of this is his improvement on being put on depot fluphenazine decanoate. Again, his tendencies to exaggeration and clang associations suggested mania, but the other features of this illness were lacking. Suspicions of drug or alcohol psychoses were not confirmed and there was little in his overall life-pattern to suggest a formulation of aggressive psychopathy. The definitive diagnosis will probably only be made with the passage of time.

How does this case assist in formulating a scheme of management for these uncommon patients? Present advice is conflicting. Hospital admission has been recommended (Enoch & Trethowan, 1979) but found ineffective. Certainly there is a good case for a period of assessment in hospital, because Ganser is possibly merely a presenting symptom of diverse causation and the underlying illness or problem (if any) needs to be identified. This patient's disturbance subsided with admission but, on the face of it, the principle of bringing hospital addicts and illness simulators into hospital seems basically unsound.

However, it may be that our patient has schizophrenia after all, and a satisfactory outcome, however inadvertently, may be achieved with treatment of that condition. This patient certainly improved following admission, but whether this was due to admission to hospital as suggested by Enoch & Trethowan (1979) or to the initiation of antipsychotic medication is unclear. Because of conduct disorder, these patients are likely to appear to forensic psychiatrists.

The hospital staff were reluctant to have this man in and these people are likely to be unpopular patients. However, a brief admission would be worthwhile if it clarified the diagnosis and apparently produced remission, as in our case. Much planning and persuasion of hospital staff have to be undertaken if admission of such patients is contemplated.

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Contents: MAOIs and narcotic analgesics/Comparison of electrical measurements on constant voltage and constant current ECT machines/Self-poisoning/Late paraphrenia/Comparative trial of a new antidepressant/The symbolic significance of Munchausen's syndrome/Down-regulation of post-synaptic serotonin receptors as a mechanism for clomipramine-induced anorgasmia/Abnormal intestinal permeability: an aetiological factor in chronic psychiatric disorders?/Mental handicap and double-blind trial design/Gilles de la Tourette's syndrome in Down's syndrome/Onset of schizophrenia in men and women/Chronic schizophrenia and long-term hospitalisation/DST in apathetic hyperthyroidism/Catatonic signs in schizophrenia/Chronic depression in general practice/John Langdon Down.

MAOIs and Narcotic Analgesics

SIR: Brown & Linter's review (*Journal*, August 1987, **151**, 210–212) was a welcome review of the confused area of interactions between MAOIs and narcotic analgesics, and provided some useful practical guidelines.

The interaction between dextromethorphan and the MAOIs is, however, contentious. The authors refer to a letter by Rivers & Horner (1970) concerning a patient receiving phenelzine (60 mg/day) who died after consuming 2 oz (about 55 ml if standard fluid ounces are assumed) of a cough mixture containing dextromethorphan. This is open to question, as the dose of dextromethorphan was not stated, nor the other ingredients of the preparation, and nor could the death of the patient definitely be attributed to an interaction (Ascione, 1976). A second letter in the same journal also reporting a dextromethorphan–MAOI interaction death (Shamsie & Barriga, 1971) is similarly doubtful, as the preparation also contained phenylephrine, a drug known to interact adversely with MAOIs. Animal experiments (Sinclair, 1973) show an interaction between dextromethorphan and MAOIs. However, the fatal doses used in the rabbits were very high, and a similar reaction can also be produced with imipramine in rabbits. The strict relevance of these animal studies must thus be questioned, since imipramine and MAOIs are used in clinical practice together, albeit with great care, without reported incidents.

The statement that dextromethorphan is absolutely contra-indicated with MAOIs must thus remain open to considerable doubt.

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Comparison of Electrical Measurements on Constant Voltage and Constant Current ECT Machines

SIR: When calculating the energy produced by the constant voltage ECT machine (*Journal*, August 1987, **151**, 244–247), we used a formula based on the assumption of a 50% modified sine wave. The manufacturers (Ectron) state that the machine produces "55% sine waveforms". Examination of the curves produced by the machine in our trial indicates that the waveform is more accurately described as a 60% modified sine wave. In the latter case we have underestimated the energy produced by the constant voltage machine by approximately 40%. Consequently, all quoted values for energy produced by the constant voltage machine should be multiplied by 1.39. The conclusions in the paper are unaffected by the above.

I am grateful to Dr Barrington of Herrison Hospital, Dorchester, for pointing out this error.

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Self-poisoning

Sir: I take the point raised by Halasz (*Journal*, August 1987, **151**, 267) when he mentions my use of the word 'epidemic' in describing a rise in incidence of deliberate self-poisoning in the 1960s and early 1970s (*Journal*, May 1987, **150**, 609–614). I would, however, like to reduce any confusion that might arise from his letter by reminding readers that I was referring not to a suicide epidemic, but to a rise in the incidence of deliberate self-poisoning, the vast majority of instances of which are parasuicides, i.e. non-fatal acts.

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Late Paraphrenia

Sir: Grahame's letter about late paraphrenia (*Journal*, August 1987, **151**, 268) raises important issues which are dealt with rather too casually. Holden may, no doubt, wish to answer criticisms directed at his paper, but it is worth pointing out that he could not possibly have interviewed his patients using the Geriatric Mental State (GMS) since attentive reading of his text will show that the study was a retrospective one based on case notes.

Grahame appears unshakeable in his belief that late paraphrenia is merely a form of schizophrenia in old age because of the high proportion of cases with first rank symptoms in his sample. We would suggest that both the history of the concept and the results of more recent, systematic prospective studies using the Geriatric Mental State (Naguib & Levy, 1987) should lead to a more cautious approach to this perennial problem. As we have pointed out elsewhere (Levy & Naguib, 1985), the proportion of patients with first rank symptoms in our own larger and unselected group of patients studied prospectively was much lower (16 out of 43, compared with Grahame's 14 out of 25). Thus, even if one accepts the debatable proposition that first rank symptoms necessarily indicate schizophrenia, clinical evidence does not indicate a simple equivalence between late paraphrenia and schizophrenia of late onset. Furthermore, the evidence from HLA sub-typing points in the opposite direction (Naguib *et al*, 1987).

The role of organic factors, both as reflected by cognitive tests and by CT scan measures (Naguib & Levy, 1987), is also rather more complex than Grahame is prepared to concede. We have found minor cognitive changes and ventricular enlargement in a group of late paraphrenics, and follow-up 3.7 years later did not suggest that this was a harbinger of 'organic brain syndrome', as Grahame seems to believe, nor did it bear any obvious relationship to the clinical picture in the survivors (Hymas *et al*, in preparation).

The concept of late paraphrenia or persistant persecutory state in the elderly is one which is peculiarly robust. It is unfortunate that the draft of ICD–10 (World Health Organization, 1987), currently undergoing field trials, will, if accepted, mean that such patients will be distributed among at least four different sub-categories, thus making them somewhat difficult to identify. Nevertheless, identification should be possible, and we believe that the application of a variety of new techniques to the investigation of the problem should begin to clarify the nosological status of this entity or entities. In the meantime, it would be wise to keep an open mind about the question rather than to adopt unduly dogmatic attitudes towards it.

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Comparative Trial of a New Antidepressant

Sir: I strongly agree with Dunn's comments (*Journal*, August 1987, **151**, 269) on the study by Levine *et al* (Journal, May 1987, **150**, 653–655), comparing fluoxetine and imipramine.

There can surely be no doubt that new putative antidepressant compounds require testing against placebo in properly controlled double-blind studies before their efficacy can be regarded as established. Comparative trials against other 'established' antidepressants can never prove the efficacy of a new compound (although studies against the standard may have a place later in a compound's development). The main problem with trials against a standard antidepressant is that the investigators predict no difference between the two groups of patients. However, there is no way of testing the absence of a difference between groups. In contrast, testing a compound against a placebo leads to the prediction of a difference which can be tested by ordinary statistical techniques. Indeed, it is extremely easy to obtain a result of no difference between two treatment groups. One simply enters too few patients for statistical significance or makes any of the other design errors which beset even the best psychopharmacology trialists.

To take an example from the Levine *et al* study, patients in the imipramine comparison group were treated with 75–150 mg/day. In the event seven patients were treated at the 75 mg dose. This is well accepted to be inadequate, and a recent study (Thompson & Thompson, in preparation) confirms the inefficacy of 75 mg of a tricyclic antidepressant. Thus, one-third of the standard treatment group received a treatment which was no better than placebo, and a fluoxetine-treated group faired no better in terms of outcome than the imipramine-treated group. I would suggest that this study does not provide evidence of the effectiveness of fluoxetine as an antidepressant, and that only studies against placebo can do this.

There is one point on which I would disagree with Dunn. The use of atropine as a comparison treatment in antidepressant studies is in doubt, as it induces not only the side-effects of the older antidepressants but also may have some intrinsic antidepressant activity. The more appropriate "active placebo" would be methscopolamine, which has peripheral anticholinergic effects but does not pass the blood–brain barrier. Unfortunately, this compound is difficult to obtain and is thus not frequently used. However, in view of the relative lack of anticholinergic effects in the newer generation of antidepressants which are currently being tested, this may not be such a major flaw in current antidepressant trial design.

If psychopharmacologists are shying away from placebo-controlled antidepressant studies for ethical reasons, or if ethical committees are refusing to pass placebo-controlled studies, then they should consider the ethical implications of allowing a potentially ineffective antidepressant or any other compound onto an already overcrowded market.

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The Symbolic Significance of Munchausen's Syndrome

SIR: Cremona-Barbaro's paper on the symbolic significance of Munchausen's syndrome (*Journal*, July 1987, **151**, 76–79) prompts comment. Fanciful, dramatic and grandiose presentation is the hallmark of the Munchausen patient, and there may be some relationship between feigned bereavement and the syndrome, but it is by no means "the most common story" (Scoggin, 1983). There has been no previous mention of preoccupation with opera and operatic themes in the literature. Certainly none of our patients exhibited any such interest in the arts (O'Shea, 1987: personal communication).

The aetiology of Munchausen's syndrome would seem to be multifactorial, but there is growing evidence to support a transgenerational learning theory of maladaptive communication patterns with roots in early childhood (O'Shea, 1982; Meadow, 1982).

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SIR: It is difficult to know exactly what is "the most common story" given by Munchausen patients presenting with psychosocial symptomatology. Feigned bereavement is commonly accepted to be a very frequent presentation (Simpson, 1978; Snowdon *et al*, 1978), and will be familiar to psychiatrists and to those working in A & E departments. It is overwhelmingly the commonest story given in my own small series of Munchausen patients presenting with psychiatric symptoms.

I would agree that there has been no previous mention of a preoccupation with the opera in the world literature on the Munchausen syndrome. Had there been, my point may have been less worth making. In my own experience, these patients refer to the opera more often than one would expect. A case report published some years ago illustrates this point (Cremona-Barbaro, 1983).

I would certainly not disagree with the view that the aetiology of the Munchausen syndrome is likely to be multifactorial, and it would seem reasonable to assume that maladaptive learning in early childhood is an important factor.

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Down-regulation of Post-synaptic Serotonin Receptors as a Mechanism for Clomipramine-induced Anorgasmia

Sir: In considering likely mechanisms for clomipramine-induced anorgasmia, Monteiro *et al* (*Journal*, July 1987, **151**, 102–106) have discounted an effect on central serotonergic transmission. When, however, an injection of a post-synaptic serotonin receptor agonist, 5-methoxy-N, N-dimethyltryptamine (5-MeODMT), is used to induce ejaculation in rats, this effect can be blocked by selective serotonin uptake inhibitors (Renyi, 1986). A single dose of zimeldine inhibits ejaculation when given 48 hours before administering 5-MeODMT. In contrast to Renyi's findings at 48 hours, Mas *et al* (1985) found that zimeldine, like 5-MeODMT, facilitated ejaculatory reflexes when animals were tested one hour after dosing. They also demonstrated that zimeldine's effect on ejaculation, unlike that of the direct agonist, does not occur in animals bearing mid-thoracic spinal cord transections, demonstrating that 5-MeODMT exerts its agonist effect directly on serotonin receptors in the spinal cord or periphery, while zimeldine's effect depends on intact supraspinal innervation.

Taken together, these drug-induced changes in ejaculation can be interpreted as a reflection of the sequence of synaptic events that follow serotonin re-uptake blockade, namely an initial increased concentration of intrasynaptic serotonin with enhancement of neurotransmission, leading to down-regulation of post-synaptic serotonin receptors establishing functional inhibition of neurotransmission by 48 hours (such down-regulation of serotonin receptors has been demonstrated within 3 hours of drug administration by Koshikawa *et al* (cited by Renyi, 1986).

As Mas *et al* point out, the lumbosacral segments of the spinal cord receive descending serotonergic fibres from the raphe nuclei in the same laminae of the anterior horn as the motor and autonomic preganglionic neurones innervating the genitalia.

Chemically-induced ejaculation in paraplegic rats may seem a questionable anthropomorphism to serve as a model of the human orgasm, but this research is cited merely to suggest that serotonergic dysfunction is the best hypothesis for clomipramine-induced anorgasmia.

There have been two case reports of antidepressant-induced anorgasmia in which normal orgasmic function was restored by treatment with the serotonin receptor agonist cyproheptadine while antidepressant treatment was continued (Decastro, 1985; Sovner, 1984). While neither of these cases implicated clomipramine, both drugs – a MAOI and nortryptiline – are known to exert a similar influence on serotonergic transmission. It remains, therefore, to test the acceptability and efficacy of cyproheptadine as a treatment for antidepressant-induced anorgasmia.

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Abnormal Intestinal Permeability: An Aetiological Factor in Chronic Psychiatric Disorders?

Sir: Wood *et al*'s paper (*Journal*, June 1987, **151**, 853–856) presents some puzzling areas which are in need of clarification. The authors stated that the mean cellobiose recovery rate after intravenous injection was 28% (s.d. = 10.8%) in patients who showed an abnormal ratio after oral testing and 29.3% (s.d. = 17.7%) in those with a normal ratio, over a five-hour period, concluding that there was no significant difference between these two groups of patients in the metabolism or excretion of this probe molecule after absorption. These recovery rates after intravenous injection are extremely low; indeed, in their previous work (Cobden *et al*, 1985) a mean cellobiose recovery rate of 52.0% (s.d. = 14.3%) was reported, and Menzies (unpublished) obtained an 83.4% recovery rate in normal subjects over the same time period. Irrespective of whether the patients had a normal or abnormal cellobiose/mannitol recovery ratio, due to the low cellobiose recovery intravenously in both populations one might conclude that considerable systemic metabolism of this probe marker had occurred. However, cellobiose, in common with most other disaccharides, is not known to be significantly metabolised within the body (Menzies, 1974). As intravenous cellobiose was not administered to a population free of psychiatric illness, these results would equally well support the hypothesis that patients with chronic psychiatric illness demonstrate abnormal systemic metabolism of cellobiose.

The authors acknowledged the low recovery rates of both probe molecules, postulating that this may have been due to inaccurately timed urine collections, leading to a similar reduction in recovery of both molecules, leaving the cellobiose/mannitol ratio unaffected. However, the mean mannitol recovery rate of 49.45% (mean, abnormal/normal patients) is almost identical to the 49.9% recovery in normal controls (Cobden *et al*, 1985), but the cellobiose recovery ratio is some 23.35% lower (52.0%, compared with 28.65%). This disproportionate lowering of the cellobiose recovery rate is not compatible with error introduced by inaccurately timed urine collections.

Furthermore, it is unclear whether the test solution administered was hyperosmolar. If it was, it is important that subjects refrain from drinking water for at least two and a half hours before and after the commencement of the study, as water would act to dilute the hyperosmolar stress, leading to difficulties in interpreting the results. As the authors have alluded to difficulties in obtaining complete timed urine collections in this group of patients, it would be reassuring to know that 'fasting' included preventing the subjects swallowing water throughout this period, a somewhat natural reaction after ingesting an extremely sweet sugary drink.

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Menzies, I. S. (1974) Absorption of intact oligosaccharide in health and disease. *Biochemical Society Transactions*, **2**, 1042–1047.

Sir: We are grateful to McGauley for drawing attention to the low five-hour recovery of cellobiose after i.v. injection in patients with chronic psychiatric disorder. As stated, the recovery is some 23% lower than in the previous study in fit, co-operative volunteers. In theory this could be due to altered metabolism, but may reflect difficulty ensuring complete bladder emptying before starting and at completion of the test in the patients we studied. It is incorrect to presume that the abnormality demonstrated in the oral test could have been due to metabolic differences, as this would have given rise to an apparent reduction in permeability to cellobiose, whereas in fact we have demonstrated the opposite.

The purpose of the i.v. test was to compare the group of psychiatric patients with abnormal oral tests with those who had a normal oral sugar test. The i.v. injection was given before the patient was allowed away from his bed and after a urine specimen was collected. The patients did not have access to food or water during the five-hour period when all urine passed was collected. Endoscopy was performed at the end of the i.v. test and no excessive gastric contents were noted. The i.v. test therefore confirmed no significant difference in metabolism or excretion in the two groups studied.

The composition of the oral test solution is clearly stated in the method section. Patients were asked not to drink, but in order to retain co-operation some freedom was allowed and it is possible that some may have drunk water during the study. However, this would tend to reduce permeability to cellobiose, whereas in fact the study demonstrated an increased permeability to cellobiose in the abnormal group.

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Mental handicap and double-blind trial design

Sir: The title "Lithium in the treatment of aggression in mentally handicapped patients: a double-blind trial" (*Journal*, May 1987, **150**, 685–689) raises an interesting question about how the limited conceptual ability implicit in mental handicap might interact with the conceptual sophistication necessary to understand a double-blind design. That the interaction between mental handicap and a double-blind design might have undertones is conceded in the statement, "The nature of the trial and the procedures involved were explained to each patient (at an appropriate level) and to a relative or responsible guardian". Just how much each participant actually understood is unknown, however, because of the lack of operational criteria showing that they understood, as distinct from being in receipt of an explanation. It appears that an opportunity to establish such criteria was lost; for example, each participant should be able to explain the procedure back to the researcher.

However, the more compelling scientific issues concern the other half of the "double-blind" design, the researchers. The outcome variable, levels of aggression, was assessed daily by the nursing staff on duty, who differed during the study; none of them were asked to guess the allocation of the patients. This would have been a prudent precaution given that, "The initial daily dose of 800 mg lithium carbonate was not sufficient, in most cases, to bring the serum lithium concentrate above 0.7 mmol/litre, and subsequently dosage adjustments were needed...". But we are not told that equivalent adjustments were made for the patients receiving placebo, so that one wonders what the nursing staff made of a group of patients who were having their medication adjusted and another group who were not having such adjustments.

Also, side-effects may have betrayed treatment allocation. Although the medical officer assessed each patient for the occurrence of possible side-effects of lithium treatment both before the trial and after one, two, three, six, nine, and twelve weeks, it is naive and unnecessary to suppose that these results, arising as they do out of interviews at specific points of time, generalised to what may have passed between the patients and the nurses who look after them 24 hours a day. It would have been easy to test the nurses themselves. Again, what was the mnesic ability of these patients? How reasonable was it to suppose that at an interview at a given point in time they would recall what had happened in between those points in time?

With this perspective, the authors' claim that blindness was maintained because classical side-effects were noted in 36% of the lithium patients and 20% of placebo patients is unwarranted, particularly as the exact means whereby the side-effects were detected remain apocryphal. Did the medical officer await spontaneous complaints? Did he or she ask a direct question, and if so what question? Was a check-list used?

Why were the measures of aggression made by different members of nursing staff? Given that they were, why was there not a reliability study of these staff? Given that there wasn't such a study, why were the results not analysed for individual nurses? The five-point scale whereby level of aggression was assessed is, on paper, no better than ordinal, the more so given that it may have been used in different ways by the different nursing staff; there was therefore good reason to suppose that the scale did not have interval qualities, and no evidence was presented to gainsay that, so how do the authors justify the use of parametric statistics?

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SIR: According to the Declaration of Helsinki, "In any research on human beings each potential subject must be adequately informed of the aims, methods, anticipated benefits and potential hazards of the study and the discomfort it may entail.... Where physical or mental incapacity makes it impossible to obtain informed consent... permission from a responsible relative replaces that of the subject in accordance with national legislation". Thus it is not necessary for a mentally handicapped patient to be able to understand a double-blind design in order to participate in such a trial.

Regarding blindness, in the method section of our paper we described how dummy results for serum lithium concentrations were provided for placebo patients. The medical officer responsible for each patient adjusted the dosage of trial medication accordingly, so that both he and the nursing staff remained unaware of whether the patient was being treated with lithium or placebo.

Records were kept by the nursing staff of the type, duration, and severity of side-effects, as and when they occurred, using a check-list of common lithium side-effects.

It cannot be possible in a hospital to have daily assessments carried out by the same person over a 16-week period. Before undertaking this trial we carried out a reliability study, using the same 5-point rating scale for aggression, to assess the correlation between scores given by different nurse assessors. Statistical analysis of the results showed that while the score of 2 (mood uncertain) was the least reliable, scores of 1 (well-behaved) and 3 or more (overt aggression) achieved satisfactory levels of correlation between separate assessors. For this reason the analysis of our trial results concentrated on these scores.

In our trial it seemed reasonable to use parametric methods for analysis of the scores obtained with the

5-point scale because the data being analysed were themselves summary statistics. However, due to the discrete nature of the data, and to the question of how different intervals on the fixed point scales related to each other, it would, alternatively, have been appropriate to adopt a non-parametric approach.

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Gilles de la Tourette's Syndrome in Down's Syndrome

SIR: We recently documented the occurrence and treatment of Gilles de la Tourette's syndrome (GTS) in an individual with Down's syndrome (*Journal,* May 1986, **148**, 601–604). Corbett (*Journal,* April 1987, **150,** 569) suggests that carbamazepine may have accounted for the late onset of GTS in this patient. However, as we indicated in the case report, the patient had been observed by nursing staff to have consistently displayed motor and phonic tics since the age of 11, when she was first institutionalised. We also mentioned that GTS had been diagnosed according to DSM–III criteria, which stipulate that age of onset must occur between 2 and 15 years. In contrast, carbamazepine had only first been used by this patient when she was aged 24, following the onset of a possible seizure disorder.

Corbett correctly points out that pharmacological compounds are increasingly being recognised as precipitants of tics. As indicated in the case report, although we attempted to correlate the presence and frequency of tics to the patient's previous use of medication we felt unable to do so accurately in view of the limited information available. However, tics appeared to have been continuously present since at least the age of 11, an interval that spanned the non-continuous use of several medications specified in the case report, and antedated the introduction of carbamazepine by 13 years. Although carbamazepine possibly exacerbated her symptomatology, it was clearly not responsible for the onset of GTS manifestations in this patient.

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Onset of Schizophrenia in Men and Women

SIR: Stromgren has referred to studies indicating that onset of schizophrenia occurs later in women (*Journal,* January 1987, **150,** 1–7). Our experience in India is different.

We have been engaged in a multicentre 5-year follow-up study of schizophrenia, sponsored by the Indian Council of Medical Research, to find out factors associated with the course and outcome of schizophrenia. The centres selected for this study were Lucknow, Madras, and Vellore. All patients who attended psychiatry clinics in the participating centres between 15 October 1981 and 15 October 1982 and who satisfied a modified form of Feighner's criteria for the diagnosis of schizophrenia were included in the study (245 men and 141 women) (Verghese *et al,* 1985). The age of onset was calculated from the age of the patient at the time of inclusion and the time when the earliest abnormality was noticed (duration of illness). Both these pieces of information were given by the close relatives of the patients. Age of onset was found to be 25.87 ± 6.67 years for men and 25.89 ± 7.33 years for women, which suggests that age of onset of schizophrenia is not later in women in all parts of the world.

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Chronic Schizophrenia and Long-Term Hospitalisation

Sir: It is always useful for a psychiatrist interested in long-term patients to re-read *Institutionalism and Schizophrenia* (Wing & Brown, 1970), as Wing has reminded me (*Journal*, January 1987, **150**, 129–130). I had hitherto thought that to do so with an eye to the questions it raises was fully in tune with the authors' intentions.

I do not dispute the book's conclusion that the negative handicaps of patients with schizophrenia are influenced by their current social milieu. My letter (*Journal*, September 1986, **149**, 382) was prompted partly by concern that its evidence was given insufficient weight by Mathai & Gobinath (*Journal*, May 1986, **148**, 509–516). I raised the different question of whether or not the institutional environment has a *cumulative* effect on negative handicaps such that they worsen progressively in the long-term, contrary to the case outside hospital (Bleuler, 1978).

I find it difficult to accept Wing's assertion that no one pattern of relationship between negative handicaps and length of stay in hospital is described. A summary (p. 87) states that "patients who were living in the most under-stimulating social environment were likely to show the greatest clinical poverty, and this complex was likely to be more severe the longer the patient has been in hospital"; a graph is said to show (Fig. 4.4 and p. 82) "the striking relationship between length of stay and three other factors, one 'primary' (social withdrawal), one 'secondary' (attitudes to discharge), and one 'social' (contact with the outside)"; and most revealingly (p. 183), "if social improvements had not been taking place at the hospitals the tendency would have been for the patients to have become more withdrawn and reticent, simply with the passing of the years. That such deterioration was mainly prevented, and that so large a proportion as one-fifth of the patients actually improved, is a considerable achievement".

I pointed out that the longitudinal course of individual patients shows the same tendency for deterioration to be concentrated in the early years, with later consistency, or even improvement, described outside hospital. The statistical association between length of stay and negative handicaps appears to result from selection combined with changes in the initial pattern of schizophrenic symptoms (Abrahamson, 1983).

The methodological issue is illustrated by Wing & Brown's treatment of the association between length of stay and unfavourable attitudes to discharge. The same graph is used to calculate the rate at which patients supposedly changed their attitudes year by year and leads on to the recently reiterated conclusion (Wing & Furlong, 1986) that negative attitudes accumulate gradually as part of a process of 'institutionalism'. This assumes that patients' initial attitudes have always been similar and ignores the possibility that those changes that do accumulate may follow discrete events such as loss of family or a home or transfer to a particular ward.

As the institutional era winds down it is important to refine the information with which we are left. That negative schizophrenic symptoms may be sensitive to the immediate effects of an impoverished environment yet resistant to its assumed long-term, cumulative moulding is relevant to future care and to any socio-medical model.

Equally, evidence that attitudes to discharge reflect realities rather than being explained by the gradual development of a secondary handicap has practical and ethical implications (Abrahamson & Brenner, 1982).

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DST in Apathetic Hyperthyroidism

Lahey (1931) drew attention to a form of hyperthyroidism which he called 'apathetic hyperthyroidism', occurring mainly in the elderly and lacking many of the cardinal features of thyroid overactivity. The predominant features were depression and apathy, and subsequent reports have confirmed his initial description (Thomas *et al*, 1970; Brenner, 1978). We report a patient fitting the description of apathetic hyperthyroidism in whom the dexamethasone suppression test (DST) was carried out.

Case report: A 69-year old widow presented with a three and a half year history of social withdrawal and isolation to the degree that in the six months prior to presentation she led the life of a recluse. In addition, she complained of persistent depression, hopelessness, loss of interest, loss of drive, anergia, and suicidal ideas. On examination she appeared thin and frail. Her pulse was 76 per minute and regular, blood pressure was 130/80 mm Hg. There were no tremors, eye signs, or goitre. A diagnosis of depressive illness was made and she was prescribed lofepramine. In addition to other routine investigations thyroid function tests were made, and the results showed a raised serum thyroxine (167 nmol/l, reference range 50–150 nmol/l) and a low TSH (0.1 mU/l, reference range 0.5–5.0 mU/l). DST showed normal suppression of cortisol levels. A diagnosis of apathetic hyperthyroidism was made and she was started on carbimazole (10 mg tds).

Apathetic thyrotoxicosis occurs most often in elderly patients, and the salient characteristics that are helpful in establishing the diagnosis include typical placid apathetic facies, a small goitre, the presence of depression or lethargy, absence of ocular manifestations usually associated with hyperthyroidism, substantial muscular weakness and wasting, excessive weight loss, and cardiovascular dysfunction with atrial fibrillation (Thomas *et al*, 1970). Our patient showed many of these features. It is also of interest that the DST showed non-suppression. This is a confirmation of the reports by Kronfol *et al* (1982) and Martin & Waltz (1984), showing that DST is unaffected in depressions secondary to thyrotoxicosis.

The pathogenetic events leading to a state of apathy and depression in these patients is unknown. However, it has been postulated that there is a depletion of catecholamines or a lack of end-organ sensitivity following continual stimulation of the sympathetic nervous system.

A diagnosis of 'apathetic thyrotoxicosis' must be borne in mind when an elderly patient presents with symptoms of depression and apathy. Treatment of the endocrine dysfunction will often produce a marked improvement of the condition.

We acknowledge the assistance of Dr Oyebode.

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Catatonic Signs in Schizophrenia

SIR: Catatonia is a not uncommon syndrome of varied aetiology (Gelenberg, 1976). We report a patient with catatonia in whom a curious combination of catatonic signs existed. Our use of terms is in accordance with the definitions of catatonic signs cited by Hamilton (1976).

Case report: Mrs L, a 60-year-old institutionalised patient with chronic schizophrenia, was prone to episodes of transient catatonic stupor, usually lasting a few hours at a time, at a frequency of about one per year. Despite extensive biochemical, radiological and electroencephalographical investigation no causative factor was demonstrated, and the catatonia was concluded to be a stress-related feature of her schizophrenic illness. The catatonia was invariably characterised by stupor and diffuse waxy flexibility.

During one episode of stupor, however, we noticed that different parts of her body responded differently to the examiner's touch. Active negativism was present from the neck up: there was strong resistance to the examiner's efforts to open her eyelids, lower her jaw or rotate her head. Waxy flexibility characterised her upper limbs: these could be moulded into positions which were maintained for several minutes despite potential discomfort. *Mitgehen* was demonstrated in her lower limbs: gentle pressure led to the limb moving readily in the direction of the applied pressure, despite the examiner's injunctions that she need not move. It thus appeared that there existed a cranio-caudal gradient of voluntariness to movement in obedience to the examiner's guidance, leading to what would seem to be a combination of logically antithetical catatonic signs. She recovered within three hours; her subsequent course was uneventful.

We are unable to ascribe any clinical significance to this combination of seemingly antithetical catatonic signs.

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Chronic Depression in General Practice

SIR: After 10 years in general practice I have developed a small following of people suffering from chronic depression. Turning to the textbooks for help with management, I discovered that of 14 by British authors, 10 of which were published since 1980, only one had a reference to the problem in the index and this was the only book to have a section (of some substance) devoted to chronic depression. Resistant depression was mentioned in two other books, but I detected uncertainty as to the definition of this. I realise the distinction between the two may be difficult, sometimes only to be made after a long time. However, they are different, and possibly some clarification is needed here.

Other reading suggests that it is a significant problem which has been recognised for several years: a leading article in the *British Medical Journal* (1971), after examining a variety of studies, suggested that "for practical purposes we would not go far astray if we reckoned that one-fifth of our severely depressed patients would become chronic." DSM–III suggests that in 20–35% of cases of major affective disorder there is a chronic course. However, it is not clear how they would classify it, dysthymic disorder being otherwise entitled depressive neurosis. At an international symposium Kieholz (1972) discussed chronic depression – "an important, albeit somewhat neglected, form of the disease".

The management of acute endogenous depression, once identified, seems relatively easy with careful assessment, optimistic support, social consideration, medication, and sometimes hospitalisation and ECT. The management of chronic endogenous depression seems more difficult, all of these aspects of care presenting greater problems. I would be interested in readers' comments.

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John Langdon Down

SIR: Recently, while researching 19th century methods of disposal of the dead, I visited Woking Crematorium, the oldest in Britain. I was invited into the boardroom in order to study the death certificate of the first person to be cremated at Woking. Mrs Jeannette Caroline Pickersgill of Regent's Park died, aged 71, of broncho-pneumonia and asthema (*sic*) in March 1885. The certifying doctor was John Langdon Down, who was in private practice in Harley Street at the time. This eminent Victorian, one-time Medical Superintendent of Earlswood Asylum for Idiots, founder of Normansfield Mental Hospital, who has been credited with a description of Frohlich's syndrome 40 years before Frohlich, and who is best remembered for giving his name to Down's syndrome, also earned himself a small but honourable mention in the history of cremation.

ROBERT WILKINS

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A HUNDRED YEARS AGO

Mental overwork treated by issues

Dr A. P. Langeveld, writing in the *Weekblad van het Nederlandsch Tidjdschrift voor Geneeskunde* on functional affections of the brain and spinal cord, mentions the case of a schoolmaster who was suffering from mental overwork. He complained of a painful sensation of pressure in the head and along the spine. He could not speak for five minutes or walk more than a few steps, and could scarcely read or write. Cold douches, a seton in the arm, mental rest, iodide of potassium, arsenic, and aromatic sulphuric acid producing no improvement, the writer remembered the anatomical connections of the spinal veins, and considered that a congested condition of these might very easily produce cerebral and spinal troubles. The connection of these veins with those on the surface has been pointed out by Breschet. A large blister was applied near the sacrum, and kept open by antimonial ointment, and a week later a second similar blister was applied near the first. It was interesting to mark the diminution of the pain in the head and back as the consequence of the very painful sacral sores. The mental condition began slowly to improve, but when any attempt was made to discontinue the irritant ointment it immediately became worse again. After a time, as the sacral sores were very deep, two issues were applied near the left scapula. After a considerable time the patient was able to take exercise, and ultimately to resume his occupation. The sores were healed by iodoform vaseline.

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Researched by Henry Rollin, Emeritus Consultant Psychiatrist, Horton Hospital, Surrey.

British Journal of Psychiatry (1987), **151**, 711–717

Book Reviews

Editor: Sydney Crown

Research Paradigms in Psychosomatic Medicine. Edited by G. A. Fava and T. N. Wise. Basel: S. Karger A. G. 1987. 272 pp. SFr 175, US$97.25, £79.60.

This volume will be of wide appeal. It gathers together critical reviews of a variety of research procedures and gives a series of updated reviews on recent research.

There is a general introduction on methodological issues in psychosomatic research, contributed by the editors, then four chapters on life events, the quantification of psychological stress, rating scales of anxiety and depression, and personality assessment. The latter chapter brings some order and hope to a subject bedevilled by decades of lurching from subjectivism to superficial objectivity and back again.

A chapter on behavioural assessment, using pain as an example, gives a behavioural analysis of diagnosis and treatment which can be applied to a variety of somatic problems. Another contributor describes a variety of measures that can be applied to somatisation and abnormal illness behaviour.

The vexed issue of body image in eating disorders is discussed. There is a masterly review of the psychophysiological approach that has promised so much and, with increasingly refined instrumentation, continues to have promise for the future.

The chapter on neuropsychology is a delight. Mirsky & Duncan emphasise that it is the way in which the subject goes about the test that is relevant rather than the final score, and this approach requires a high degree of training in the psychologist.

Holsboer's "Psychoendocrine strategies" is outstanding. It is devoted largely to a description of the hypothalamic pituitary adrenal axis in affective illness and the direct effects of steroids on neurological function. There is some fascinating material on the possible direct effect of corticotrophin-releasing hormone and ACTH on cerebral function, and an analysis of the effects of endocrines on the immune system and the effects of the immune system on cerebral functions through the release of peripherally produced hormones and peptides, an area of interaction which would seem most fruitful for understanding psychosomatic inter-relations. The theme is continued in a chapter on "Mind and immunity". A final chapter makes out a lawyer's case that anorexia nervosa and depressive illness have much in common.

This is a very rich feast at a very rich price. Despite the diverse nationalities of the authors, the text throughout is written in a very elegant style of English, although a remarkable number of spelling errors have slipped through. The chapters on questionnaires overlap considerably in their discussions of reliability and validity, and some pruning or cross-reference would have been helpful. It is irritating that different systems of referencing are used for different chapters.

I would encourage everyone interested in this field to read this volume – however much they may know about some of the issues discussed, the range is so great that the reader is bound to learn much that he did not know before.

Oscar W. Hill, *Consultant Psychiatrist, The Middlesex Hospital, London*

Integrated Family Therapy: A Problem-centred Psychodynamic Approach. By David Will and Robert M. Wrate. London: Tavistock. 1985. 161 pp. £17.95 (hb), £7.95 (pb)

This brief book, supported by a foreword from Nathan Epstein, is the best introduction I know to the practice of family therapy. The field can be bewildering for beginners because of the wide range of theories and methods applied, each with its eloquent exponents, but with so far little research-based evidence for the validity of the underlying ideas or the efficacy of the particular technique of intervention. The authors, both adolescent psychiatrists and also teachers at the Scottish Institute of Human Relations, note the evangelicism that persists in the family therapy literature and that often prevents students from integrating what is helpful in the different models ("... the prosetylising phase of family therapy development has encouraged therapists to practice exclusively one particular approach ..."). Their book aims to remedy this.

Will & Wrate integrate three models: the active approaches of the McMaster, problem-centred school (experiential and insight-oriented); Minuchin's structural family therapy; and psychoanalytic understanding of family transactions and of the complexities of individual's inner lives. Their's is an "open, directive approach" in which, in contrast to strategic methods, there is mutual collaboration with the family in exploring, understanding, and resolving the family's problems. The unacknowledged role of psychodynamic understanding in many family therapy interventions is stressed, as is the gap between what therapists say and what they do. This, too, the authors aim to overcome.

The stages of therapy are listed and the executive skills of therapists discussed in detail. The book ends with a helpful outline for a family therapy training programme, and a chapter on research.

I note only two shortcomings. Firstly, there is no mention of families with young children. The special skills needed to help pre-school and early school-aged children participate in therapeutic family transactions, and trainees and parents to facilitate and understand the significance of play, are not described. Secondly, and this is related to the somewhat sketchy account of family therapy research, there is little discussion of the indications for or against family therapy, as compared with other treatment interventions.

This attractive book is written clearly and enlivened throughout by case histories and brief clinical examples. Everyone concerned with the practice of child and adolescent psychiatry should read it.

SULA WOLFE, *Honorary Fellow, University of Edinburgh Department of Psychiatry*

The Medical Basis of Psychiatry. Edited by G. WINOKUR and P. J. CLAYTON. Philadelphia: Balliere Tindall. 1986. 599 pp. £49.50.

The notion of constructing a textbook of psychiatry on the basis of treating psychiatric problems as analogous to other medical problems, defining psychiatric illness as a medical illness with major emotional and behavioural aspects, and presenting the material in the familiar pattern of definition, epidemiology, signs and symptoms, aetiology, treatment, and differential diagnosis, although commonplace to British psychiatrists, is positively revolutionary in the USA and reflects the increasing ascendency of biological psychiatry in that country. Nevertheless, the contributors to this volume by no means neglect the psychosocial and psychotherapeutic aspects of the various disorders included.

Thirty-seven of America's most prestigious psychiatrists have combined to produce an uncommonly fine textbook. Each chapter is a well-referenced and up-to-date review of its subject, often supplemented by brief, but cogent, editorial interpolations. The contents are organised into four sections; 17 chapters on adult syndromes, covering the usual range of conditions, 4 chapters on child psychiatry, covering conduct disorders and hyperactivity, major affective disorders, infantile autism and schizophrenia in childhood, and emotional disorders; 7 chapters on symptom clusters; and 5 chapters on special areas. Included in this last group are the neurobiological basis of psychiatric illness, genetics of psychiatric disorders, use of the laboratory in psychiatry, epidemiology of psychiatric illness, and suicide and attempted suicide.

The entire contents are of a consistently high standard and it is difficult to select any chapters as being of particular distinction. Reflecting my own interests, I found the chapters on organic brain syndromes presenting with little or no cognitive impairment (Popkin), schizophrenia (Tsuang & Loyd) and special and unusual psychiatric syndromes (Nasrallah) to be particularly noteworthy for their lucidity and topicality.

The book is recommended both as a useful revision primer for the trainee and as a comprehensive reference work for the mature practitioner. Its expense is commensurate with its size and excellent presentation.

KENNETH DAVISON, *Consultant Psychiatrist, Newcastle General Hospital*

Between Give and Take: A Clinical Guide to Contextual Therapy. By I. BOSZORMENYI-NAGY and B. R. KRANSER. New York: Brunner/Mazel. 1986. 432 pp. $35.00.

Boszormenyi-Nagy, one of the founders of the family therapy movement, has, over a period of forty years, developed an approach called 'contextual therapy'. In this book he and his co-worker elaborate the principles and practice of the method.

The distinctive concept of contextual therapy, 'relational reality', amplifies and applies Martin Buber's notion of 'genuine dialogue'. Dialogue means mutual commitment: a trustworthy relationship in which relating partners have responsible concern for the validity of each other's interest. Four basic dimensions of relational reality guide the therapist in assessment and treatment: *factual predeterminants*, e.g. poverty or child abuse; *individual psychology*, using concepts of individuation and differentiation greatly influenced by Erickson's work and by object relations theory; *patterns of transaction and power*, drawing upon systems theory; and *'relational ethics'*. The latter fundamental ethical dimension, stating the obligation to achieve a 'fair give and take', involves such questions as what a person owes, deserves, and needs in significant relationships. The authors claim that an enhanced personal freedom derives from 'entitlement' which, earned through offering care, flows from the resolve to accept active and personal responsibility for the consequences of 'relational reality'. That responsibility extends to generations as yet unborn.

The focus of therapy is on the balance of fair exchanges between 'individual members in their close relatedness', rather than on the family. The family therapist needs to practice 'multi-directed partiality', i.e. a 'sequential siding' with each member – very different from collusion – while being accountable to all, including absent members. An important chapter stresses the necessity for the therapist to realise his own relational reality.

The contextual approach is highly optimistic. Rather than focusing on pathology, the aim is to tap residual resources of trustworthiness in close relationships, however sterile and destructive they may appear to be.

This overlong book is wordy, repetititious, diffuse, and replete with difficult and sometimes nebulous concepts such as 'intrinsic relational tribunal' and 'rejunction'. Nevertheless, it has much to offer a patient reader, not least the verbatim transcripts of clinical sessions. Its major contribution is the thought-provoking insistence on the importance of ethical choice in the process and context of psychotherapy.

ROBERT F. HOBSON, *Reader in Psychotherapy, University of Manchester*

Decline and Fall of the Freudian Empire. By HANS EYSENCK. Middlesex: Penguin Books. 1985. 224 pp. £3.95.

It has become fashionable over recent years among those who are unconvinced of the value of psychoanalysis to follow Karl Popper and pronounce it irrefutable, thereby denying it scientific status but allowing it some epistemological significance. Eysenck will give no such escape route for Freud, his ideas, or psychoanalysis. For him, psychoanalysis should be taken as a science in that it puts forward testable hypotheses which can be refuted, and he is satisfied that they have been. Furthermore, he sees it as not only bogus but dangerous.

For anyone who wants to take psychoanalysis seriously there are some very worrying and perplexing things about Freud, the theories that have developed from his work, and the clinical practice deriving from it. Eysenck identifies some of these and begins to build an effective attack in his usual compelling style. However, his purpose emerges as annihilatory rather than constructive, and because of that the book is seriously weakened. To anyone with first-hand experience of psychoanalytic practice, the latter is unrecognisable in the way it emerges as the book proceeds. When he is likening it to prostitution, warning readers never to trust anything that analysts say, comparing it to Marxism (it used to be National Socialism), or describing those who use it with difficult clients as simply requiring to feel powerful, then he really loses credibility. He is careful to say that psychoanalysis has never been fully accepted by academics or the scientific community, and suggests that it is only a minority of the older generation of American and British psychiatrists who remain committed and that these will be eroded as a newer, fresher generation take their place. I don't see much scientific rigour in putting that picture forward, although it certainly could take in people who are not familiar with the amount of training in psychodynamic psychotherapy, use of psychoanalytic models, and treatment procedures going on in British psychiatry and allied disciplines in the 1980s. I would suggest that in this country one can see and welcome a diminuation in the use of psychoanalytic theory as a popular quasi-philosophical 'catch all', accompanied by an increase, albeit as yet insufficient, in the critical evaluation of psychoanalysis as a clinical method.

I have no doubt that the book will be music to the ears of those who want to see psychoanalysis dead and buried, but for those working alongside professional colleagues of all disciplines actually in the field of patient care what has really declined is the interest in this kind of 'bee-in-the-bonnet' approach to scientific disagreements. This is particularly true of the younger generation of clinical behaviourists who are refreshingly open-minded and keenly critical. They are no more ready to take Eysenck for granted than they are Freud, however loudly they may buzz.

P. L. G. GALLWEY, *Consultant Forensic Psychiatrist, St George's Hospital, London*

The Sociopathic Personality. By BENJAMIN B. WOLMAN. New York: Brunner/Mazel. 1987. 202 pp. $25.00.

This is a rather muddled book which attempts an overview of all kinds of deviant behaviour in comparison with or in relation to sociopathic (which the author equates with psychopathic) behaviour. Thus brief paragraphs on alcoholism, drug addiction, the moral crisis, criminal violence, dictators and fanatics, etc. all appear under a series of sub-headings. Sometimes the subject matter is repeated in another part of the book.

Many paragraphs start in the same way: "Not all alcoholics are sociopaths and not all sociopaths are alcoholics but...", "Not all sociopaths are violent and not all violent people are sociopaths...", "Not all anti-social and self-righteous individuals are sociopaths but...". There are sweeping statements which research does not uphold, for example: "They plan to commit suicide but they never do it" and "All sociopaths I have seen were hypochondriacs".

The style is anecdotal in the main, mixed up with reviews of some of the established literature and references to more obscure papers. The reviews, however, are presented too briefly to impart the full message, and are wholly uncritical, so that unless one already knows the literature it is difficult to sort the worthwhile from the worthless.

The author wanders frequently into personal statements about the political arena, in particular the persecutions of the Nazi regime ("The Nazis were aggressive-sadistic psychopaths"). Nixon and Khomeini are both mentioned, as are terrorist gangs, the decline in moral standards, and the need to give young people today more purpose in life.

The only two references I tried to follow up were incorrectly indexed, and the subject index is rather sparse.

It is, therefore, a sadly disappointing book. The author clearly has had long personal experience of sociopathic patients, but the material is presented in such an undisciplined and idiosyncratic way as to detract from or obscure its potential for contributing to our knowledge of this subject.

J. STUART WHITELEY, *Consultant Psychiatrist and Medical Director, Henderson Hospital, Surrey*

Sexual Aversion, Sexual Phobias and Panic Disorders. By HELEN S. KAPLAN and DONALD F. KLEIN. 1987. 166 pp. $25.00.

Sexual avoidance disorders may form a spectrum from sexual aversion, through sexual phobia, to panic. Despite the authors' distinction, their book might strike therapists on this side of the Atlantic either as repetition of what is known by most sex therapists or, in its plea for the use of psychotropics, unproven. Kaplan offers guidance in the assessment and management of sexual avoidance disorders, with the management emphasising, as is customary now, psychodynamic, cognitive, and behavioural aspects. The 'new' aspect they emphasise is the additional use of tricyclic antidepressants in the treatment of the panic disorders. No objective data is offered in support, and the notion that psychotropic medication is likely to be helpful with these problems may bring a wry smile to the face of many sex therapists.

Clinical case descriptions are supplied, but the whole book gives the impression of being composed too rapidly, so that it is poorly thought out both conceptually and in presentation. Only the authors' reputation will ensure that departments buy this book.

SIDNEY CROWN, *Consultant Psychiatrist, The London Hospital*

A Decade of Progress in Primary Prevention. Edited by MARK KESSLER and STEPHEN E. GOLDSTON. Hanover: University Press of New England. 1986. 424 pp. £18 (pb), £28 (hb).

The contents of this book were initially presented as papers at the Vermont conference on the primary prevention of psychopathology, held in 1984. This marked a decade since the first conference, and provided an opportunity to review and reassess the past and look to and give direction for the future.

The publication is divided into five parts. The first is a review by Professor E. L. Cowen of Rochester University, which looks at recent accomplishments in the field of prevention and star-gazes in regard to future developments. He supports both person-centred strategies (i.e. development of interventions enhancing people's ability to adapt and deal with stress) and systems-level strategies (which diminish stress sources and develop people's opportunities).

The second part looks at research, including the fields of psychiatric genetics, social stress, risk factors for schizophrenia and other psychological disorders, child psychiatric development and developmental epidemiology studies (which provide the basis for specific preventive interventions). There is also a review of the literature on infant mental health. It is now possible to identify and deal with at-risk behaviour in infancy, although more work needs to be directed at the most effective early intervention.

The third part consists of reports of results from prevention projects that have been undertaken and further ideas for developing more. Work is presented focusing on the element of constituent validation, i.e. ensuring that the programme makes sense to the people who are going to use and benefit from it, which is so necessary for the activities to continue after the researchers leave.

The next chapter in this section is by Professor Caplan, concerning crisis intervention and support services promotion. He discusses a model of primary prevention and illustrates case work with adults and children facing the stress of surgery or death. Other topics in this section include public education and communicating information, self-help organisations, and education involving professionals for prevention.

The fourth section deals with the politics of primary prevention, with a resumé of the supporters and adversaries in the American scene. The state-backed programmes, the national prevention policy, and the Federal viewpoint with the role of the National Institute of Mental Health are also outlined.

The fifth and final part is a summary attempting to bring together the varying themes and aspirations for the future.

The multiple prospectives of primary prevention of psychopathology presented in this book as a result of papers delivered at a conference have been packaged in a style of variable appeal. The keen observer of this speciality will find some aspects of this book of some interest, if he or she is patient enough to overcome the rather lengthy and verbose sections.

SIMON M. WISEMAN, *Clinical Lecturer, Whittington Hospital, London.*

The Psychology of Pain (2nd ed.). Edited by RICHARD A. STERNBACH. New York: Raven Press. 1986. 255 pp. $34.00.

Sternbach is a pioneer in the assessment and treatment of pain maintained by psychological mechanisms. The first edition of *The Psychology of Pain* (1978) has been widely read. In the second edition, with the same authors, advances can be seen. In addition to psychology there are chapters on neurophysiology, neurochemical processes, hypnosis, and psychiatric aspects of pain. Authors include Craig, Chapman, Fordyce, Melzack, Merskey, and Pilowsky, as well as Sternbach.

This book is recommended for all those working with chronic pain who wish to understand more about the psychological, neurophysiological, and psychiatric mechanisms involved. The layout and printing are better than the first edition and the book is good value for money.

STEPHEN P. TYRER, *Consultant Psychiatrist, Royal Victoria Infirmary, Newcastle upon Tyne.*

Countertransference. Edited by HERBERT S. STREAN. New York: The Haworth Press. 1987. 131 pp. $22.95.

Did Freud wear blue jeans, snap his fingers, and chew gum? If so, perhaps he really did issue the "famous" injunction to psychoanalysts which Reuben Fine tells us about: "Keep cool, don't let yourself get involved".

Current issues in Psychoanalytic Practice, Volume 2, Numbers 3 and 4 have been put between hard covers. The book consists of a collection of papers of dubious quality and little originality. It is probably of some use to students of psychotherapy who wish to familiarise themselves with the clinical issues and has bibliographic value for those preparing review articles.

STEPHEN WILSON, *Consultant Psychiatrist/Psychotherapist, Littlemore Hospital, Oxford*

Psychiatric Polarities: Methodology and Practice. By PHILLIP R. SLAVNEY and PAUL R. MCHUGH. Baltimore: The John Hopkins University Press. 1987. 136 pp. £13.75.

In this book the authors consider certain theoretical issues in psychiatry, following their previous work *The Perspectives of Psychiatry,* published in 1983, which examined the basic types of approach characteristic of psychiatry. The issues considered in the present book concern, as the title suggests, dichotomies in psychiatry. The basic dichotomy, the authors suggest, is that between the mind and the brain. The former connotes subjective experience, inner realities, meaning, and so forth; the latter is objectively observable, subject-matter of the sciences, operating (one supposes) according to causal principles. The polarity between these two kinds of phenomena, the unassailable legitimacy of each, means that psychiatry inevitably comprises an ambiguity. It will always have to learn, for example, both from the natural sciences and from the humanities. This basic polarity gives rise to others, which are discussed in turn. These include the contrast between Hebraic and Hellenic conceptions of man (roughly, emphasis on the individual compared with on generalities) and aspects of the physician-patient relationship, e.g. whether it should be 'contractual' or otherwise, 'paternalistic' or otherwise, etc.

Arguably the authors have too simple a view of the mind/brain dichotomy, and they stretch the sense in which other polarities presuppose it. But in fact these weaknesses, if such they be, detract little from the book, for what it achieves is clear presentation of some major issues in psychiatry, which do indeed often have the form of either/or dichotomies. The authors bring out what lies behind each side of the conflict, and show that extreme general claims are less useful than detailed consideration of particular cases or kinds of case. The book is an excellent introduction to the issues and probably an excellent antidote to (premature) dogmatism. It is especially recommended to the beginner.

DEREK BOLTON, *Senior Lecturer, Institute of Psychiatry, London*

Hysteria and Related Mental Disorders: An approach to Psychological Medicine (2nd edn). By WILFRED D. ABSE. Bristol: Wright. 1987. 466 pp. £70.00.

The first edition of this book was based on the wartime experience of Abse as a military psychiatrist in the UK and in India. The present edition represents a considerable revision which, according to the author, explores the nature of disturbances of consciousness, ego, and psychology and its increasing forensic relevance. Linguistic development, dissociation, and the recent notions of splitting are all examined. The text is nearly twice as long as that of the first edition. The price in 1966, for comparison, was 42 shillings.

Abse writes clearly. The material he presents is analyzed partly in traditional terms relating to symptoms which are accepted as hysterical, and partly in terms of current psychoanalytic thought. The original Freudian ideas are also described in considerable detail. A large number of clinical histories are presented, and Abse clearly has a good deal of skill in interviewing patients with profound difficulties and in improving their relationships and adjustment. There are several drawbacks, however. Important material which should figure in any serious discussion of hysteria is neglected. For example, the work of Slater, Trimble, and Roy does not appear, and hypnosis is considered without reference to Barber. The systematic data, which shows that there is a relationship between hysteria and sexual difficulty in some cases of the former, but not in all, is ignored, and problems of potency are assumed automatically to be hysterical when due to a psychological cause. Epilepsy is treated as a manifestation of a primary process, and protracted vaginismus is said, without evidence, to be connected with repressed, sadistic impulses such as revenge upon the penis and the man. Phobias are treated in one place as hysteria (page 107). Dissociation in different states, such as hysteria and schizophrenia, is taken to be the same, which is not known. The Ganser and Munchausen syndromes are described inadequately, even omitting some of the most helpful psychoanalytic views on the latter, and the syndrome of Gilles de la Tourette is placidly accepted as a psychological disorder, despite mention of the use of haloperidol. The dynamic interpretation of cases, in its most far-reaching form, is presented in an assured fashion without any sort of meaningful proof. Complex and peculiar hypotheses are maintained at a great remove from factual data.

Altogether, the book relies on authority more than proof and does not present a balanced account.

HAROLD MERSKEY, *Director of Education and Research, London Psychiatric Hospital, London, Canada*

Affective Disorders in the Elderly. Edited by ELAINE MURPHY. Edinburgh: Churchill Livingston. 1986. 235 pp. £28.00.

This book is concerned with arguably the most important group of mental disorders to afflict the elderly, for while affective illnesses are amenable to treatment, too often they relapse and generate states of chronic invalidity.

Murphy has assembled 16 authors who between them contribute 14 chapters. Three are devoted to therapies covering psychological, physical, and drug treatments. That on psychological approaches is informative, well balanced, and extremely helpful; that on physical treatment is competent; that on drugs tells us little that is new and often assumes that simple therapies are likely to succeed in the majority of cases.

Chapters on biological and social factors in causation are each well written, but one on the relationship between physical illness and depression tends to cloud rather than clarify issues, with close inspection of the trees obscuring the forest of evidence that if you want to stay happy in old age, you should avoid major physical ill-health.

A chapter on course and outcome is presented with customary clarity of thought by Felix Post, who reviews recent evidence as well as his own definitive studies. A chapter on suicide gets my vote (and that of my Senior Registrar) for being "best contribution by any author" for not only assembling interesting facts, but drawing attention to aspects of suicide prevention.

The affective disorders of mania and the anxiety states are allocated a chapter each. The first is a model of information and coherent presentation, the second largely 'waffle' but with some helpful hints on treatment. Sketchy reviews of Freud's changing theories as to the causation of the psychoneuroses are not needed in this setting.

The distribution of affective disorder is well presented by Swartz & Blazer and the editor's obligatory 'concept' chapter is happily brief and to the point. Finally, there is a solidly competent chapter on characteristics (clinical features) and a rather better one on managing the depressed elderly person at home, addressed primarily to the general practitioner.

I started this book with a feeling of keen anticipation, and put it down feeling rather disappointed. As there are many excellent contributions therein, some explanation is necessary. There is far too little emphasis on the management of the difficult and frequently relapsing case, and I would have preferred a chapter devoted entirely to this topic. Again, some contributors padded out their offerings with data more appropriate to general psychiatry, and generated the suspicion that they were lacking in specialist knowledge. These deficiencies spoilt an otherwise good book.

GARRY BLESSED, *Consultant in the Psychiatry of Old Age, Newcastle General Hospital, Newcastle-upon-Tyne*

Psychological Trauma. By BESSEL A. VAN DER KOLK. Washington: American Psychiatric Press. 1987. 237 pp. $25.00.

The book aims to explore the human response to traumatic events – those that "overwhelm both psychological and biological coping mechanisms." The intention is to present a "general theory of the human trauma response". The author is Director of the Trauma Centre, Harvard Medical School. He argues that psychological damage resulting from uncontrollable terrifying life events has been relatively neglected in the past 70 years but that interest has revived following the Vietnam War, so that post-traumatic stress disorder (PTSD) has appeared in DSM–III. His approach is construed as psychobiological.

Features of PTSD are described and related theories briefly reviewed. The review also encompasses the experiencing of trauma by children, the Chowchilla kidnapping, and physical and sexual abuse.

The essence of the trauma response is seen to be the severance of affiliative bonds. The author's ideas are developed in relation to attachment theory and research into biological correlates of affiliative behaviour and the disruption of attachment. PTSD is argued to be associated with the loss of the ability to modulate arousal: traumatic events induce a biological vulnerability linked to endogenous opioid systems.

The scope of the book is evidenced by chapters on the effect of abuse on children's thought, trauma in the family, and on group, individual, and stress management orientations to treatment.

A book which draws attention to PTSD is welcome, but the theorising often seems loose and undiscriminating and the review of related research uncritical. European research related to the two World Wars is neglected. The approaches to treatment described are interesting and eclectic. It must be hoped that a more usefully articulated theory will evolve in due course.

ANTONY D. COX, *Professor of Child & Adolescent Psychiatry, Royal Liverpool Children's Hospitals*

Antimanics, Anticonvulsants and Other Drugs in Psychiatry. Edited by GRAHAM D. BURROWS, TREVOR R. NORMAN and BRIAN DAVIES. Amsterdam: Elsevier. 1987. 451 pp. US $133.35, Dfl. 300.00.

This is the last of a series of four handbooks, and contains a useful mixture in which patients and the clinical aspects of their management are to the forefront. Ample space is given to novel information, reviews, summaries, and conclusions which can be appreciated irrespective of the preceding volumes. The first four chapters consider the current status of lithium, carbamazepine, and antipsychotic drugs in the treatment of hypomania and mania. The specific advice given for clinical situations is particularly helpful. Intermediate chapters give a detailed appraisal of anticonvulsants, including their use in the mentally handicapped and in pregnancy. Pseudoseizures, and the effect of anticonvulsants on affect and cognition, are well reviewed. Anticholinergic drugs, beta-blockers in schizophrenia and in anxiety, and the effects of cortico-steroids and of oral contraceptives are all discussed in detail.

The latter half of the book also has a clinical leaning, dealing with tic disorders, Huntington's chorea, and Parkinsonism, but the focus returns to drugs used in phobic disorders, in alcoholism, and in the treatment of dementia. The role of analgesics and psychotropic drugs in pain management is clearly outlined. The concluding chapters cover sexual dysfunction, psychiatric reactions to non-psychotropic drugs, and the issue of compliance.

The style and format make comfortable reading and, matching the content, the print and finish of the book reach the highest standards. It would be difficult to find a better guide to the therapeutic areas described here. The book provides liberally the sort of information essential for broad clinical practice and needed frequently in liaison work in a District General Hospital.

JOHN POLLITT, *Consultant Physician in Psychological Medicine, Hayes Grove Priory Hospital, Kent*

Stress Response Syndromes (2nd edn). By MARDI JON HOROWITZ, Northvale, NJ: Jason Aronson. 1986. 358 pp. $35.00.

This is an important book on an important subject. It fully deserves its second edition within 8 years of publication. Post-traumatic stress disorders are, sadly, increasingly relevant in psychiatry as society becomes both more violent and more caring. This book is at one level a primer on psychotherapeutic help for victims of trauma – by rape, sudden bereavement, or injury. It describes with great sensitivity and detail the feelings of fear, rage, shame, guilt, and numbness associated with trauma and focuses in particular on 'intrusive images' of violence that can haunt victims for years afterwards. It shows how, if these feelings are to be overcome, the meaning of the trauma in the patient's biography has to be understood; how, for example, a man with violent fantasies towards women will be doubly traumatised when a female passenger in his car is killed; or a woman who deals with low self-esteem by flirting may be plagued with shame following rape; or how a teenage girl will be devastated if her mother dies of a heart attack just after they have had a blazing row. The twin dangers faced by trauma victims are described: either emotional shut-down or being flooded with unmanageable feelings. It shows how, in the safety of a psychotherapy session, psychological controls can be relaxed enough for painful feelings to be contemplated and for psychological reintegration to occur. It demonstrates the great tact that is needed to decide when to offer support and sympathy and when to probe, and how different personality styles demand different approaches.

This book has many other important levels. Horowitz is that rare phenomenon: someone who is equally respected as a psychoanalyst and a researcher. As well as being a clinical treasure chest, the book also contains an elegant outcome study showing the efficacy of twelve-session brief psychoanalytic therapy in stress disorders, a scholarly review of Freudian theory on trauma, and an important integrated psychoanalytic/cognitive theoretical schema. Despite occasional verbosity it is very readable. It is an exemplary book, which all who are interested in applied psychotherapy should read.

JEREMY HOLMES, *Consultant Psychiatrist, North Devon District Hospital, Devon*

Key cases in Psychotherapy. Edited by WINDY DRYDEN. Beckenham: Croom Helm. 1987. 269 pp. £22.50.

It is difficult to do justice to this book in a short review. The editor's aim has been to show how therapists working with the benefits and limitations of their different schools of thought and practice grapple with patients who challenge their underlying assumptions and become instrumental in changing therapists' theory and practice.

Wessler, Lazarus, Prochaska and Ellis's experiences have been well integrated, and stimulate considerable interest. Thorne and Rowan arouse the anxieties they themselves felt over the risks taken when therapy moved into unchartered waters. Morrison, Aveline, Wile, Butt and Bannister each demonstrate their own significant lessons. Some contributions, however, may confuse the novice or irritate the experienced therapist. Prospective readers should taste a little of each contribution before making a final decision to buy.

BRIAN LAKE, *Honorary Lecturer, University of Leeds*

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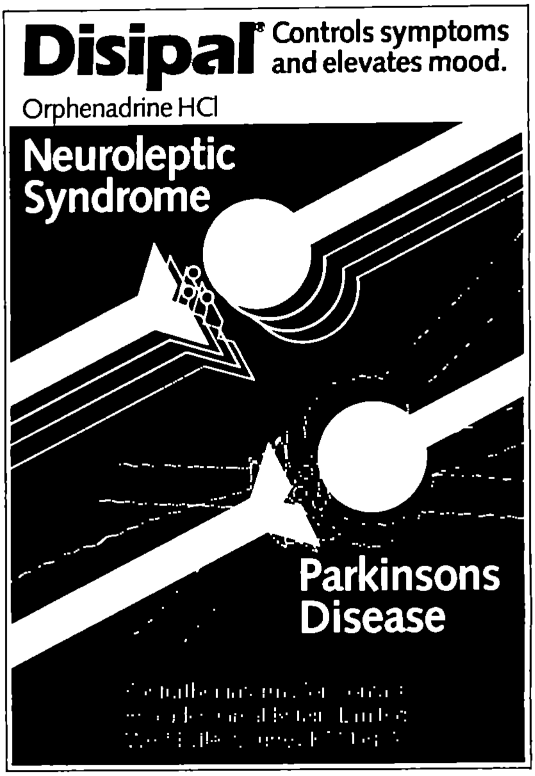

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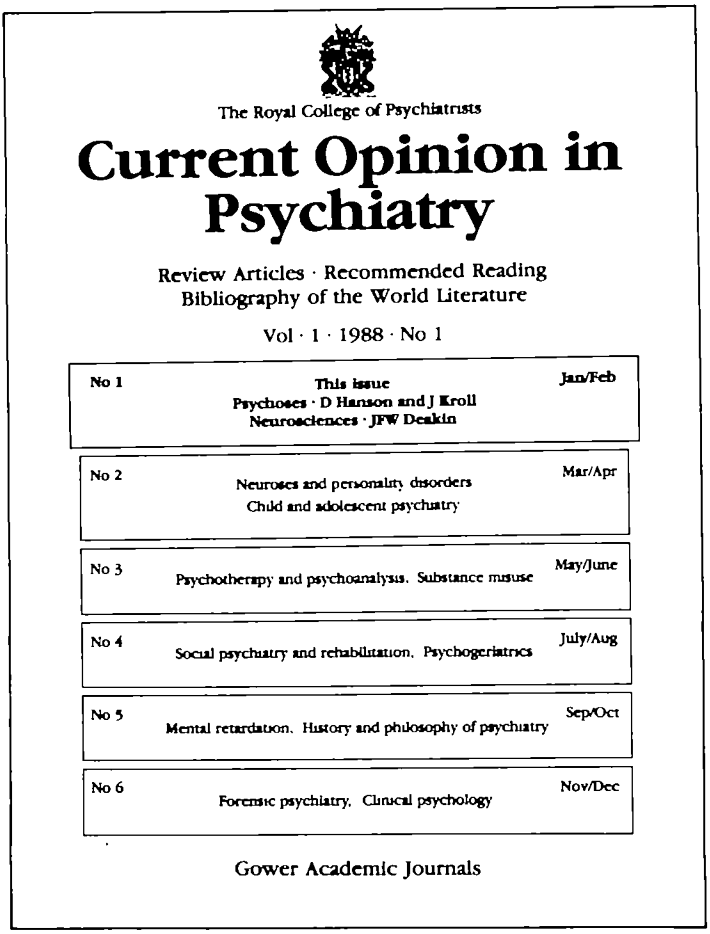

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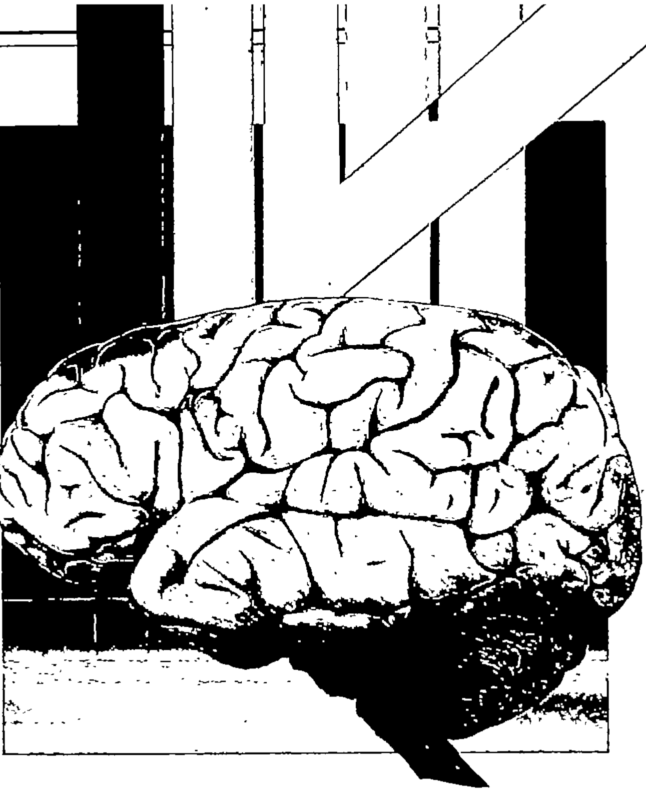

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Annotation

The Place of Benzodiazepines in Psychiatric Practice

PETER TYRER and SIOBHAN MURPHY

For the past 20 years, benzodiazepines have been the most commonly prescribed psychotropic drugs. It is now difficult to imagine the excitement produced by this new class of compounds. Existing anti-anxiety drugs, mainly the barbiturates, were known to be dangerous in overdosage, tend to cause addiction, and have many side-effects. Previous compounds, including opium, alcohol, chloral, and bromides, were similarly burdened. The benzodiazepine era began almost 30 years ago, in style. "Four hours after being given chlordiazepoxide on New Year's day 1958, one of 12 chronically anxious but therapeutically recalcitrant patients previously studied by Tobin and N. D. C. Lewis telephoned that for the first time in many years he was totally free from symptoms!" (Hordern, 1968). The early studies were all enthusiastic and confirmed the therapeutic potential of the drug (Tobin *et al*, 1960; Jenner *et al*, 1961). In the 'tranquilliser decade' of the 1970s, prescriptions of benzodiazepines increased at a rate that was perceived as alarming, 'the relentless march of the psychotropic drug juggernaut' (Trethowan, 1975). This concern was related more to the inappropriate use of these drugs for treating personal problems than to the demonstration of dangers with these compounds. The dangers, however, appeared to be remarkably few; the drugs were safe in overdose, had greater efficacy than the barbiturates (Lader *et al*, 1974), and had virtually no unwanted effects, apart from sedation when given in excessive dosage. It was therefore hardly surprising that they proved so popular with clinicians. As Priest (1980) commented when benzodiazepine prescription was at its peak: "it is a tremendous boon to the medical profession to have active weapons in the fight against misery that are not only effective but are relatively safe when abused by despairing and desperate patients".

Since this time a dark shadow has been cast over the benzodiazepines by the spectre of dependence. While it had been realised from earlier studies that dependence could be a problem (Hollister *et al*, 1961), this was thought to apply only when doses well above the therapeutic range had been used. Even with high dosage, doubt was cast upon the role of benzodiazepines in producing dependence, as in so many published reports alcohol and other addictive drugs were taken as well as benzodiazepines (Marks, 1978). As by far the larger proportion of drugs taken was for therapeutic purposes, on prescription, and, by and large, clinicians were perceived to prescribe these drugs responsibly (Mellinger *et al*, 1978), there did not seem to be much need for concern. However, the demonstration that regular treatment with benzodiazepines in therapeutic dosage could produce significant withdrawal problems when the drug was stopped (Pevnick *et al*, 1978; Tyrer, 1980; Winokur *et al*, 1980; Petursson & Lader, 1981; Tyrer *et al*, 1981, 1983) led to a change in attitudes. The wonder drugs of yesterday were now addictive and 'even worse than heroin' according to many accounts in the media. Doctors have been reviled for prescribing them and patients are now being encouraged to sue doctors for making them dependent.

The medical profession has reacted with some confusion. Some doctors (e.g. Snaith, 1984) feel that benzodiazepines no longer have a place in clinical practice, whereas others (e.g. Oswald, 1986) have gone the other way and suggest that benzodiazepine hypnotics should be available without a prescription as they are so much safer than the alternatives, particularly alcohol. It is therefore difficult for the average psychiatrist in practice to know when to prescribe benzodiazepines and how to treat the many patients who have taken benzodiazepines regularly before presenting to the psychiatric services. It is also difficult to know which benzodiazepine to prescribe, because of conflicting accounts of their advantages and dangers.

Indications for prescription

Although benzodiazepines have an important place in the treatment of epilepsy and disorders of muscle tone, most psychiatrists use them for the treatment of anxiety and insomnia. In the past, the presentation of either of these complaints at the general practitioner's surgery was often followed by the reflex prescription of a benzodiazepine, nicely summarised in Marinker's epigram "you are a case of diazepam, you had better have some anxiety" (Marinker, 1973). Nowadays all practitioners are much more circumspect about prescribing a benzodiazepine. However, in anxiety occurring at a time of major stress, now formally diagnosed as acute stress or adjustment reactions in both ICD-9 (World Health Organization, 1978) and DSM-III (American Psychiatric Association, 1980), it may be appropriate to prescribe a benzodiazepine if distress is severe and prescription is likely to be short-term.

Even for this indication, care must be taken to prescribe benzodiazepines judiciously; they produce a degree of anterograde amnesia and this could provoke the mental mechanism of denial in subjects who need to come to terms with the stressful event to achieve a satisfactory psychological adjustment. In instances such as bereavement and other major personal loss, prescription needs to be for as short a time as possible and then only to relieve the worst of the symptoms. In other forms of anxiety, benzodiazepines are much less appropriate. This includes the unfocused anxiety of anxiety neurosis and generalised anxiety disorder (in DSM-III), which is one of the commonest psychiatric disorders presenting to the general practitioner. It has been shown that relatively simple psychological treatments such as reassurance and explanation can replace benzodiazepines successfully in primary care (Catalan & Gath, 1985) and avoid the need for any psychotropic drugs. If drug treatment is needed for persistent or disabling anxiety symptoms that are not part of an adjustment or stress reaction, other drugs can be used. There is now good evidence that antidepressants are more effective than benzodiazepines in this group (Johnstone *et al*, 1980; Kahn *et al*, 1986). For many years it has been known that anxiety presenting in the form of acute panic attacks also responds better to antidepressants (Klein, 1964), and the same has also been shown with agoraphobia (McNair & Kahn, 1981).

The use of benzodiazepines in the treatment of insomnia has changed much less than that for the treatment of anxiety. It is still common practice in many psychiatric hospitals for patients to be prescribed a benzodiazepine hypnotic almost as a matter of routine. This may be appropriate if it is only given for a short time or on an occasional intermittent basis, but if it becomes regular, it can lead to continued prescription because of dependence. There is now abundant evidence that 'rebound insomnia' (Kales *et al*, 1978) occurs when a benzodiazepine hypnotic is taken for a few weeks only and then suddenly withdrawn (Kales *et al*, 1983*a*). As other problems with benzodiazepines, noticeably impairment of cognitive and psychomotor function, are less important for a hypnotic than an anti-anxiety drug, there is a belief developing that it is appropriate to give a benzodiazepine long-term for the treatment of insomnia but not for the treatment of anxiety. This is a dangerous assumption, not least because the use of benzodiazepines in the treatment of anxiety and insomnia is interchangeable, and their marketing for separate purposes is a commercial rather than a pharmacological decision. There is no reason why psychological treatments should not be used similarly for insomnia as for anxiety, and much more use should be made of intermittent drug therapy.

The other major use for benzodiazepines is in the treatment of alcohol-withdrawal reactions (Kaim *et al*, 1969). This practice has also come under closer scrutiny since benzodiazepines have been shown to produce dependence. Clearly, if one form of dependence was merely being replaced by another, the use of benzodiazepines for detoxification purposes could not be recommended. In practice, far greater attention is paid to reducing doses of benzodiazepines (and other agents) used for detoxification, so that drug treatment is withdrawn between 7 and 10 days (Thorley, 1982). This also makes good clinical sense, as alcohol-withdrawal symptoms have a short time course.

Duration of treatment

Benzodiazepines exert their therapeutic action within 60 min and reach a peak of efficacy within 2 h of administration. Tolerance to their effects develops quickly (Aranko *et al*, 1983, 1985), but is not nearly so marked as with the barbiturates and other older anti-anxiety drugs. The ideal form of treatment is intermittent flexible dosage, in which medication is taken occasionally at a time of increased symptoms but never on a regular basis (Tyrer, 1982). This may avoid the development of dependence, although hard evidence for this is lacking.

There is now good evidence that a significant proportion of patients taking long-term benzodiazepines in regular dosage develop pharmacological dependence. This is mainly manifest in the form of a withdrawal syndrome which includes the features of pre-existing anxiety together with panic, perceptual disturbances such as hypersensitivity to all sensory modalities, and depersonalisation, tinnitus, paraesthesiae, muscle pain and weakness, and, rarely, epileptic fits and schizophreniform psychoses, including first-rank symptoms of schizophrenia (Petursson & Lader, 1981; Tyrer *et al*, 1981, 1983; Busto *et al*, 1986; Roberts & Vass, 1986). This has led to greater caution in prescribing benzodiazepines for long periods, but the implied assumption from these studies is that shorter treatment for a few weeks is safe from the risk of dependence. This assumption seems to be misplaced, as there is now good evidence that regular treatment with a benzodiazepine for periods of 4–6 weeks is followed by a significant incidence of withdrawal problems if the drug is stopped suddenly after this time (Fontaine *et al*, 1984; Murphy *et al*, 1984; Power *et al*, 1985). The idea of giving a short sharp course of benzodiazepines followed by sudden withdrawal is therefore no longer considered to be an appropriate use of these compounds. If intermittent flexible dosage is not possible and regular treatment is preferred, it is wise to confine treatment to a few weeks and then to reduce the drug gradually after this period in the same way recommended for long-term use of benzodiazepines (Tyrer *et al*, 1983; Higgitt *et al*, 1985).

The psychiatrist is more frequently faced with a patient who has taken benzodiazepines for many months or years, for whom he has to make a decision whether to continue treatment. The first thing to remember is that a substantial proportion of such long-term users do not have significant problems when reducing and stopping their benzodiazepines. Studies of unselected referrals suggest that only 35–45% of long-term users have withdrawal symptoms, and over 50% can stop their medication without withdrawal problems (Tyrer *et al*, 1981, 1983). If there is no good reason for continuing prescription of a benzodiazepine, then a straightforward withdrawal regime can be adopted, determined either by the psychiatrist or by the patient to carry out in his or her own time (Tyrer, 1986).

For those who have had apparent withdrawal symptoms whenever they have reduced their benzodiazepines, the situation becomes more complicated. A range of treatments is available, including withdrawal under cover of antidepressants (Tyrer, 1985), concomitant treatment with beta-blocking drugs such as propranolol (Tyrer *et al*, 1981), and psychological aids, including relaxation training and techniques based on cognitive therapy (Higgitt *et al*, 1985; Tyrer *et al*, 1985; Sanchez-Craig *et al*, 1986). However, success depends a great deal on the motivation of the patient, and this is often lacking. The clinician is frequently placed in the position of reviewing the treatment of a patient who does not appear to be helped by benzodiazepines but who cannot seem to cope without them. The patient naturally asks whether any permanent harm can come from continuing benzodiazepines in the long term. This question cannot be answered definitively, but from present evidence there is no unequivocal permanent handicap caused by benzodiazepines in short or long-term dosage. It is worth bearing in mind that the presence of benzodiazepine receptors in the central nervous system implies that there is a naturally occurring substance (an endogenous ligand) that is concerned with the physiological control of anxiety. If this exists, it must be pharmacologically similar to the benzodiazepines, so the notion of benzodiazepines being exogenous poisons is difficult to sustain. However, the existence of endogenous opioid receptors that are binding sites for exogenous opiates shows the dangers of the assumption that what is natural is therefore safe. Currently there are exciting developments that suggest we are close to identifying the exact nature of the endogenous ligand (Stephenson, 1987); if it is discovered, it will have great implications for the development of newer drugs for treating anxiety.

Although benzodiazepines impair memory and other higher aspects of mental function (Lader & Petursson, 1983), this does not always have much impact in discouraging patients from continued use. In the present state of knowledge it is too punitive to withdraw benzodiazepines from long-term users who do not want to stop treatment, and a withdrawal programme forced on an uncooperative patient is an exercise in futility. The usual outcome of this approach in general practice is that the patient leaves the doctor's list and finds another practitioner who is more willing to prescribe benzodiazepines. However, the practice of giving long-term repeat prescriptions of benzodiazepines is not satisfactory. The patient's drug use needs to be regularly monitored, and there should be encouragement to attempt withdrawal whenever the time appears to be propitious.

Which benzodiazepine?

Even if it is accepted that benzodiazepines have a place in psychiatry, there is argument over which preparation is most appropriate for treatment. This task has been made somewhat easier recently by the introduction of the limited list in the United Kingdom. For years it has been realised that there are too many benzodiazepines on the market (Tyrer, 1974; Drugs and Therapeutics Bulletin, 1978). The introduction of the limited list was unsatisfactory in several respects, but there is no evidence that it has been a major loss to treatment. The major difficulty with the benzodiazepines is that some patients have had difficulty in stopping their preferred drug and replacing it by one on the limited list (Drugs and Therapeutics Bulletin, 1987), which suggests dependence on the preferred drug and absence of cross-tolerance when changing to another benzodiazepine.

The most common grouping of benzodiazepines is based on their elimination half-lives, which is roughly correlated with their duration of effect. Anxiety is often markedly episodic (e.g. in phobias) and, even if persistent, varies greatly during the day, and insomnia needs only to be relieved for a maximum of 8 h. It is therefore natural that both pharmaceutical companies and clinicians have tried to develop and promote the use of benzodiazepines with relatively short elimination half-lives up to a maximum of 8 h. Unfortunately, it seems likely that benzodiazepines of short half-life are more prone to cause pharmacological dependence than those with long half-lives (Tyrer *et al*, 1981; Owen & Tyrer, 1983; Kales *et al*, 1983*b*; Griffiths *et al*, 1985).

There are also suggestions that benzodiazepines of higher potency are more prone to dependence than others, but this makes interpretation difficult, as many of the short-acting benzodiazepines are also potent ones, and the average daily dose is less than 5 mg (triazolam, lormetazepam, loprazolam, alprazolam, lorazepam). Unfortunately, there have been insufficient comparative studies to enable the clinician to construct a 'league table' of dependence risks. There are many anecdotal reports (Drugs and Therapeutics Bulletin, 1985) and at least one comparative study (Tyrer *et al*, 1981) suggesting that lorazepam is at particular risk in producing dependence. It is difficult to know whether lorazepam has been singled out unfairly purely because of wide publicity in the media or whether it indeed has a higher potential for dependence. It is a fairly potent benzodiazepine of intermediate half-life (about 14 h) and has been associated with many more reports of dependence than its fellow benzodiazepine of equivalent half-life, oxazepam (Hanna, 1972; Griffiths *et al*, 1984). In the United States, alprazolam, a benzodiazepine of similar potency and half-life, is also getting a reputation for greater risk of dependence than other benzodiazepines. Despite the concern over lorazepam in regular oral dosage, it does have definite advantages in intramuscular dosage over diazepam, as it is absorbed more quickly and excreted more rapidly. It can be of value in treatment of disturbed patients, particularly those with mania (Bick & Hannah, 1986; Jobling & Stein, 1986).

Recommendations

If a psychiatrist feels that drug treatment for an anxious disorder is necessary, benzodiazepines will still figure prominently in the list of options. Wherever possible, their long-term use is to be avoided, because of the risk of pharmacological dependence. They are best used for periods of no more than 2 weeks in regular dosage, or in intermittant flexible dosage. Special care has to be taken when prescribing potent benzodiazepines of short half-life, particularly triazolam and lorazepam, because of growing evidence that they are more likely to lead to the development of dependence.

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Review Article

Borderline Personality
A Review of Recent Research

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Borderline personality has been, for many years, a discredited diagnostic concept. In 1979 a review of the literature concluded that its validity status was very uncertain. The authors have reviewed research conducted since then and discuss it in terms of the Robins & Guze (1970) criteria. In spite of existing unclear issues, the balance is tipping in favour of the validity of borderline personality, as diagnosed with new research criteria. This development is taking place in the context of a growing interest in the area of personality disorders.

A useful starting point in this discussion of the validity of borderline personality is the review of Liebowitz (1979), which stated that, "When the St Louis approach to diagnostic validity is used as a guideline, the conclusion reached is that available data do not weigh conclusively for or against borderline's status as an independent entity". Since this statement was made, several sets of clinical and research criteria have yielded new data which permit a critical re-evaluation of the diagnosis. These new publications will be reviewed to assess whether the statement that the data "do not weigh for or against" can still be supported. This literature is predominantly American. "Borderline Personality" does not appear in ICD-9 (World Health Organization, 1978), but the term is used by one quarter of the psychiatrists in the UK (Macaskill & Macaskill, 1981; Tarnopolsky & Berelowitz, 1984). Such cases represent about 15–25% of the personality disorder diagnoses in a local ward; they are usually recorded here as suffering from explosive or hysterical personality disorders, or from a depressive illness (Kroll *et al*, 1982). Furthermore, this work illustrates some of the complex issues confronting researchers in personality disorders.

Diagnostic systems

Three well-known diagnostic systems are relevant to this paper: DIB, Spitzer and DSM-III criteria, and Kernberg's psychoanalytic construct.

Gunderson and co-workers (Gunderson & Singer, 1975; Gunderson & Kolb, 1978; Gunderson *et al*, 1981) abstracted symptoms and interactional patterns characteristic of the borderline personality, and developed a semi-structured interview (Diagnostic Interview for Borderlines, DIB) which examines social adaptation, impulse/action patterns, affects, psychotic phenomena, and interpersonal relationships, and which excludes major psychoses. A summary score indicative of borderline personality is derived from 140+ individual questions.

Spitzer *et al* (1979), on the basis of some empirical research, partitioned a hitherto muddled field into two personality disorders: Schizotypal personalities, related to schizophrenia, and Borderline personalities, discussed here. They are chronically unstable, vulnerable individuals, with difficult relationships, poor self-control, and low sense of identity, as described in the DSM-III eight-item checklist (APA, 1980).

Kernberg (1977, 1981) differentiated neurotic, psychotic and borderline "intrapsychic organisations", which are to some extent independent of manifest symptomatology. "Borderline personality organisation" is defined by (a) absence of a stable sense of identity; (b) use of primitive defence mechanisms (splitting and projective identification); and (c) partial retention of reality testing. Kernberg's interviewing method requires both phenomenological and psychodynamic expertise.

The DIB and the DSM-III checklist are essentially phenomenological, and they overlap and also differ. The DIB is wider, including several items on psychotic-like phenomena (brief psychotic episodes, drug-related psychoses, depersonalisation/derealisation) and on social functioning (the adoption of caretaker roles, work performance, and social presentability). However, this is partially counterbalanced by the scoring method: individual DIB items may not influence the final rating if the overall score from that section is too low. The DSM-III checklist contains two items not present in the DIB, namely "identity disturbance" and "affective instability". Kernberg's system is not symptom-based but shares with the DIB the emphasis on certain psychotic features. Furthermore, some of the DIB/DSM-III items are clearly derived from psychoanalysis. For example, "identity disturbance" (DSM-III) originates in Eriksonian ideas; "caretaker roles" (DIB) describes a narcissistic identification.

Reliability and validity

There are so few 'objective' indicators of psychiatric disorder that diagnostic validity is difficult to assess. One could reduce validity to a matter of reliability (i.e. agreement between different assessors about the presence of the disorder), but a more substantial approach consists of marshalling information from a number of areas, and constructing a convincing argument for or against the existence of the disorder (Robins & Guze, 1970). Using this approach we will discuss validity under six headings: identification of a characteristic phenomenology; phenomenological independence from other psychiatric disorders; follow-up data; family studies; laboratory investigations and psychological tests; and treatment response. But first we need to examine the reliability of the diagnostic instruments.

Reliability

Reliability refers to the consistency of an assessment between raters and over time. It is best measured with the coefficient kappa ($\varkappa$), which expresses better than chance agreement ($\varkappa = 0$, chance agreement; $\varkappa = 1$, perfect agreement). In general values above 0.7 are acceptable.

Using the DIB, acceptable $\varkappa$s, in some cases above 0.8, have been demonstrated for live interviews (Kroll *et al*, 1981*a*; Frances *et al*, 1984; Hurt *et al*, 1984), and case notes (McGlashan, 1983*a*; Armelius *et al*, 1985, calculated by us); for the agreement between notes and interviews (Armelius *et al*, 1985); and for two interviews of the same patient by different clinicians at least one week apart (Cornell *et al*, 1983). Individual subscales are also reliable, with only one exception (Frances *et al*, 1984).

For the DSM-III criteria, acceptable $\varkappa$ values above 0.7 were obtained with clinical interviews (Frances *et al*, 1984); perusing case notes (McGlashan 1983*a*); and with a new structured interview ($\varkappa = 0.85$; Stangl *et al*, 1985).

Reliability of Kernberg's method has not been adequately reported (Kernberg *et al*, 1981). Bauer *et al* (1980) showed that blind assessors could differentiate borderline from psychotic "organisation" ($P = 0.024$) using interview transcripts.

In summary, the DIB is the most thoroughly tested and reliable research tool. The promising new DSM-III-based interviews have the advantage of addressing the full range of DSM-III personality disorders. It would be wise to continue using more than one set of criteria as the systems are being reassessed and modified. The reliability of Kernberg's method is not convincing, but the method addresses a different level of pathology, which is not easily measured. Other diagnostic systems which have not gained widespread acceptance include a checklist (Sheehy *et al*, 1980); the Ego Functions Inventory (Perry & Klerman (1980); and two self-report questionnaires (Conte *et al*, 1980; Hyler, in Hurt *et al*, 1984).

Validity: an application of the criteria of Robins and Guze

A unitary clinical description

"In general, the first step is to describe the clinical picture of the disorder" (Robins & Guze, 1970). Does research with the criteria reviewed identify a homogeneous group of patients?

Many studies applied both DIB and DSM-III criteria to case notes (McGlashan, 1983*a*; Pope *et al*, 1983), and interviews (Frances *et al*, 1984; Akiskal *et al*, 1985*a*). Kroll *et al* (1981*b*, 1982) and Barrash *et al* (1983) thoroughly examined 252 consecutive admissions. The DIB identified a larger number of cases than the DSM-III, and there were, naturally, some false positives and false negatives, mostly cases who met the DIB criteria and not the DSM-III. The commonest diagnosis for these discordant cases was non-borderline personality disorder. Cluster analysis improved the agreement and yielded a high sensitivity (0.83) and specificity (0.89) for the DIB against the DSM-III criteria.

In most studies there is a potential source of disparity: the DIB is a research tool, whereas the DSM-III data is obtained in routine clinical interviews. Gunderson *et al* (unpublished) addressed this using a structured DSM-III interview and identified about four-fifths of in-patients with personality disorders as borderline (DIB 84%; DSM-III 74%); sensitivity and specificity were both high, but better for the DIB against the DSM-III than the other way round.

A further improvement requires consideration of the reliability, frequency and discriminative power of each individual item or symptom. Table I shows these with data given by Frances *et al* (1984). When faced with low reliability, it is worth attempting to refine rather than discard the item in question. Kroll

TABLE I

Assessment of certain items diagnostic of borderline personality disorder[1]

Reliability (x)[2]		*Frequency, %*[3]		*Discriminative power (RR)*[4]	
		Gunderson's Diagnostic Interview for Borderlines[5]			
Impulsivity/action pattern	0.79	Social adaptation	86	Impulsivity/action pattern	3.3
Brief psychosis	0.70	Affects	84	Brief psychosis	3.1
Social adaptation	0.69	Interpersonal relationships	68		
Interpersonal relationships	0.67				
		DSM-III Borderline Personality Disorder			
Self-damaging acts	0.69	Impulsivity	89	Emptiness/boredom	4.4
Inappropriate anger	0.68	Affective instability	84	Intolerance/aloneness	3.8
Impulsivity/unpredictability	0.65	Inappropriate anger	80	Impulsivity/unpredictability	3.7
		Interpersonal relationships	77	Interpersonal relationships	3.5
		Emptiness/boredom	75		

1. This table was compiled by us from data published by Frances *et al* (1984).
2. Reliability: items with (*x*) ⩾0.65 are included.
3. Frequency: items present in at least 75% of the cases are listed.
4. Relative risk: calculated by us against non-borderline personality disorders (percentage of cases with item present divided by percentage of controls with item present); values above the median in each set are included; range for DIB was 1.2–3.2, and for DSM–III was 2.4–4.4
5. Each DIB category is a subscale comprising several items; each subscale is scored 0, absent; 1, moderate; 2, severe; only the percentage of score 2 is shown here.

et al (1981*b*) and Gunderson *et al* (unpublished) have examined the diagnostic value of items that appear in only one set.

All this research and work with other criteria (Gunderson, 1977; Sheehy *et al*, 1980; Soloff & Ulrich, 1981) has identified a characteristic borderline diagnostic core of unstable interpersonal relationships, with idealisation and denigration of others, intense unpredictable feelings, and impulsive, often self-destructive behaviour. Similarly, Maudsley psychiatrists think that the most frequent items among the borderlines were a pattern of unstable, intense interpersonal relationships and impulsiveness or unpredictability in potentially self-damaging areas (both DSM–III items); the most discriminating item, however, was brief, stress-related, psychotic episodes or regressions (a Gunderson item) (Tarnopolsky & Berelowitz, 1984). Bateman (unpublished) reported the only diagnostic study of borderline disorders conducted in the UK by a local author. He compared in-patient DIB-diagnosed borderlines with PSE-diagnosed neurotic and psychotic controls. A particularly high level of anxiety and irritability, externalised as violent destructive behaviour, anger, and hostility at interview, distinguished borderlines and neurotics. In addition, they presented depressive and non-specific psychotic features.

Kroll *et al* (1982) highlighted certain differences between British and American patients: "the British borderlines (DIB and DSM–III criteria) reported minimal drug abuse and no drug-related psychosis . . . evidenced no interest in caretaker roles; and although the majority reported derealisation and depersonalisation, so did the majority of British non-borderline patients". These differences should be further investigated.

Reported agreement for Kernberg's method with DIB and DSM–III was over 70% for in-patients, and about 60% for out-patients (Kernberg *et al*, 1981; Koenigsberg *et al*, 1983; Nelson *et al*, 1985). Some flaws limit the conclusions drawn from these studies, and further work using Kernberg's ideas should be undertaken, as his are the only criteria firmly based on a unitary psychological theory.

In summary, although some concordance is expected for sets developed within the same psychiatric culture (and Spitzer consulted with both Gunderson and Kernberg), the agreement found between DSM–III and DIB is high, considering the uncertainty about psychiatric diagnosis generally. However promising, these results say nothing about which instrument is more valid, a question to be resolved only by observing the relationship of external factors, such as prognosis, family distribution and treatment response, with the diagnostic criteria. In turn, the analyses ought to be extended to individual items in each set, a type of study only just being published (Goldberg *et al*, 1986; Soloff *et al*, 1986). So far we conclude that there is enough agreement in terms of reliability and clinical description to justify proceeding to another step of inquiry.

Phenomenological discrimination from other disorders

The question here is whether patients with an operationally diagnosed borderline personality can be distinguished from patients with other psychiatric conditions, in particular with schizophrenia, affective disorders, and other personality disorders.

Schizophrenia. Several studies with different methods have now placed the phenomenological distinction between borderline personalities and schizophrenic in-patients beyond reasonable doubt (Gunderson *et al*, 1975; Kolb & Gunderson, 1980; Soloff & Ulrich, 1981; Kroll *et al*, 1981*b*, 1982; Pope *et al*, 1983). For example, Gunderson *et al* (1975), using patients taken from the International Pilot Study of Schizophrenia, found that the borderline in-patients had significantly fewer psychotic symptoms than the schizophrenic group, with no evidence of thought disorder. The borderline group was also characterised by derealisation, a frenetic and stormy life-style, unusual and occult experiences, marked interpersonal difficulties, and suicide threats. Kroll *et al* (1981*b*) found only one DSM-III schizophrenic among 21 DIB-positive in-patients, and Pope *et al* (1983) found no DSM-III schizophrenics among 33 in-patients diagnosed as borderline according to Gunderson criteria.

However, the distinction between out-patients is less clear and less studied. Sheehy *et al* (1980), comparing borderlines (own criteria) and schizophrenics (Carpenter criteria), found that deficient management of impulses, intolerably unpleasant feelings, and idealisation/denigration of others were significantly more prevalent among the borderline patients. Pronounced failure of reality testing was more frequent among the schizophrenics, and was the best predictor of group differences. But Koenigsberg *et al* (1983) found that borderline out-patients had only non-significantly higher DIB scores than schizophrenic out-patients. These results are conflicting and also questionable. Sheehy's borderline criteria were not applied with research rigour, and Koenigsberg reported only global DIB scores for borderline patients versus a small heterogeneous control group of schizophrenic and manic-depressive patients. Therefore, while the distinction between florid forms of schizophrenia and borderline personality is well established, the distinction between milder forms of the two disorders requires further research.

Affective disorders. The coincidence of affective illness with borderline personality is much greater than statistically expected (Gunderson & Elliott, 1985; Perry, 1985). This relationship is also more complicated than the previously postulated inclusion of borderline personality under schizophrenia, and Gunderson & Elliott (1985) have listed four hypotheses to explain it. Two are that one disorder can be reduced to the other; for example, (a) drug-taking or promiscuity to relieve feelings of emptiness, dysphoria or depression, or (b) depression resulting from impulsivity and unsatisfactory relationships; the third (c) is that both disorders coexist independently in the same subjects; and the fourth (d) – their own, in the Meyerian tradition – postulates that affective symptoms or character traits arise from an interaction of influences peculiar to each individual. Research partially supports all the hypotheses, although Gunderson & Elliott's analyses lead them to accept the fourth.

In this section, evidence about the phenomenological distinction between borderline personality and a variety of affective disorders is considered. Gunderson & Kolb (1978) were able to discriminate borderline personality from neurotically depressed in-patients by the presence of drug-related psychotic experiences, anhedonia and dysphoria, interpersonal difficulties, and paranoid experiences. Sheehy *et al* (1980), with less formal methods, obtained similar results in a series of out-patients. Barrash *et al* (1983), summarising the findings of Kroll's group on 252 in-patients, found 48 with DIB-positive borderline personality and 77 patients with "affective disorders" not further specified; only three patients with affective disorder were DIB-positive. Soloff & Ulrich (1981) found that total scores, scaled section scores, and 19 individual DIB items all effectively differentiated borderline personality from RDC major (unipolar) depressives. It was also repeatedly noted that the items characteristic of each disorder are different (e.g. impulsivity *v.* affective state); and that the attendant emotions are different, the borderline personalities' 'depression' having schizoid qualities of boredom and emptiness. Borderline personalities also feel easily disappointed and let down, want to hurt themselves and are well aware of their rage.

By contrast, other studies of in-patients (Pope *et al*, 1983) and out-patients (Akiskal *et al*, 1985*a*) have found a proportion as high as 50% of major and minor affective illnesses among DIB-positive patients. It is of local interest that the British sample ($n = 47$) studied by Kroll *et al* (1982) showed seven DIB-positive cases, three with a secondary diagnosis of depressive neurosis and one with a primary diagnosis of major affective illness (DSM-III). Also, Bateman's (unpublished) pilot study in London found that among eleven in-patient DIB-borderlines, ten met the

PSE criteria for minor, and one for major, depressive disorders, although they differed in other ways (see above).

Independently of the procedural differences between the papers, our view is that this issue cannot be fully solved with single cross-sectional descriptive studies where the distinction between long-term personality traits and episodic affective symptoms is obscured; nor in a single centre, because of sample variations influencing the intake of patients to individual institutions. We will take up this discussion below with data from follow-up, family, and therapeutic studies.

Personality disorders. Several studies have failed to discriminate between *in-patients* with DIB borderline and non-borderline personality disorders (Kolb & Gunderson, 1980; Kroll *et al*, 1981*b*, 1982); Pope *et al* (1983) found that DSM-III borderline overlapped with histrionic personality disorder in women, and with antisocial in men. However, severity may be relevant, because cases could be distinguished in at least three *out-patient* samples (Sheehy *et al*, 1980; Perry & Klerman, 1980; Koenisgberg *et al*, 1983). More importantly, Barrash *et al* (1983) reanalysed with cluster analysis Kroll's in-patient samples and were able to distinguish between borderline and other personality disorders.

It should also be objected that (a) the diagnoses of non-borderline personalities were not standardised; and (b) assessing one single personality disorder ignores their multidimensional presentation. Both objections were met by Stangl *et al* (1985), who found, among out-patients, overall agreement ($\varkappa > 0.7$) for the presence of any personality disorder, and for the presence of three individual types, borderline, histrionic, and dependent. The most frequent combination was borderline and histrionic, a finding related to that reported by Pope *et al* (1983), predicted by the DSM-III subgroup of 'dramatic' personality disorders, and obtained with cluster analysis by Kass *et al* (1985). The data of Frances *et al* (1984) further support the multidimensional viewpoint: in a sample of 26 borderline and 50 non-borderline out-patients, two-thirds of each group met the criteria for at least two DSM-III personality disorders.

So far this research shows, firstly, that some individual personality disorders (borderline or other) may be distinguished from each other, and secondly, that they may coexist in a variable number of subjects. This is also borne out by borderline and schizotypal personalities: (a) a large percentage of cases meet the criteria for both disorders (Spitzer *et al*, 1979; Frances *et al*, 1984), but (b) the two clusters can be separated by statistical analysis (Spitzer *et al*, 1979; Gunderson *et al*, 1983; Barrash *et al*, 1983). Furthermore, (c), no schizotypal patients were found in other borderline personality disorder samples (Pope *et al*, 1983); or the overlap was small (Kroll *et al*, 1981*b*, 1982; Akiskal, 1985*a*; Gunderson *et al*, unpublished). George & Soloff (1986) identified the risk of over-reliance on a single symptom group to distinguish the two. More about this is given in the next two sections.

Follow-up

Follow-up studies establish whether the patients can be said over time to have some other disorders which can better explain their original symptoms (Robins & Guze, 1970). A central question was whether borderline personalities were in fact not suffering from an early form of schizophrenia. In a five-year follow-up before the DIB and DSM-III were introduced (Carpenter & Gunderson, 1977), all the schizophrenic patients retained their original diagnoses but there was persistent diagnostic uncertainty about the borderline group. Despite the uncertainty, however, only one of the 24 borderlines was subsequently diagnosed as schizophrenic. More recently, no schizophrenics were found among DIB or DSM-III borderline samples after 4–7 years (Pope *et al*, 1983, $n = 27$) and after three years (Barrash *et al*, 1985, $n = 30$).

The second question concerned the relationship between borderline personality and affective illness. Pope *et al* (1983) found that of the mixed (borderline and affective) cases, 74% had possible/probable affective illness at follow-up, while the corresponding figure for the 'pure' borderline was only 23%. Akiskal (1985*a*) found a similar figure, 20% for melancholic episodes among 'pure' borderlines. Moreover, major depressions were equally prevalent among borderline and other personality disorders at three years, which argues against a *specific* link between borderline and affective disorders (Barrash *et al*, 1985).

The third question concerned the stability of borderline personality over time. The majority retained their original diagnosis (65% Pope, 60–90% Barrash), but some, in addition, received other personality disorder diagnoses as well, mainly in the 'dramatic group' (Pope). In only one or two cases was the diagnosis of schizotypal personality considered possible or present. McGlashan's (1983*b*) informal follow-up (average 15 years) on a large sample showed not only that few (16–24%) borderline personalities changed into schizophrenics, but also that 55% schizotypals developed schizophrenia.

The last question concerned social functioning. The presence of similar social outcome is a weaker argument for validity than the persistence of the diagnosis, for there is no one-to-one relationship between psychopathology and social functioning. Nonetheless, after five years, borderline patients had significantly better social functioning than schizophrenics (Carpenter & Gunderson, 1977), although at two years there had been no difference (Gunderson *et al*, 1975). In later studies, 'pure' borderline personalities presented outcomes intermediate between those of schizophrenic illness (worst) and affective illness (best) (Pope *et al*, 1983; McGlashan, 1983*b*, 1986).

In summary, borderline personalities tend to retain their diagnoses over time, but in addition they may present with other personality disorders, frequently of the 'dramatic' type. Several studies show that only a minority develop schizophrenia, in contrast with schizotypals. The majority of borderline personalities do not develop affective illness; a variable number display affective symptoms at follow-up, but probably no more than for other personality disorders. And the other way round? No one has yet examined whether affective illness resolves into a borderline state (Gunderson & Elliott, 1985). The hypothesis that borderline personalities are a variety of major affective illnesses is weakened by follow-up studies. Anecdotal evidence also indicates that symptoms tend to dilute with increasing age (confirmed by McGlashan, 1986), and that a tolerant partner, who buffers the patient's tendency to overreact, also helps. These issues too demand further attention. The natural history of the borderline disorders is only partially written; more work contributing to the debate about validity and providing a baseline for therapeutic trials is necessary.

Family studies

Robins & Guze (1970) argue that finding an increased prevalence of the same disorder in the relatives and in the index patients supports the validity of that diagnosis. Twin studies help to disentangle the relative aetiological importance of heredity and environment.

In this section we must consider both borderline (DSM–III or DIB criteria) and schizotypal personalities (the current concept of "borderline schizophrenia" in DSM–III), and their relationship to schizophrenia. In the Danish Adoption Study, Kety *et al* (1968) identified "B–3 or borderline schizophrenics" among index cases and relatives. The B–3 relatives were in the main relatives of schizophrenic index cases, supporting a genetic link between the two. A sample of B–3 subjects was used by Spitzer to define the criteria for DSM–III Schizotypal Personality Disorder. Kendler *et al* (1981) blindly applied Spitzer's criteria to the Danish records and confirmed that the schizotypals were more frequent among the biological relatives of chronic schizophrenic patients than among either relatives of controls or relatives of index B–3 cases. Gunderson *et al* (1983) went further, blindly reexamining the same records to look at both schizotypals and borderlines. They confirmed that among the B–3 relatives of chronic schizophrenics, the most prevalent diagnosis was schizotypal personality, not borderline. They also showed that the commonest diagnosis in the B–3 *index* cases was borderline personality (9 out of 10), and that their B–3 relatives had borderline rather than schizotypal features. These two studies therefore allow for two genetic propositions: (a) the mentally ill biological relatives of chronic schizophrenics are schizotypal and not borderline, and (b) the mentally ill relatives of borderline personalities are, in the main, themselves borderline. These propositions were supported by other samples; for example, among students and university staff (Baron *et al*, 1985). Also, Loranger *et al* (1982) found that borderline personality was ten times more common in the treated relatives of borderline patients than in the relatives of schizophrenic patients. Monozygotic twins of schizotypal patients have schizotypal disorders (33%) and not borderline (0%) (Torgersen, 1984). The most frequent diagnoses among relatives of borderline patients were 'dramatic' personality disorders (Pope *et al*, 1983).

The situation is complicated by the alternative hypothesis that borderline personality is genetically linked to affective illness (Stone, 1977). Among others, Soloff & Millward (1983) are often quoted to support this hypothesis, as they found that more borderline than depressed probands had relatives with "mood swings". This result, however, refers to a mixed group of 19 borderlines, 9 schizotypals and 20 cases who met both criteria; further analyses revealed the "depression" was actually more prevalent among relatives of schizotypals than borderlines. The homogeneity of the sample seems to be a crucial methodological precaution. Pope *et al* (1983) and Andrulonis & Vogel (1984) simply separated 'pure' borderlines from those who also had an affective illness, and found that the prevalence of depression was raised only in the relatives of the second, mixed, group. Torgersen (1984) found the same: "all the co-twins with an affective disorder were co-twins of schizotypal and borderline patients with a concurrent affective disorder as well". On the other hand, numerous objections can be raised against these studies. The re-examinations of the Danish Adoption records are a secondary retrospective analysis, vulnerable to circularity – this was the sample from which the schizotypal set was originally defined. Torgersen's was the only twin study, and he was not blind to who was twin to whom. The identification and/or diagnosis of cases and relatives is open to question in many of the studies, and can be criticised in some of them.

In summary, we are left with consistent evidence, albeit of diverse quality, leading to an attractive hypothesis: that 'borderline' and 'schizotypal' are dimensions that coincide in some subjects, but nevertheless have separate pedigrees. What is needed to further test this is a multi-centre twin study (to ensure adequate numbers and avoid idiosyncratic samples), with careful attention paid to the distinction between pure and mixed cases, and to the identification and assessment of the relatives at risk, not just the probands.

Laboratory investigations and psychological tests

Biological data is as sparse and inconclusive in the area of borderline personalities as it was for the whole of psychiatry 15 years ago (Robins & Guze, 1970).

Much of the work with biological markers to assess the relationship with affective disorders is questionable, either because the cases studied had both syndromes concurrently (Carroll *et al*, 1981), or because watertight controls were not available. Akiskal *et al* (1985*b*) described a REM sleep pattern in borderlines similar to that found in depressives; there were differences, however, between those borderlines who had had an affective diagnosis at any time in the past and those who had not. Coid *et al* (1984) found a raised level of plasma metencephalin among self-mutilators who met DSM-III borderline criteria.

Singer (1977) has reviewed the literature on psychological tests. In brief, borderline personalities show ordinary reasoning on highly structured tests, but on projective ones they "demonstrate flamboyantly deviant reasoning and thought processes". Test results distinguish between borderline and schizophrenic in-patients, but for out-patients the distinction is less clear. Borderline patients' responses to WAIS, Rorschach and other tests have been reported (Kernberg *et al*, 1981; Soloff & Ulrich, 1981). Borderline personalities in both the USA and the UK showed a characteristic Minnesota Multidimensional Personality Inventory (MMPI) profile: 8 = schizophrenia, 4 = psychopathic deviate, and 2 = depression (Kroll *et al*, 1981*b*, 1982). MMPI scores also differentiate borderline from schizotypal personality (Goldberg, 1985; Stangl *et al*, 1985).

In one study, family features were blindly rated: Gunderson *et al* (1980) found that over-involvement between the parents, with neglect of the offspring, distinguished borderline from schizophrenic and neurotic families.

In summary, it is premature to draw conclusions from the biological data, but the results of psychological tests are in keeping with clinical observations (e.g. regression in unstructured situations) and with other research (e.g. difficulty in differentiating borderline from schizophrenic out-patients). The observation of a particular family pattern should be replicated with wider samples and other methods.

Treatment response

Although not specifically mentioned by Robins & Guze, treatment response contributes to the delineation of a disorder: therapeutic success may suggest the existence of a specific pathogenic or aetiological factor (e.g. Teitelman *et al*, 1979). Few controlled studies of treatment exist: this is probably because of the lack, until recently, of reliable diagnostic criteria; because psychoanalytic psychotherapy is only described in case reports; and because of the nihilism of many psychiatrists about personality disorders. Early pharmacotherapy studies illustrate, more than anything else, the conviction of the writers as to which of the major psychoses borderlines should be affiliated, and/or existing differences in their patients' presentation.

Antidepressants (Klein, 1977) and low dose neuroleptics were used (Brinkley *et al*, 1979; Serban & Siegal, 1984). Recently two double-blind, placebo-controlled trials reported the efficacy of moderate dose of neuroleptics on chronic severe populations. On one sample of volunteers, the effect on psychotic-like symptoms was noted (Goldberg *et al*, 1986); on another, of in-patients, the effect on psychotic-like and affective symptoms was noted (Soloff *et al*, 1986). The latter also tried amitriptyline and patients got worse, a point raised by Gunderson (1986) to argue the independence of borderline personalities from affective disorders (cf. also Gardner & Cowdry, 1985). These important papers improved the therapeutic trials to acceptable current standards; but Goldberg *et al* too hastily suggest that the diagnostic criteria be changed to fit the target of a particular drug. The approach advocated in this review is wider.

The classic Menninger Clinic project (Kernberg *et al*, 1972) compared supportive psychotherapy, classical psychoanalysis and "expressive psychotherapy" conducted in an in-patient unit, to the third of which borderlines responded best. The sophistication of this study make the thought of replication daunting, but it is nevertheless surprising that nobody has attempted it.

In summary, good studies are just becoming available and have not yet contributed substantially to the issue of validity. Double-blind trials of medication, with detailed analyses of the effects, should continue and also be expanded to include psychotherapy with a design that helps identify the relative benefit of each (cf. Di Mascio *et al*, 1979).

Discussion

The papers reviewed, over 70 in all, published mainly between 1980 and 1986, show progress towards demonstrating the validity of borderline personality in terms of verifiable criteria. Three contributions were influential in this development.

In conceptual terms, Spitzer *et al* (1979) distinguished borderline ("unstable") from schizotypal ("borderline schizophrenia") personalities, a distinction empirically supported by themselves and others. The most important input to the research field, however, was Gunderson's Diagnostic Interview for Borderlines. The DIB conceives of borderlines in categorical terms, but this simplification has proved fruitful. The DIB provides a diagnostic score, like a screening instrument. More work is necessary on the relationships between the 140+ individual items; on the existence of subgroups (as a positive score can be obtained with different combinations of diagnostic statements); and on the prediction of external indicators. The third contribution, Kernberg's psychodynamic theory, is potentially amenable to ordinary research but little has been published so far. A fair assessment of Kernberg's psychoanalytic thinking has not been attempted here.

Research reviewed show that a group of patients with similar traits and symptoms sustained over a number of years can be identified in many institutions, in different countries, and in the community (Drake & Vaillant, 1985). It is frequently asked if borderlines are not a product of the North American culture. Borderline pathology has been recognised for years by psychoanalysts of different continents, and more recently by researchers in Europe (Kroll, Dahl, Torgersen, Bateman, Coid), in Australia (Mellsop) and among Hispanic samples in the USA (Castaneda & Franco, 1985). These patients are "unstable", "unpredictable" or "inconsistent" in their affects, impulses, identify and relationships, and are prone to transient psychotic episodes. Several research diagnostic criteria exist and they partially overlap, as is the case for other well-established psychiatric diagnoses: schizophrenia, for example, where they are also only partially concordant (Brockington *et al*, 1978). It will not surprise anybody if research shows that different sets, dimensions, or items of the borderline concept have different predictive power in terms of other variables; for example, prognosis (as shown for schizophrenia sets by Brockington *et al* (1978)), or heritability (as shown for schizophrenia by McGuffin *et al* (1984)).

Borderline personality and schizophrenia

To sustain its validity, the borderline concept had to prove first its separateness from schizophrenia. Independent studies with various criteria give the common message that borderline personalities and in-patient schizophrenics are reliably distinguished. Their longstanding features of borderline personalities stand out (e.g. impulsivity, chaotic lives, stormy relationships) and are remarkably different from those of the schizophrenics. In spite of some similarities (e.g. depersonalisation or derealisation) their symptoms also differ (e.g. characteristic phenomenology and course of the psychotic phenomena, cf. Pope *et al* (1985)). Furthermore, outcome, treatment response, psychological testing, and family studies all indicate that borderline personality as currently diagnosed is not a variety of schizophrenia. However, in out-patient settings, the distinction with milder, torpid, or remitted schizophrenics has not been studied well and is not so clear. We will suggest why, below.

Borderline personality and affective disorder

A variable, sometimes high proportion of cases present with both affective and borderline symptoms, at intake and follow-up. Firstly, depressions are common and multifarious conditions, the presence of which do not necessarily suggest that the concomitant personality disorder is dependent on them. Clinically, the assumption made is different: that some personalities are predisposed to depression. Secondly, the most characteristic features are not identical; the quality of the depression is frequently different. Barrash *et al* (1985) have shown that *other* personality disorders are equally at risk for depression, arguing against a specific unity between borderline and affective disorders.

Cases where both symptoms co-exist may explain some ambiguities: the development of affective symptoms such as dysphoric mood or self-harm cause borderline personalities to be admitted to hospital, when they appear more like affectively ill patients; among out-patients, while not in crisis, the more schizoid features such as aloofness and doubts about themselves may predominate, obscuring the distinction from remitted schizophenics (a hypothesis to test).

Thirdly, an excess of cases with depression has been found at follow-up, but these were considerably reduced when affective disorders were controlled for at the initial interview. It follows that 'pure' and 'mixed' cases should be independently analysed in future studies. The same applies to family studies: an excess of affective disorders was found among the relatives of the mixed (borderline plus affective) cases, not among the relatives of 'pure' borderlines. It also seems – procedural weaknesses considered – that the most frequent diagnosis among the latter is borderline personality itself.

Fourthly, the precise variety of affective disorder should be specified, as different hypotheses about the unity apply. Biological markers would contribute valuably to this debate. Similarly, Kernberg argues that some but not all depressives function at borderline level; and depression is less likely in personalities with both borderline and antisocial features, which is consistent with the psychodynamic link of aggression and depression (Perry, 1985).

At one point it was somewhat dramatically thought that if borderlines could not be reduced to a schizophrenic-like condition they would be subsumed *in toto* under affective disorders, removing any basis for their validity. On present evidence this is not likely, but it might be either that some borderlines are particularly prone to depression, or that one subgroup is truly a sub-affective disorder. Depressive features were common in two studies in the UK, where the issue could be profitably examined. But if the different proportions of cases with depressive symptoms reflect uncontrolled selection of patients at intake, an epidemiological survey – or at least a multi-centre study – will be required.

Borderline and other personality disorders

The distinction between borderline and other pathological personalities must be discussed in terms of categories and dimensions. Following Frances (1982), these are not mutually exclusive concepts. Categories are extreme, paradigmatic or severe forms of a continuum, cases where a particular sector of the personality pathology is so extreme that by comparison others are overshadowed. Adopting a categorical model is expedient and simplifies research – witness the DIB. Somewhere in the process, however, the researcher is faced with a triple variation: (a) cases that differ in severity, (b) cases that only partially satisfy the criteria, and (c) cases where the category appears accompanied by others – now it may be called a dimension. The researcher is required to change from an ordinary to a wide-angle lens. A multidimensional approach is more true to clinical practice, and more realistic – although more complex to handle – for therapeutic trials.

The papers reviewed support the validity of borderline personality and the standing of a multidimensional approach. Succinctly: (a) the individual diagnostic items can be reliably assessed (e.g. Frances *et al*, 1984); (b) they cluster in stable sets, confirmed statistically (e.g. Spitzer *et al*, 1979; Barrash *et al*, 1983); (c) varieties exist, defined by the severity or completeness of the sets (e.g. Koenigsberg *et al*, 1983; Kass *et al*, 1985); (d) they appear both in pure forms and in combination (e.g. Pope *et al*, 1983; Frances *et al*, 1984; Stangl *et al*, 1985); and (e) some combinations are more frequent (e.g. borderline plus histrionic or schizotypal). However, these studies have not examined the whole spectrum of personality disorders, only the specific varieties noted above. The true distribution of pure/mixed forms would only come from epidemiological surveys. The need to study the existence of varieties within the current concept of borderlines is confirmed by many studies. The search for a unitary description of the borderline personality has led to the relative neglect of its possible heterogeneity. However, the existence of subtypes has always been argued for by descriptive (Grinker *et al*, 1968; Andrulonis & Vogel, 1984), pharmacological (Klein, 1977), and psychoanalytic (Rosenfeld, 1979) findings. The DIB captures a pathognomonic pole of impulsivity and unpredictability with proneness to psychotic episodes. Another pole, of identity pathology, as yet less reliably assessed, and less studied, should be explored further (it is relevant for the assessment of psychotherapies based on object-relations theory.) It is also possible that varieties will become more firmly established on aetiological grounds (e.g. brain damage (Andrulonis & Vogel, 1984)), by the proximity to affective disorders or by response to treatment.

Researchers should identify their cases with more than one set of criteria and assess for other personality disorders as well, to contribute to the debate about validity and about multidimensionality. It is foreseeable that new work will modify the diagnoses now employed and progressively change current theoretical and pragmatic assumptions about personality disorders.

Conclusion

We can now modify Leibowitz's statement to say that the scale has tipped in favour of the diagnostic status of borderline personality. What before was a vague, mythical or muddled concept has achieved in many ways the significance of other commonly used psychiatric diagnoses. By this we mean that it has become a reasonable working description, assessed with acceptable objective criteria, allowing systematic investigation of its contents and of its limits, and useful to guide some clinical decisions. However, we would caution against the complacent reification of the diagnosis, as research in new – and some old – issues is clearly needed. These developments have taken place in the context of a growing interest in the whole field of personality disorders. If the 1970s were years of a renewed interest in schizophrenia, and the 1980s are witnessing considerable research in affective disorders, the 1990s will show unexpected developments in the investigation of personality disorders.

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'The Schizophrenias as Nervous Types' Revisited

GORDON CLARIDGE

In 1972 the author proposed that schizophrenia represents an aberration of central nervous processes that underlie cognitive and personality characteristics which can be observed in many healthy people. The present paper brings the evidence for that theory up to date and concludes that over the past 15 years it has received considerable empirical support. The way in which the theory confronts the question of whether or not schizophrenia is a disease is also clarified.

Some 15 years ago (Claridge, 1972), a perspective was offered on schizophrenia – or, more strictly, the schizophrenias – based on the idea that they "represent, in an exaggerated form, cognitive and personality characteristics found distributed in the general population." This conclusion was arrived at on the basis of evidence from genetic and other studies of schizophrenia, data from the author's own research, and theoretical concepts drawn from experimental and personality psychology which supported that 'dimensional' view of the psychotic states. Since then, a great deal of additional data has accumulated in these fields of research, and some of the theoretical issues have been further clarified. There have also been changes of viewpoint elsewhere about schizophrenia, with decline of the antipsychiatry movement and, as a backlash, the strong return in conventional psychiatry to organic disease explanations. It therefore seems timely to return to the ideas presented in the 1972 paper and see how they now stand, when judged against other contemporary opinions about schizophrenia.

The schizophrenias as nervous types – 1972

A first, and very important, feature of the 1972 model was its essentially *biological* nature, when compared with other theories (e.g. those of the antipsychiatrists), which, although also arguing for a continuity between psychosis and health, did so on entirely different grounds. A second, and related, point concerns the meaning of the concept of 'nervous types' around which the model was constructed.

The term 'nervous type' was first used by Pavlov (1928) to explain the fact that animals employed in his conditioned reflex experiments varied widely in their behaviour. He proposed that individual nervous systems differ in certain important physiological properties which can be observed overtly as variations in canine temperament. Since then, the idea has been elaborated and applied to human personality by several East European psychologists (Teplov, 1964; Nebylitsyn, 1971; Strelau, 1985). It has also formed the cornerstone of theory for most psychologists in the West who have sought a biological explanation of human individual differences. Notable here is Eysenck (1967), whose writings, more explicitly than most, also articulate another feature; namely the assumed continuity between those biological characteristics that underlie *normal* individual differences in behaviour and those responsible for *abnormal* or aberrant functioning: according to Eysenck (and others who take a similar viewpoint), particular varieties of 'nervous type' can take extreme forms, and it is they who represent the psychiatrically disordered.

Attempts to apply 'nervous type' theory to psychiatric illness are least controversial in the case of the neuroses and other mild forms of disorder. Thus, few would seriously challenge the suggestion that in anxiety neurosis the symptoms partly derive from the patient's more fearful temperament and hence more physiologically reactive nervous system (Gray, 1982); or that some kinds of antisocial behaviour are motivated by chronic 'sensation seeking' of biological origin (Zuckerman, 1979); or that each of these qualities can be found to a lesser degree in many psychologically normal individuals.

Not so well accepted is the possibility that the same may apply to the psychoses, including schizophrenia – that there may be many perfectly healthy people who have (to borrow a term from later in this paper) a 'schizotypal' nervous system, which stands in the same relationship to schizophrenia as, say, the anxious person's nervous system does to anxiety neurosis. Yet even in 1972, the idea was by no means new. For example, the antecedents of the continuity viewpoint could be seen in Kretschmer's (1925) notion of 'schizothymia–cyclothymia' as a dimension of normal temperament; and there already existed plausible polygenic models of inheritance to

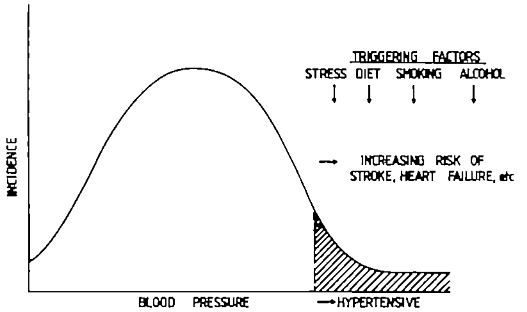

FIG. 1 The continuum of blood pressure as disposition to hypertension-related disease.

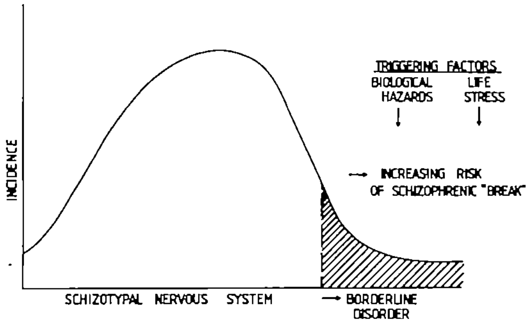

FIG. 2 The hypothetical continuum of 'schizotypy' as predisposition to schizophrenic disorder.

account for the graded manifestation of psychotic characteristics (Gottesman & Shields, 1968).

What was lacking in 1972, however, was a systematic body of information on two points: (a) how to measure schizophrenic-like traits in normal people, and (b) whether such traits have correlates elsewhere in behaviour and in the central nervous system. The thesis developed then was largely based on existing, rather scattered, evidence and on extrapolations from data collected in the course of psychophysiological investigations of clinical populations.

Another ambiguity in the earlier paper was its failure to deal with a crucial issue – how to reconcile a dimensional view of the psychoses with their undoubted status as diseases. Thus, the original model made no clear distinction between 'nervous type' as a descriptor of the enduring biological and personality characteristics of an individual, and its use to describe the same person when psychiatrically disturbed. Put another way, it did not distinguish those trait-like qualities of biological variations that have to do with the *predisposition* to illness and those which come into play during illness itself, giving rise to symptoms.

Predisposition and Illness

One frequently encountered hurdle to acceptance of these ideas is the conceptual difficulty of reconciling a view of schizophrenia as disease with the proposition that the latter has a certain continuity with psychological health. That the two are not as incompatible as they might seem becomes evident, however, if we consider what *kind* of disease schizophrenia could be. The suggestion to be made here has in fact been developed fully elsewhere as a *general* model for psychiatric disorder (Claridge, 1985), although this paper is concerned only with how it can be applied to schizophrenia.

The argument rests on a parallel that can be drawn between schizophrenia and the systemic physical disorders, the example to be used here being the hypertension-related diseases (Fig. 1). A necessary condition for such diseases to occur is sustained raised blood pressure. Yet blood pressure itself, as a naturally occurring characteristic, is merely a sign of a functioning organism and, like many other such traits, varies quantitatively in the population. On the other hand, it can also act as vehicle for disease, under certain pathogenic conditions and in some individuals with particularly high levels. In this example, therefore, blood pressure can be said to have *dual* properties: as a normal adaptive trait and as a potential predisposition to disease. The diseases (e.g. stroke) which it helps to mediate when acting as disposition can be distinguished by virtue of the new and increasingly catastrophic discontinuities of function that they bring about. In the case of the physical systemic diseases, therefore, there is no difficulty – and no controversy – about reconciling the presence of features in aetiology that are dimensional and those that involve a break in normal functioning.

A similar perspective can usefully be taken on schizophrenia (Fig. 2). The schizophrenic 'break' can indeed be seen as a discontinuity in behaviour – and therefore in brain function, thus preserving intact the notion of schizophrenia as a disease. However, this is perfectly compatible with the underlying disposition being dimensional, and describable as biological and psychological traits which, in the absence of triggering factors and when occurring in moderate degree, merely represent ways in which healthy human nervous systems vary.

In the 1972 paper the so-called 'borderline states' were quoted as evidence, in the clinical domain, of continuity in schizophrenia. Since then such conditions have received increasing attention, particularly in American psychiatry (Stone, 1980;

TABLE I
Psychotic trait scales

Scale	*Reference*	*Comment*
Schizotypy	Golden & Meehl (1979)	Based on MMPI items
Chapman scales		
Perceptual aberration	Chapman *et al* (1978)	Also scales of Nonconformity and Hypomania
Magical ideation	Eckblad & Chapman (1983)	
Social & physical anhedonia	Chapman *et al* (1976)	
STQ Schizotypal personality (STA) Borderline personality (STB)	Claridge & Broks (1984)	Modelled on DSM-II criteria for 'schizotypal personality disorder' and 'borderline personality disorder'
Predisposition to hallucination	Launay & Slade (1981) Bentall & Slade (1985 *b*)	Perceptual item content
Schizophrenism	Nielsen & Petersen (1976)	Attention/thought process items
Schizoid cognitions	Rust (1987)	Derived from multi-scale inventory of psychotic cognition
Psychoticism (P-scale)	Eysenck & Eysenck (1975)	Part of Eysenck Personality Questionnaire (EPQ)

Schizophrenia Bulletin, 1979, 1985). Although precise definition still remains a problem, the clinical form of the borderline disorders and the probable genetic relatedness of the schizotypal variety to schizophrenia (Kety, 1985) suggest that they do indeed represent muted forms of full-blown psychosis. As such, they provide another instance of the parallel drawn here between schizophrenia and the physical systemic diseases, where the existence of mild or subclinical varieties of disorder is well recognised.

In summary, as Gottesman & Shields reaffirmed in their 1982 book, schizophrenia can readily be seen as a diathesis-stress disorder, the underlying genetically determined disposition to which has a varying quality. How the latter manifests itself as phenotypic variation in the general population is a serious question for research on the aetiology of schizophrenia, and is one which the studies reviewed in the remainder of this paper were designed to answer.

An update of evidence

The bulk of the work to which we shall refer here has attempted to measure in normal subjects – usually adults – characteristics which can also be found in schizophenic patients. This research has generally proceeded through two phases: one, descriptive, in which personality and cognitive traits thought to be relevant are identified and measured by questionnaire; and a second in which high-scoring individuals on such questionnaires are examined with respect to some objective characteristic drawn from the schizophrenia literature or chosen on theoretical grounds as potentially significant in understanding the schizophrenic process.

Another strategy, which converges on the questionnaire method, is that used in 'high-risk' research. There the subjects are chosen according to their genetic affinity to a known schizophrenic, on the grounds that such individuals are more likely to show the traits that describe the disposition to schizophrenia. This method can merely entail studying the adult relatives of schizophrenics, but is found in its most complete form in the longitudinal follow-through studies of children considered to be at high risk for schizophrenic breakdown. A complete review of high-risk research is beyond the scope of this paper; but a recent volume has brought together details of most of the projects either completed or currently in progress throughout the world (Watt *et al*, 1984). Reference will be made to these investigations where the results can be brought to bear on the findings from questionnaire studies.

Questionnaire measurement of psychotic traits

Since the 1972 paper was published there has been a steady stream of questionnaires purporting to measure psychotic traits in normal adults: the more prominent of these are listed in Table I. Although most are recent, the thinking behind several of them goes back much further than 1972. In particular, the term 'schizotypy' was first coined in 1962 by Meehl to denote a form of schizoid personality detectable from a subset of Minnesota Multidimensional Personality Inventory (MMPI) items, validated against the full MMPI profile of schizophrenics and pre-schizophrenics (Golden & Meehl, 1979).

The remaining scales shown in Table I have had various labels attached to them and have differed considerably in comprehensiveness, item content, and theoretical standpoint from which they were devised. The most complete set is that constructed by Chapman and his colleagues, who have concentrated on measuring separately distinguishable features of schizotypy, such as anhedonia and perceptual aberration, items being drawn from clinical signs of schizophrenia. The validity of these scales, when used in normal subjects, has been successfully tested against clinical data obtained from the Schedule for Affective Disorders and Schizophrenia, Lifetime version (SADS-L) interview procedure (Chapman & Chapman, 1980; Chapman *et al*, 1980).

Other, more recently developed, instruments are Rust's Inventory of Schizoid Cognitions, the present author's two-scale STQ (modelled on the DSM-III criteria (American Psychiatric Association, 1980) for borderline states), and the Predisposition to Hallucination Scale devised by Slade and his colleagues, who have taken the slightly different approach of focusing on a single psychotic symptom, rather than sampling a range of schizotypal characteristics.

Standing somewhat apart from the other questionnaires are the psychoticism or P scales constructed by Eysenck & Eysenck (1976). These differ from the other scales in having been developed directly out of Eysenck's theory of personality. Consequently they purport to measure a person's *general* predisposition to any form of psychotic disorder, rather than schizophrenia in particular. Furthermore, the items in the published version, referred to in Table I, draw little from the clinical domain, focusing more on personality characteristics, especially of a socially deviant kind.

A question that naturally arises is how far these various scales relate to one another; since, despite the use here of the umbrella term 'schizotypy', it is already clear that they are unlikely to measure a unitary characteristic. To the author's knowledge there have been no systematic correlational or factor analytic studies in which all of the scales described have been included. Nevertheless, some conclusions can be drawn from three partially overlapping investigations: one by Chapman *et al* (1982), one by Muntaner & Garcia-Sevilla (1985), and an unpublished study conducted by Faily & Venables. The results broadly agree in suggesting that the various scales currently in use tap at least three separable components: perceptual and cognitive characteristics of a manifestly schizophrenic nature; feeling traits of an anhedonic kind; and deviant or antisocial behaviours and attitudes.

Thus, since 1972 considerable progress has been made in developing psychometric instruments for detecting psychotic traits in non-clinical populations. The existence of such traits in healthy adults now seems beyond reasonable doubt.

Experimental research

As mentioned earlier, all of the questionnaire scales just described have been used in studies attempting to characterise high-scoring individuals on experimental measures chosen according to their relevance to schizophrenia; mostly measures that have already been shown to discriminate clinically diagnosed schizophrenics from unselected groups of normal subjects or other psychiatric patients. The purpose of such research has been twofold: (a) to validate the questionnaires themselves, and (b) to throw further light on the aetiology of psychosis.

The domains of behaviour sampled and the experimental paradigms guiding particular studies have differed greatly, mirroring the equally variegated nature of research on the clinical syndrome itself. Three main approaches can be distinguished: psychophysiological, cognitive, and hemisphere function.

Psychophysiologial studies

In the argument of the case in 1972 for a nervous typological model of schizophrenia, particular reference was made to certain data, collected in the author's laboratory, suggesting that a unique feature of the psychotic nervous system might be its tendency to physiological 'dissociation', demonstrable empirically as a grossly altered sensitivity to sensory stimulation, inappropriate to the individual's prevailing level of tonic arousal. It had been suggested earlier (Claridge, 1967) that this might reflect some aspect of altered homeostasis in the nervous system, perhaps involving a weakening of inhibitory control.

In 1972 there was actually very little direct evidence that the characteristic just described might be a permanent feature of the nervous system in some healthy, but 'schizotypal', individuals. Since then, however, it has been possible to explore the idea in a series of interlocking studies having a common experimental strategy: the measurement, at varying levels of tonic arousal (electrodermal level), of individual subjects' sensory response, monitored with either EEG evoked potential or visual threshold procedures. To date, experiments have been conducted with drug-free schizophrenics (Claridge & Clark, 1982), the first-degree relatives of schizophrenics (Claridge *et al*, 1985), a rhesus monkey under LSD-25 (Claridge *et al*, unpublished), and normal subjects with high scores on an early version of the Eysenck P scale (Claridge & Birchall, 1978). In all of these cases the same unusual 'dissociation' of function was observed, when compared with the various control comparisons made. The findings would therefore strongly support the author's original suggestion that a dysregulated mode of CNS functioning, rather than altered reactivity *per se*, might be one important biological characteristic disposing to schizophrenia. This conclusion would be in line with that of Haier *et al* (1980), who found that psychiatric vulnerability, assessed in otherwise normal subjects, was best predicted not by a single biological indicator but by an altered profile of response which they interpreted as signifying an 'unbalanced' or poorly regulated mode of central nervous control.

In distinction to the studies just described, other workers have confined themselves to examining a *single* psychophysiological measure, in order to determine whether 'schizotypal' subjects, like schizophrenics, show an abnormality of 'arousability', usually as indexed by some aspect of electrodermal responsivity. In evaluating these findings it is important to bear in mind the great heterogeneity found on such measures among schizophrenics themselves, a fact articulated in the putative distinction made by Venables and his colleagues between 'responder' and 'non-responder' forms of schizophrenic syndrome (Gruzelier & Venables, 1975), and also mirrored in the differing findings reported by those workers who have studied high-risk children from a similar point of view: there both abnormal hyperresponsivity (Mednick & Schulsinger, 1968) and its opposite (Kugelmass *et al*, 1985) have been said to characterise such children.

Questionnaire-based studies using normal adults reveal the same picture. Thus, Nielsen & Petersen (1976) reported increased physiological responsiveness in subjects with high scores on their specially constructed scale of 'schizophrenism' (see Table I); while the opposite has been found by Simons (1981, 1982) using the Chapman anhedonia scales and by Robinson & Zahn (1985) using the published Eysenck P scale.

It is possible that the above findings reflect the use of scales tapping different aspects of 'schizotypy'; or alternatively, as suggested earlier, that schizotypy is characterised more by an *instability* of physiological reactivity, leading to extremes of response in either direction, depending on the conditions of testing (Claridge (1987) gives a fuller discussion of this point). Either way, the heterogeneity of psychophysiological response observed in these studies can at least be said to map well on to parallel data obtained with schizophrenic patients and genetically at risk children.

Cognitive studies

The relevant work here has been carried out against the background of the considerable research that has been done on the cognitive aspects of schizophrenia, in both patient and high-risk samples (see Nuechterlein & Dawson (1984) for a recent review). As in the clinical literature, it has been guided by several different paradigms.

One 'classic' approach, which continues a theme from the author's 1972 paper, has involved looking for evidence of the loosened or divergent styles of thinking that would be expected in appropriately selected normal subjects. Subsequently, Woody & Claridge (1977), using the Eysenck P scale and a standard battery of 'creativity' tests, demonstrated that high scorers were indeed more divergent. More recently a similar finding was reported by Miller & Chapman (1983), using the latter's scales in conjunction with a continuous word association task: subjects with high scores in Perceptual Aberration/Magical Ideation gave a larger number of idiosyncratic responses. Miller & Chapman's result converges closely on several similar observations in high-risk research, including a finding by Mednick and his colleagues who, using the same task, reported more deviant associations in the children of schizophrenic parents (Griffith *et al*, 1980).

Other approaches to the problem have drawn on more contemporary ideas in cognitive psychology that have inspired attempts in schizophrenia research to identify the stage or component of information processing which mediates psychotic symptoms. One hypothesis – that processing is slower at a very early stage of input – has received support from several studies using the 'backward masking' procedure, which tests the subject's efficiency in reporting a target stimulus presented at varying intervals after a strong, non-informational visual mask. Reduced efficiency on this task has been found right along the schizophrenic 'spectrum', including not only fully diagnosed schizophrenics and subjects with schizotypal personality disorders (Braff, 1981; Saccuzzo & Schubert, 1981), but also normal subjects with the schizotypal MMPI profiles referred to earlier (Steronko & Woods, 1978; Nakano & Saccuzzo, 1985).

Another view of information processing in schizophrenia is that offered by Frith (1979), who has argued that schizophrenics have difficulty in limiting the contents of consciousness, due to weak inhibitory selection of information at the early, preconscious stage of processing. An appropriate methodology for testing this hypothesis is that of 'priming', in which the subject's response to a probe target (e.g. a word) is manipulated with a briefly presented prior stimulus. Predictable differences have been found using various applications of this general technique, in studies of schizophrenic patients (Bullen, 1982) and of normal subjects with high scores on the Eysenck P scale (Bullen & Hemsley, 1984) and on the schizotypal personality scale of the author's STQ inventory (Beech & Claridge, 1987). The last experiment, one of a number currently in progress in the author's laboratory, was specifically designed to test *negative* priming, which is thought to tap inhibitory effects in selective attention (Tipper, 1985). The results of all of these studies support the idea that schizotypal individuals, like schizophrenics, do indeed show weakened inhibition during information processing.

Another, rather different line of research is that pursued by Slade and his colleagues into the possible mechanisms of hallucinations. As noted earlier, their questionnaire concentrates specifically on disposition to hallucinations as a normal characteristic, and their experimental investigation of the new scale has had a corresponding focus, derived from signal detection theory (Bentall & Slade, 1985*a*). Carrying out an analysis of normal subjects' performance on an auditory detection task, they showed that high scorers on their hallucinatory disposition scale differed significantly in perceptual bias, such that they were more likely to report that a target stimulus was there when it was not. Notably, Bentall & Slade found an identical effect in hallucinating, compared with non-hallucinating, schizophrenic patients.

Studies of hemisphere function

The third field of experimental research to be considered here is relatively recent, although the clinical evidence from which it is derived has been extensively documented (Walker & McGuire, 1982; Gruzelier, 1983). The possibility that the unusual cerebral asymmetries observed in schizophrenic patients – particularly with reference to the processing of linguistic material – may find their counterpart in normal individual differences in laterality is one that has been explored recently by the author and colleagues, in relation to their scale of schizotypal personality. These studies, with one exception, have used a divided visual field procedure to present various kinds of stimuli, including nonsense syllables (Broks, 1984) and single letters (Rawlings & Claridge, 1984). It has been found that highly schizotypal normal subjects are indeed comparable to schizophrenics in showing either weakened or even reversed laterality, a particular feature being their relatively efficient processing of such material when it is routed first to the right hemisphere.

Another investigation in this same series looked at the auditory modality (Broks *et al*, 1984). The procedure adopted here was the story comprehension test developed by Green and his associates and used by them to demonstrate, among other things, that both schizophrenics and the children of schizophrenics show poor recall of binaurally, compared with monaurally, presented prose (Green *et al*, 1983). Of two experiments by the author and colleagues in normal adults, one showed exactly the same effect for high schizotypes. The other, using slightly different stimulus material, did not, although it did confirm part of the findings of the author and his associates in the visual modality; namely that schizotypal individuals show reduced asymmetry, due to a superior performance in (in this case) the left ear.

In addition to the similarities, there are also marked differences between schizophrenics and normal schizotypes; notably that the former regularly perform poorly and the latter in general perfectly well when linguistic material is presented through what is usually regarded as the most efficient route; that is, through the right ear or right visual field. The view on this currently held by the author and his co-workers would draw on ideas such as those put forward by Randall (1980), Wexler (1980) and others and suggest that schizophrenia may be associated with *enhanced* communication between the two halves of the brain, perhaps due to decreased inhibition. In the normal schizotype this might not be disadvantageous but might become increasingly so under certain conditions or when present to an extreme degree.

The schizophrenias as nervous types – 1987

From the studies just reviewed, the theory put forward in 1972 now looks even more substantive. There seems to be irrefutable evidence that logically related biological, cognitive, and personality characteristics ascribed to schizophrenia can also be observed in some healthy individuals. Furthermore, the surrounding evidence, especially from genetics research, indicates that these characteristics constitute inherited traits predisposing to the disorder.

Nevertheless – although this is equally true of research on patients – a unifying theme has still to emerge; in the sense of one sure idea being found which can account for data that crosses such varied domains as psychophysiology, cognitive psychology, and the neuropsychology of hemisphere function. It is, however, noted with interest that all three of these approaches have, in one guise or another, led to the conclusion that a crucial feature might be some deviation in the setting-point for mechanisms that exert inhibitory control over psychobiological function. The exact form that this might take, or the exact level in the nervous system at which it might operate, is of course entirely speculative; but Johnson's (1985) recent suggestion that some aspect of 'feed-forward' inhibition could be involved is very persuasive.

There are also two other problems not as yet entirely resolved. One concerns the fact that, at a descriptive level, schizotypy seems not to be a unitary dimension, but to consist of several subcomponents which between them encompass personality traits of social nonconformity as well as more manifestly psychotic characteristics. A related question is whether, and if so how, such components map on to the clinical syndromes themselves. Do they, for example, either singly or in varying combinations, form dispositions to different varieties of schizophrenic reaction? A further possibility that must be considered is that the term 'schizotypy' is already too narrow and that the traits discussed here encompass the entire range of psychosis. For there is increasing reason to believe that the schizophrenias and the affective psychotic disorders are aetiologically less distinct than was once believed (Claridge, 1985; Crow, 1986).

Although a precise knowledge of the 'schizotypal nervous system' is still lacking, the model developed in this and the 1972 paper does at least establish a *general principle* for future research on the aetiology of schizophrenia; namely the need to complement knowledge about normal variations in behaviour and brain functioning with observations about possible exogenous biological and social factors that help to trigger the symptoms of illness in the predisposed individual. The interfacing of these two domains might ultimately prove crucial to understanding. After all, to recall the analogy with the physical systemic diseases, it is unlikely that the causes of stroke or heart disease could ever have been properly understood without knowledge of normal vascular function.

Adopting the strategy just described would place more burden than hitherto in schizophrenia research on the study of healthy, but identifiably predisposed, individuals who might provide clues to aetiology not available, or obscured, in the currently psychotic person. That conclusion is of course identical to that which first inspired longitudinal high-risk studies of schizophrenia (Mednick & McNeil, 1968) and which continues to do so. It is therefore satisfying that some of the findings of the cross-sectional experiments referred to here converge on the results of those projects. Indeed, there are already signs of an increasingly blurred distinction between high-risk research proper and research carried out from the viewpoint of normal differential psychology (Siever *et al*, 1985). The fact that some of this work (e.g. Haier *et al*, 1987) is beginning to make use of the newer brain imaging techniques – albeit in the quest for functional differences rather than discrete lesions – might also help in the narrowing of the other gap in schizophrenia research which it has been the purpose of this paper to encourage: that between 'disease' and 'dimensional' theories of the disorder.

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Post-mortem Study of Chronic Schizophrenic Brains

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Volume measurements were carried out on 29 brains from institutionalised, chronic schizophrenic patients and 30 age and sex-matched controls using a stereological method. The volumes of the total fixated brain, the hemispheres, the cortex, and the central grey matter of the schizophrenic brains were significantly reduced while the volumes of the ventricles were significantly enlarged compared with the control group. The patients had been diagnosed by a psychiatrist on the basis of their case records as having either a Type I or Type II syndrome according to the symptoms they presented in the first years of their disease. The Type II patients were found to have significantly enlarged ventricles compared with the Type I patients.

Kraepelin in 1896 and Bleuler in 1916 expressed the view that a neuropathological basis of schizophrenia would eventually be found. Nevertheless, Winkelman & Book (1949), who reviewed a number of histopathological papers written from 1924 to 1944, could state in 1948 that "excellent investigators have concluded that dementia præcox has no organic foundation". They reported the results from Dunlap (1924), Spielmeyer (1930), Bamford & Bean (1932), Peters (1937), and Scheidegger (1942), who found no changes in the post-mortem brains from schizophrenia patients which could not be duplicated in 'normal' controls. They also reported investigations showing unspecific findings in schizophrenic brains. Watanabe (1934) found acute and severe nerve cell disease in acute cases of schizophrenia and fatty changes in nerve cells in Ammon's horn. Miskolczy (1937) reported disappearance of the cortical neurones of the frontal, parietal, and temporal regions, lipoid infiltration of the nerve cells, and mild glial proliferation. Winkelman & Book (1949) found focal and general loss of nerve cells, degenerative changes, and fairly uniform hyperplasia and hypertrophy of macroglia in ten selected cases of schizophrenia from a group of 60.

Since then, various studies have been carried out on schizophrenic patients. Using encephalography, Huber (1957), Haug (1962), and Asano (1967) found enlarged ventricles in schizophrenic patients. In 1976, Johnstone *et al*, from computerised tomographic (CT) head scans, reported significantly enlarged lateral ventricles in chronic schizophrenic patients.

The histopathological findings and the observation of enlarged ventricles in some schizophrenic patients have been a continuous encouragement to seek further knowledge of this complex disease.

In 1983, Weinberger *et al* made a selective review of recent post-mortem studies, pneumoencephalographic studies and CT findings. They concluded that qualitative non-specific pathology of the limbic system and the diencephalon is associated with many cases of schizophrenia. Mild, but statistically significant, ventricular enlargement consistent with, but not diagnostic for, limbic or diencephalic pathology is the most prevalent finding, along with anterior cerebellar vermis atrophy, atypical lobar asymmetries, and regional decreases in brain density, the clinical relevance of which awaits clarification.

In the work of Kovelman & Scheibel (1986), attention is directed to the biological substrates of schizophrenia, including their own observation of varying degrees of structural disorganisation and disorientation of the hippocampal pyramidal cells making up the cellular layer of the cornu ammonis. In their conclusion they emphasise the structural and physiological studies, which are mutually persuasive in their indication of left hemispheric dysfunction, deficits or interhemispheric integration and limbic system involvement.

Undoubtedly, psychiatric research has often presented positive findings, but few of these have led to a clearer understanding of the pathogenesis of psychiatric illness or to more effective treatment. In 1982, Crow hypothesised that schizophrenics represent two symptom groups, each reflecting a different underlying disease process. The constellation referred to as the Type I syndrome is characterised by such positive symptoms as hallucinations, delusions, and thought disorders. This type of illness, most often seen in acute schizophrenia, shows a good response to neuroleptics and an absence of intellectual impairment. The outcome is described as reversible, and the postulated pathology could be due to a disturbance in the dopamine system. The Type II syndrome is characterised by such negative symptoms as affective flattening, poverty of speech, and loss of drive. This illness has a more chronic course and a poor response to neuroleptics, and the patients may develop intellectual impairment. The outcome may be irreversible. While the Type I syndrome is not associated with enlargement of the ventricles, the brain cell loss and structural changes in the Type II syndrome have been related to cerebral atrophy. While the two syndromes may represent independent dimensions of pathology, they do not constitute separate diseases. Patients may have symptoms of both types, either at the same time or sequentially.

To date, there have been many investigations of quantitative observations in schizophrenic patients, but less attention has been paid to post-mortem quantitative findings. The aim of this study was to determine the volume of the cortex, white matter, ventricles and central grey matter in post-mortem brains of chronic schizophrenics compared with control brains in a double-blind examination using a stereological method (Regeur & Pakkenberg, in preparation), and to relate the results to the positive/negative symptoms of the patients, as well as other parameters.

Method

Material

Twenty-nine brains from chronic schizophrenic patients were collected from two psychiatric wards over a period of 18 years (1967–1985) and matched according to age and sex with 30 brains from control patients collected in the period 1973–1985 from four pathology department wards in Copenhagen. The brains were removed within 72 h after death and kept in 10% formalin for at least 5 months to ensure a stable volume (Haug *et al*, 1984). Exclusion criteria for schizophrenic patients were strokes or tumours in the central

TABLE I

Schizophrenic patients: sex, age, treatment, and course of illness

Case	*Sex*	*Age*	*Age at first admission*	*Time in psychiatric ward: years*	*Treatment*			*Dyskinesia*[1]	*Time from start of treatment to dyskinesia: years*
					Number of ECT	*Number of insulin comas*	*Neuroleptics (low–middle–high): years*		
1	M	70	14	37	16	–	26	BLM + extremities	26
2	F	79	60	7	–	–	Few months	–	–
3	M	53	32	15	24	–	15	–	–
4	F	88	44	27	–	–	2	–	–
5	F	75	30	44	–	–	16	BLM	5
6	F	74	34	40	7	–	5	BLM	1
7	F	76	40	33	–	–	16	BLM	5
8	M	78	36	43	–	–	15	–	–
9	F	73	25	50	–	–	8	–	–
10	M	79	41	33	21	28	23	–	–
11	M	84	29	53	8	43	28	–	–
12	M	78	22	56	–	–	13	BLM + extremities	9
13	F	89	32	33	–	–	9	–	–
14	F	71	31	49	–	–	8	BLM	6
15	M	78	60	18	5	–	17	BLM + tremor	7
16	F	71	30	41	7	–	22	BLM + extremities + tremor	4
17	F	91	53	48	–	48	6	–	–
18	M	65	25	40	–	–	3	–	–
19	M	52	15	30	22	28	25	BLM	25
20	M	82	31	48	–	–	Few months	–	–
21	F	61	33	19	12	–	22	–	–
22	F	84	39	44	–	–	18	BLM + body + tremor	5
23	M	68	20	4	–	–	10	BLM	10
24	M	41	22	17	12	–	20	–	–
25	M	45	20	24	40	28	23	–	–
26	M	80	24	56	–	–	30	–	–
27	F	79	16	45	12	33	29	–	–
28	F	90	40	11	–	–	14	BLM + body + extremities	16
29	F	87	45	17	2	–	15	BLM + head	5
		73.8	32.5	33.9	14.5	34.7	16.2		9.5
		(41–91)	(14–60)	(4–56)	(2–40)	(28–48)	(2–30)		(1–26)

1. BLM = bucca-lingua-masticatory syndrome.

nervous system (CNS) and drug abuse. For controls, exclusion criteria were death from cancer, any history of alcohol or drug abuse, earlier or present strokes, tumours or infections in the CNS, any record of dementia, and being comatose for more than 24 h prior to death. All relevant parameters are shown in Table I.

Table II shows the general information concerning the patients.

A psychiatrist had classified the patients as Type I or Type II on the basis of their symptoms during the first years of their illness. These results are shown in Table III, with the respective diagnosis, and whether or not the patients were demented in their final years, and whether or not there was a family history of schizophrenia.

Nine patients had seven first-degree relatives who were schizophrenic and ten second-degree relatives with this diagnosis. Nine first or second-degree relatives suffered from such other psychiatric diseases as paranoid psychosis, manic–depressive illness, or anxiety neurosis. Eight of the nine patients with affected relatives had a paranoid form of schizophrenia.

Five first or second-degree relatives committed suicide. In one case, a female patient had a father, an uncle and two cousins who were schizophrenic; two uncles and an aunt committed suicide.

Socially, most of the patients were not very well integrated. Only one finished high school and none were academics. Ten of the 29 had been married. The women had mostly been employed as domestic help, the men as unskilled labour. Four patients had for a time had clerical work and one had been a musician; none had had employment subsequent to admission to a psychiatric ward, except for the musician.

One patient had had a history of lues cerebrospinalis, another had been an alcoholic when he was young. Thirteen patients developed dyskinesia, all of them oral dyskinesia, and five of them additional symptoms such as hyperkinesia of the head, body, or extremities and/or hand tremor. All 13 had received high-dosage neuroleptics (Table I).

Table IV shows the general information for the controls.

Procedure

The brains were weighed and fixed. After at least 5 months in 10% formalin, the brains were weighed again, the

TABLE II

Schizophrenic patients: height, weight, brain data, and terminal illness

Case	*Height: cm*	*Weight: kg*	*Total weight of fixed brain: g*	*Time from death to fixation: 24 h*	*Fixation time: months*	*Duration of terminal illness*	*Cause of death*
1	175	69.5	1450	1	5	Min	Coronary occlusion
2	152	50.0	1020	2	74	Days	Cardiac failure
3	173	103.9	1420	2	108	Days	Bronchopneumonia
4	–	83.2	1085	3	114	Days	Bronchopneumonia
5	–	70.2	1246	1	156	Months	Carcinoma of the anus
6	157	68.6	970	1	174	Months	Pulmonary embolism
7	155	52.0	970	2	120	Days	Penetrating gastric ulcer
8	178	70.0	1260	2	71	Min	Atherosclerosis universalis
9	157	33.8	1140	3	120	Months	Bronchopneumonia
10	178	73.2	1142	1	60	Months	Carcinoma of the liver
11	174	57.8	1305	1	18	Days	Cardiac failure
12	166	72.0	1325	1	66	Min	Atherosclerosis universalis
13	160	69.1	880	1	192	Months	Atherosclerosis universalis
14	–	56.5	1120	2	198	Days	Bronchopneumonia
15	169	46.5	1220	2	39	Weeks	Bladder cancer
16	151	65.1	1120	1	84	Weeks	Bronchopneumonia
17	163	62.0	1156	3	72	Months	Bronchopneumonia
18	185	72.7	1268	2	132	Months	Lung cancer
19	186	56.0	1427	1	26	Days	Coronary occlusion
20	168	53.0	1145	1	63	Days	Bronchopneumonia
21	152	46.7	1072	3	106	Weeks	Bronchopneumonia
22	165	45.1	1270	1	107	Weeks	Bronchopneumonia
23	182	63.5	1510	3	72	Days	Bronchopneumonia
24	184	70.0	1485	2	70	Months	Chronic pancreatitis
25	175	58.0	1330	2	74	Months	Colonic cancer
26	171	70.0	1320	3	7	Days	Pulmonary embolism
27	162	65.0	1007	1	10	Days	Bronchopneumonia
28	156	56.5	1006	2	77	Weeks	Carcinoma of the liver
29	164	43.1	1103	2	60	Months	Colonic cancer
Mean	167.6 (151–186)	62.2 (33.8–103.9)	1196 (970–1510)		85.3 (5–198)		

TABLE III
Schizophrenic patients: diagnosis, classification, and factor of inheritance

Case	*Diagnosis*	*Type*	*Demented in final years*	*Notified factor or inheritence (first degree)*
1	Schizophrenia paranoides	I+II	+	−
2	Schizophrenia paranoides	I	+	−
3	Schizophrenia paranoides	I	−	−
4	Schizophrenia paranoides	II	−	+
5	Schizophrenia paranoides	I	+	−
6	Schizophrenia paranoides	I	−	−
7	Schizophrenia paranoides	I	+	−
8	Schizophrenia simplex	I	+	−
9	Dementia præcox	II	+	−
10	Schizophrenia paranoides	II	+	+
11	Schizophrenia katatonica	II	+	−
12	Schizophrenia simplex	I	+	−
13	Schizophrenia paranoides	I	−	−
14	Schizophrenia paranoides	II	+	−
15	Schizophrenia paranoides	I	+	+
16	Schizophrenia hebephrenica	I	−	−
17	Schizophrenia paranoides	I	−	−
18	Schizophrenia simplex	II	+	−
19	Schizophrenia simplex	I+II	+	−
20	Schizophrenia simplex	II	+	−
21	Schizophrenia simplex	II	−	−
22	Schizophrenia paranoides	I	+	−
23	Schizophrenia paranoides	II	+	+
24	Schizophrenia hebephrenica	I	−	−
25	Schizophrenia paranoides	II	−	−
26	Schizophrenia katatonica	II	+	+
27	Schizophrenia paranoides	I	−	−
28	Schizophrenia paranoides	I+II	+	−
29	Schizophrenia paranoides	II	−	+

meninges were removed, and the cerebellum and brain stem cut off corresponding to the upper level of the pons. The brains were incubated in 20% gelatin in 2% aqueous phenol, and then placed for 7 days in a refrigerator at 4°C, after which the surplus of gelatin was trimmed off, leaving the brain with a gelatin coat approximately 2 mm thick. The brains were then cut in coronal slices 6 mm thick, with a cutting apparatus specially designed for the purpose. Each slice was photographed, colour slides were produced, the slides were placed in a Kodak Carousel S-AV, and the picture was projected down on to a table by way of a mirror. Each slice was point-counted using a double-lattice grid with 64 coarse points and 1024 fine points (Weibel, 1979). The length of the brain was measured leaving out the first and last slice, and the volumes of the hemispheres, cortex, white matter, ventricles and central grey matter were determined using Cavalieri's theorem (1966):

$$\mathrm{vol}_{\mathrm{cerebrum}} = \bar{t} \times a(\mathrm{frame}) \times \Sigma P$$

where

$$\bar{t} = \frac{\text{length of brain} - (\text{first} + \text{last slice})}{\text{number of slices} - 2}$$

a(frame) = area per point on the grid, corrected for magnification

ΣP = the total number of points falling on the object.

The coefficient of error for the total procedure is 0.028.

Statistical methods After ensuring normal distribution (χ^2 of fit), differences between groups were tested by comparing two regression lines, schizophrenic versus control, taking into account the fact that brain volumes in humans are correlated to body height. Since this was the case for all parameters except the volume of the ventricles, the ventricle volume was tested by two-tailed Student's t-test. Only $2P$ values less than 0.05 were considered statistically significant.

TABLE IV
Controls

Case	*Sex*	*Age*	*Height: cm*	*Weight: kg*	*Total weight of fixed brain: g*	*Time from death to fixation: 24 h*	*Fixation time: months*	*Duration of terminal illness*	*Cause of death*
1	F	75	166	84.7	1270	1	11	Min	Coronary occlusion
2	M	71	175	72.0	1355	1	11	Days	Cardiac failure
3	F	69	162	81.8	1125	3	11	Days	Bronchopneumonia
4	F	67	167	62.0	1205	1	11	Min	Pulmonary embolism
5	M	74	170	74.0	1320	3	11	Days	Mesenteric thrombosis
6	M	55	177	92.0	1360	3	5	Hours	Coronary occlusion
7	M	59	179	70.0	1450	2	6	Days	Coronary occlusion
8	M	57	174	80.0	1550	1	6	Min	Coronary occlusion
9	M	55	178	96.0	1500	2	6	Min	Coronary occlusion
10	F	41	156	89.0	1305	1	5	Min	Coronary occlusion
11	M	71	175	72.0	1650	2	13	Min	Coronary occlusion
12	M	68	165	47.7	1310	2	9	Days	Chronic bronchitis
13	M	72	163	70.2	1175	2	10	Min	Coronary occlusion
14	M	84	170	80.8	1400	1	9	Days	Septic shock
15	F	95	–	–	940	1	108	Min	Coronary occlusion
16	F	57	162	50.0	1360	1	6	Min	Coronary occlusion
17	F	60	–	–	1155	1	102	Days	Coronary occlusion
18	F	84	–	–	923	1	108	Min	Atherosclerosis universalis
19	F	75	158	48.0	1175	1	5	Hours	Coronary occlusion
20	F	65	159	61.0	1450	1	5	Hours	Coronary occlusion
21	F	86	–	–	930	1	126	Min	Coronary occlusion
22	F	93	155	48.0	1176	1	108	Days	Bronchopneumonia
23	M	73	171	73.0	1270	3	96	Min	Coronary occlusion
24	F	88	–	–	1184	3	108	Min	Pulmonary embolism
25	M	57	182	73.0	1343	2	5	Hours	Coronary occlusion
26	M	83	166	44.0	1133	2	5	Min	Coronary occlusion
27	F	66	160	53.0	1253	2	5	Min	Coronary occlusion
28	M	82	177	56.0	1129	2	5	Min	Cardiac failure
29	M	75	182	73.0	1360	1	7	Hours	Coronary occlusion
30	M	84	158	51.0	1230	1	5	Min	Coronary occlusion
Mean		71.4 (41–95)	168.3 (155–182)	68.1 (44–96)	1266		30.9 (5–126)		

Results

There was a statistically significant difference between the controls and the schizophrenics as regards total brain weight (controls $\bar{x}_c = 1314$ g, $CV_c = 0.10$; schizophrenics $\bar{x}_s = 1205$ g, $CV_s = 0.15$), $2P = 0.0018$; the hemispheres ($\bar{x}_c = 1046$ ml, $CV_c = 0.13$, $\bar{x}_s = 959$ ml, $CV_s = 0.16$), $2P = 0.015$; the cortex ($\bar{x}_c = 578$ ml, $CV_c = 0.16$, $\bar{x}_s = 510$ ml, $CV_s = 0.16$), $2P = 0.0030$; central grey matter ($\bar{x}_c = 47.9$ ml, $CV_c = 0.14$, $\bar{x}_s = 44.5$ ml, $CV_s = 0.16$), $2P = 0.048$; and the ventricles ($\bar{x}_c = 26.3$ ml, $CV_c = 0.37$, $\bar{x}_s = 35.1$ ml, $CV_s = 0.47$), $2P = 0.024$.

Fourteen patients had been classified as Type I and twelve patients as Type II, while three were a mixture of Type I and Type II (Table III). The volumes of the ventricles were significantly smaller ($2P = 0.015$) in the Type I patients than in the Type II patients. There was no statistically significant difference in the volumes of the white matter ($\bar{x}_c = 395$ ml, $CV_c = 0.11$, $\bar{x}_s = 370$ ml, $CV_s = 0.23$) ($2P = 0.23$) in the schizophrenic brains compared with the controls. In the 18 demented patients, the ventricles did not differ significantly in size from those of the other schizophrenics ($2P = 0.17$), nor did the cortex volume from the demented patients differ from that of the other schizophrenics ($2P = 0.86$).

The 13 patients who developed hyperkinesia had neither significantly larger ventricles ($2P = 0.92$) nor smaller central grey matter ($2P = 0.83$) than the rest of the schizophrenic group. There was no significant difference in the length of treatment in the patients who developed dyskinesia compared with the rest of the schizophrenics ($P = 0.78$). There was no significant difference in the height ($P = 0.80$) or weight ($P = 0.14$) of the control patients compared with the schizophrenic group. The length of neuroleptic treatment was unrelated to the Type I/Type II diagnosis ($P = 0.96$). The cortex of patients treated with ECT showed no significant difference from that of the rest of the schizophrenics ($2P = 0.88$).

The ventricles of the six patients treated with insulin-induced hypoglycemic coma were of normal size ($P = 0.89$) compared with the rest of the schizophrenic group. Other investigators have found smaller ventricles in such patients (Johnstone *et al*, 1976). The lack of confirmation of their results could be due to the small number of insulin-treated patients in this study.

The nine patients who had a first or second-degree relative with schizophrenia did not have significantly enlarged ventricles ($P=0.48$) compared with the rest of the schizophrenic patients.

The size of the ventricles in the younger controls ($n=9$, age 41–65 years with a $\bar{x}_{c,young}=20.8$ ml) was significantly smaller ($P=0.024$) than in the older controls ($n=21$, age 66–95 years, $\bar{x}_{c,old}=29.6$ ml). In the schizophrenic group there was no difference between the size of the ventricles in the younger individuals ($n=6$, age 41–65 years, $\bar{x}_{s,young}=36.6$ ml) and the older ones ($n=23$, age 66–95 years, $\bar{x}_{s,old}=34.9$ ml), $P=0.83$.

There was no statistically significant difference in the size of the ventricles between the paranoid and non-paranoid schizophrenic patients ($P=0.50$).

Discussion

For many years, the most interesting observation in schizophrenic patients has been the finding of enlarged ventricles, using CT head scans. In the present study, the total brain weight and the volumes of the hemispheres, cortex, and central grey matter were found to be significantly smaller and the ventricles significantly enlarged in this group of institutionalised, chronic schizophrenics. The only parameter not affected was the white matter.

The patients in this study were relatively old. Although they had been severely affected by their disease, it had had no substantial influence on their lifespan. In this respect it differed from many other diseases with serious brain disturbances (e.g. Alzheimer's disease, multiple sclerosis, Jacob–Creutzfeldt disease, Huntington's chorea). In contrast with these degenerative brain disorders, no specific changes (Alzheimer, multiple sclerosis), specific agents (Jacob–Creutzfeldt) or specific brain localisation (Alzheimer, Huntington's chorea) were found. The present findings were therefore not surprising, but confirmed the impression of a widespread effect on the central nervous system.

A positive correlation has been shown to exist between body height and brain weight; such a correlation was not found between body weight and brain weight (Pakkenberg & Voigt, 1964). For this reason all parameters in the two groups, except the ventricular volume, were compared by way of regression lines, plotted with body height on the x-axis and the brain volumes on the y-axis. The lack of correlation between body height and ventricular volume in the present study was unexpected (Gyldensted, 1977), but could be due to the rather small number of brains.

The metabolic investigations of schizophrenic patients have shown different results. An increase in dopamine receptor density assessed by spiperone binding has been found in post-mortem brains of schizophrenic patients (Owen *et al*, 1978). In a study on a small number of patients, dopamine receptor binding as measured with ^{11}C-methylspiperone and positron emission tomography (PET) gave no significantly different ratios for the schizophrenic patients (Herold *et al*, 1985).

Elevated brain D2 dopamine receptors have been found in both drug-naive and drug-treated schizophrenics using PET (Wong *et al*, 1986). Measurements of regional cerebral blood flow (CBF) have not been unequivocal (Nilsson *et al*, 1985; Goldstein *et al*, 1985), but there seems to be a trend toward decrease of CBF in frontal and prefrontal regions of schizophrenics (Geraud *et al*, 1985; Warkentin *et al*, 1985; Devous *et al*, 1985).

The procedure for obtaining CT scans can be standardised, but interpretation of the scans themselves presents considerable methodological problems. Jacoby & Levy (1980) have pointed out the difficulty of making accurate determinations of the ventricular boundaries from the scans (partial volume effect). They concluded that ratio measurements, which are most often used, are probably as useful as any other type of analysis. However, from the perspective of a field where gross anatomical changes demonstrated at autopsy are the *sine qua non* for validating radiologically observed changes, CT scan findings from schizophrenic patients should be verified by autopsy (DiChiro, 1983).

The present study has demonstrated enlargement of the ventricles in the brains from schizophrenic patients – an observation which accords with the conclusions of other investigators using CT head scans from schizophrenics (Johnstone *et al*, 1976; Andreasen *et al*, 1982; Nasrallah *et al*, 1982*a*).

The observation that the ventricles from Type II patients are bigger than those from Type I patients corresponds with the results of Andreasen *et al* (1982), who found that 16 schizophrenic patients with ventricular enlargement tended to have a preponderance of negative symptoms and impaired functioning on tests of the sensorium, while 16 schizophrenic patients without ventricular enlargement tended to have a preponderance of positive symptoms and a normal sensorium. This was not confirmed, however, by Nasrallah *et al* (1982*a*), who found in a study of 55 chronic male schizophrenics that large cerebral ventricular size in schizophrenia was not related to age at first admission to hospital, duration of treatment, pre-morbid adjustment, response to antipsychotic drug treatment, cognitive functioning, or the presence of positive or negative symptoms. In the present study, not all patients with the Type II syndrome had large ventricles, but the difference was significant for the total group of Type II patients compared with the Type I patients. The symptoms of the Type II syndrome patients remained more or less unchanged throughout life. This supports the view of Crow (1982), who points out that it is unusual for patients who have once acquired negative symptoms to lose them, perhaps because such symptoms are closely associated with structural changes in the brain. Weinberger (1984) suggests that the difference in ventricular size in schizophrenic patients could reflect pathology that is not qualitatively distinct but quantitatively different. Patients with CT findings may, thus, represent one extreme of a pathological continuum. It would then follow that the more pathology, the more CT evidence of it, and the poorer the prognosis.

Weinberger *et al* (1982) have shown that 7 out of 35 patients with first-episode schizophreniform disorders had cerebral ventricles larger than those expected for the second and third decades of life. Thus, the enlargement of the ventricles predates the acute illness, possibly by a period of years. The size of the ventricles differs considerably, not only in the schizophrenic brains but in the control group as well. Possibly, the size of the ventricles in an individual is a predisposing factor for the type of schizophrenia which develops. If the ventricular size is a predisposing factor, and an individual with normal-sized ventricles becomes schizophrenic, this patient, most probably, will present positive symptoms, and vice versa.

This does not limit the occurrence of atrophy to only some schizophrenics. The entire distribution of ventricular size for the schizophrenic brains shows a general upward drift. The same observation was made by Weinberger *et al* (1982) using CT head scans of 35 patients with schizophreniform disorders and 17 chronic schizophrenics. Atrophy thus seems to occur in most patients, and is simply more pronounced and has a greater probability of being observed in those patients with large ventricles before the onset of their illness. This could account for the observation, also in the present material, that some Type I patients develop negative symptoms during life. It does not explain, however, that in some cases patients with small ventricles make a schizophrenic debut with typical Type II syndrome symptoms, unless these patients had even smaller ventricles before the onset of the disease.

The two follow-up investigations on schizophrenic patients – Haug (1962) using pneumoencephalography on 31 patients after a period of 0.1–4.5 years, and Weinberger & Wyatt (1982) using CT scans on six patients over a period of 2–3 years – have not revealed any change in ventricular size. It would be of interest to have a longer follow-up period on young schizophrenic patients.

It has been pointed out (Jacoby & Levy, 1980) that patients over 50 years of age have a greater probability of presenting cerebral atrophy. This, combined with the fact that 62% of the schizophrenic patients were demented in old age, a condition associated with cerebral atrophy, could be a possible source of misinterpretation of results. However, no statistically significant difference was found between the volume of the cortex from the demented patients compared with the non-demented, given a $2P$ value of 0.86. The ventricles in the 13 demented patients did not differ in size from those in the rest of the schizophrenic brains.

Studies have shown that cerebral ventricles usually are smaller post-mortem than in life. Agonal pre-mortem events, coincident illness, brain oedema, and post-mortem extraction and fixation procedures may all obscure pre-mortem ventricular enlargement, especially if such enlargement is only modest (Messert *et al*, 1972). But still the volumes of the ventricles differed significantly in the schizophrenics compared with the controls.

There was no difference in the length of neuroleptic treatment in the Type I patients compared with the Type II patients. This supports the observation that the size of the ventricles does not have a great influence on the possible benefit of drug treatment. Weinberger *et al* (1980) found a significantly better response to neuroleptics in ten schizophrenic patients with normal ventricular size tested with Brief Psychiatric Rating Scale (BPRS) scores, compared with ten schizophrenic patients with enlarged ventricles. However, they concluded that a CT scan finding, no matter how dramatic, should never take precedence over a clinically indicated trial of therapy. Their clinical experience suggested that most patients with enlarged ventricles do benefit from drug therapy, and that this benefit may be clinically valuable, even if not reflected in a significant change in BPRS score.

In recent years the importance of inheritance has been discussed. An estimated 4.2–12.3% risk of schizophrenia in first-degree relatives of schizophrenic probands was found by Rosenthal (1970) and Slater (1938). In a study by Kety *et al* (1971), 33 adoptees with a diagnosis of chronic schizophrenia, acute schizophrenic reaction, or borderline schizophrenia were found among a sample of 5483 adoptees. A significantly higher than usual prevalence of schizophrenia-related illness was found among the biological relatives of adopted schizophrenics, but not among their adoptive relatives. As pointed out by Pope *et al* (1982), many studies, particularly American, have used a broad definition of schizophrenia, such as the DSM-II. Thus, many earlier familial studies of schizophrenia probably contained cases that would no longer be considered schizophrenic. Pope *et al* found no schizophrenic relatives in 199 first-degree relatives of DSM-III schizophrenic probands ($n = 39$), suggesting a zero prevalence. Abrams *et al* (1983) found in a group of 30 schizophrenic probands an age-corrected morbidity risk in first-degree relatives of 1.61%. They conclude that the case for familial transmission of narrowly defined schizophrenia is weak. Tsuang *et al* (1980) found a morbidity risk of 3.2% in first-degree relatives in a study of 200 schizophrenics.

In the present material, 9 out of 29 schizophrenics had affected relatives. Including both first and second-degree relatives, 17 had a schizophrenic diagnosis. No attempt has been made to trace back the affected relatives, since many died more than half a century ago. No personal interviews have been conducted with any of the relatives. The schizophrenic diagnosis is limited to the information given in the case records, and this could account for an over-representation. Yet the majority of these relatives spent their lifetimes in a psychiatric ward. This would tend to support previous reports of a genetic component in this condition, at least in severe cases of the disease.

Nasrallah *et al* (1982*b*), in a study of 42 chronic schizophrenics, found a difference in ventricular size, using bifrontal and bicaudate diameters on CT head scans, between paranoid and non-paranoid patients. Significantly more paranoid patients were found to have ventricles whose diameters exceeded the control mean by more than 1 s.d. In the present material, 18 of 29 patients had a diagnosis of schizophrenia paranoides. There was no significant different in the size of the ventricles in the two groups.

Thirteen patients developed dyskinesia. The symptoms appeared 1–26 years from the start of treatment. There was no relation between the size of the ventricles and the development of dyskinesia. Patients with large ventricles had no greater probability of developing this condition. In fact, only three patients with Type II syndrome developed dyskinesia. The size of the central grey matter was almost identical in the two groups.

According to previous studies of the normal population, there exists a positive correlation between ventricular size and age. Nybäck *et al* (1982), however, did not find such a correlation between lateral and third ventricular sizes and age, in their group of definite schizophrenics. They suggest that the normal age-related enlargement of the ventricular system may be obscured in some way in patients with schizophrenic psychosis.

In the present material, the same result was found. The size of the ventricles was significantly smaller in the younger control group than in the older group. This was not the case in the two schizophrenic groups: the lack of correlation was not only due to relatively wide ventricles in the younger patients, but also to relatively small ventricles in some of the older ones. Again, the atrophy that occurs in schizophrenics seems to appear early in the development of the disease.

The present study has shown significant reduction of almost every macroscopic parameter in a group of schizophrenic brains. However, this does not lead to a better understanding of the underlying microscopic changes in the brain. The dopamine hypothesis still represents the most lasting theory of schizophrenia, pointing toward a pathophysiological condition in the basal ganglia and limbic system. For this reason, more specified quantitative investigations of the limbic regions in post-mortem schizophrenic brains will be of interest.

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The Copenhagen High-Risk Project

The Diagnosis of Maternal Schizophrenia and its Relation to Offspring Diagnosis

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The Copenhagen longitudinal high-risk study of offspring of 129 schizophrenic mothers commenced in 1962. At that time, the mothers were diagnosed according to contemporary Danish criteria. We have re-examined all of the hospital records of these mothers: 108 (84%) fulfil present-day DSM-III criteria for schizophrenia and 95 (74%) were diagnosed as paranoid schizophrenic according to ICD-8 criteria. In a follow-up at mean age 24, the offspring of the paranoid schizophrenic mothers were themselves found to be less frequently schizophrenic (5%) than were the offspring of non-paranoid schizophrenic mothers (29%).

Longitudinal high-risk studies have long been advocated in the investigation of the aetiology and pathogenesis of schizophrenia (Mednick & McNeil, 1968), and a number of such studies are now under way (Watt *et al*, 1984). The earliest of these was initiated in Copenhagen by Mednick & Schulsinger (1965) in 1962 and is still continuing. The high-risk group of this study comprises children of schizophrenic mothers.

Strauss (1984) has observed that rigorous diagnosis of parental schizophrenia is critical to high-risk longitudinal research. Diagnostic uncertainty could compromise many years of work. A particular problem facing longitudinal studies is that of changing diagnostic criteria through time. In the years since the 1962 inception of the Copenhagen study, several systems of diagnostic criteria for schizophrenia have been proposed, with the aim of increasing the reliability of the schizophrenia diagnosis. Thus, according to the criteria listed in ICD-8 (World Health Organization, 1967), the diagnosis of schizophrenia is based on the presence of described typical symptoms and symptom patterns but it is not clearly operationalised. In contrast, DSM-III (American Psychiatric Association, 1980) does provide clear operational criteria for schizophrenia. The concept of schizophrenia in DSM-III is rather conservative, with emphasis on duration.

The purpose of the present study has been to perform DSM-III and ICD-8 rediagnoses of maternal schizophrenia in the Copenhagen high-risk study, and to examine the association of these maternal diagnoses with the diagnostic outcome of the offspring for whom follow-up diagnoses according to ICD-8 have already been made (Schulsinger, 1976).

Method

The high-risk sample was initially derived by identifying all women who were admitted to hospital for severe psychosis within the vicinity of Copenhagen, and who had children in the age range 10–18. Their hospital records were read by one of us (FS), who selected those meeting diagnostic criteria of schizophrenia. The criteria at that time followed those of the Danish Psychiatric Association and were not precisely operationalised, but they were anchored in the Bleulerian concept of schizophrenia; thus they relied heavily on the presence of autism and formal thought disorder. In addition to these psychopathological criteria, it was required that the mothers should have had either at least five years in hospital; or at least three separate periods in hospital, each of at least three months' duration, with no sign of improvement during discharge; or an extended period in hospital plus a certified State Invalid Pension for schizophrenia.

The present rediagnosis of the 129 mothers according to the criteria of DSM-III and ICD-8 was carried out in connection with an extensive collection of data concerning the mothers. Employing information from the Danish central psychiatric register (Dupont *et al*, 1974), available case records from all admissions of the mothers to psychiatric hospitals and departments up to 1975 were obtained. The median number of admissions was 6.3 and the median total time in hospital was 28 months. In almost all cases this constituted considerably more record information on the mothers than had been available at the inception of the project in 1962.

The records were read by the senior author, who completed a precoded data schedule while remaining blind with respect to the diagnosis of the offspring. The schedule contained items covering the following areas: family history of mental illness; childhood background; premorbid personality traits; social adjustment and psychopathology; course of disease; social adjustment during disease; treatment; response to treatment, including side-effects; and maternal functioning. The Syndrome Check List (Wing

TABLE I
ICD–8 and DSM–III diagnoses for the mothers

ICD–8 diagnosis	*DSM–III schizophrenia*		
	Positive	*Negative*	*Total*
Schiz. simplex	2	0	2
Schiz. hebephrenic	13	0	13
Schiz. paranoid	84	11	95
Schiz. catatonic	5	0	5
Schiz. pseudoneurotic	0	2	2
Schizoaffective	3	5	8
Schizophreniform psychosis	1	0	1
Other psychoses	0	3	3
Total	108	21	129

TABLE II
Maternal DSM–III schizophrenia diagnosis by offspring diagnosis

Offspring diagnosis (grouped)	*Maternal DSM–III diagnosis of schizophrenia*				
	Positive		*Negative*		*Total*
	n	*%*	*n*	*%*	
Schizophrenic	14	(9)	1	(4)	15
Borderline	25	(17)	4	(16)	29
Other	70	(46)	6	(25)	76
Not ill	42	(28)	13	(54)	55

et al, 1974) was used for scoring the psychopathology at each admission separately.

The mothers were diagnosed as schizophrenic according to DSM–III and/or ICD–8 if, at some time during their lives, they fulfilled the criteria of these diagnostic systems. For the ICD–8 diagnoses we used the *Glossary of Mental Disorders and Guide to Their Classification for Use in Conjunction with ICD–8* (World Health Organization, 1974). With respect to schizophrenia, such diagnoses are thereby equivalent to ICD–9. The ICD–8 diagnosis of schizophrenia was supplemented by a subtype diagnosis based on the symptomatology of the first phase of the schizophrenic disease.

In 1972–1974, a ten-year follow-up psychiatric assessment was undertaken of the 207 high-risk offspring of the 129 mothers, together with a matched control group comprising 104 subjects in whom neither the parents nor the grandparents were known to be psychiatric patients (Schulsinger, 1976). At that time, these offspring were between 18 and 30 years old (mean 24 years). On the basis of this assessment a clinical diagnosis was made by one of us (HS) according to the ICD–8 criteria. Since our present concern is with the relationship of the offspring diagnoses to the maternal diagnoses, the control group is not used here. For the purpose of statistical analysis the high-risk offspring diagnoses have been grouped into the following four categories: schizophrenia, borderline schizophrenia (corresponding to the DSM–III schizotypal personality disorder), other mental illnesses, and no mental illness. A total of 173 of the high-risk offspring completed the 1972–1974 assessment. To the 13 children diagnosed as schizophrenic on the basis of this assessment we have added two subjects who were already deceased at that time, but who could be posthumously diagnosed as schizophrenic on the basis of hospital record information.

Results

Table I shows the distribution of DSM–III (positive or negative for schizophrenia) and ICD–8 subtype diagnoses for the 129 mothers. Three mothers did not fulfil the schizophrenia diagnostic criteria of either DSM–III or ICD–8. According to the latter criteria, two of these suffered from paranoid states, while the third had a paranoid psychosis subsequent to amphetamine intoxication. On DSM–III criteria they were diagnosed as schizophreniform disorder, paranoid disorder followed by major depressive disorder at a later admission, and paranoid disorder respectively.

A further 18 mothers fulfilled the ICD–8 criteria, but not those of DSM–III. Of these, ten were diagnosed as atypical psychoses, with an onset of disease later than 44 years; but, this criterion apart, they all fulfilled the other DSM–III criteria for schizophrenia. The DSM–III diagnoses for the remaining eight mothers were: two schizophreniform disorders, two schizoaffective disorders, schizoaffective disorder followed by major depressive disorder, paranoid disorder, obsessive-compulsive disorder followed by atypical personality disorder, and schizotypal personality disorder. It is noteworthy that, according to the ICD–8 criteria, 74% of the mothers were paranoid schizophrenics.

The mean age at first admission to hospital for the 108 DSM–III-positive mothers was 31.4 years (s.d. 7.6), which was, not unexpectedly, earlier than for the 21 DSM–III-negative mothers (mean 39.8, s.d. 8.5) ($t(127)=4.5$, $P=0.001$). Similarly, the mean age at first admission to hospital for all ICD–8 non-paranoid schizophrenic mothers (simplex, hebephrenic, and catatonic) was 23.3 years (s.d. 4.04) which, again as would be expected, is very much lower than that for paranoid schizophrenic mothers (mean 34.4, s.d. 7.9).

Table II shows the relationship between maternal DSM–III diagnosis (positive/negative for schizophrenia) and diagnosis, grouped as described above, among the 175 offspring. The offspring of mothers diagnosed as schizophrenic according to DSM–III criteria were not themselves significantly more often diagnosed as schizophrenic (9%) than were the offspring of mothers not so diagnosed (4%) (Fishers Exact Probability = 0.70). This lack of significance may, however, be the result of the small numbers involved. The proportion of individuals free from mental illness among the offspring of the DSM–III-positive mothers (28%) is significantly lower than that among the offspring of DSM–III-negative mothers (54%) (Fisher's Exact Probability = 0.02).

Table III shows the distribution of offspring diagnoses as a function of maternal ICD–8 diagnosis. The offspring of non-paranoid schizophrenic mothers (i.e., simplex, hebephrenic, and catatonic) had the highest rate of schizophrenia (29%) and borderline schizophrenia (21%);

TABLE III
Maternal ICD–8 diagnosis by offspring diagnosis

Offspring diagnosis (grouped)	*Maternal ICD–8 diagnosis (grouped)*								
	Schiz. paranoid		*Schiz. non-paranoid*		*Schizo-affective*		*Other*		*Total*
	n	*%*	*n*	*%*	*n*	*%*	*n*	*%*	
Schizophrenic	6	5	8	29	1	8	0	0	15
Borderline	20	16	6	21	2	17	1	13	29
Other	60	47	9	32	4	33	3	37	76
Not ill	41	32	5	18	5	42	4	50	55

that is, 50% of all of these offspring were diagnosed within what may be called a 'schizophrenia spectrum' (Kety *et al*, 1978). Only 18% were free of mental illness. A much smaller proportion of the offspring of paranoid mothers were schizophrenic (5%). This number is significantly less than the corresponding rate among the offspring of non-paranoid schizophrenic mothers (Fisher's Exact Probability < 0.001).

There was no detectable tendency for the ICD–8 subtypes to 'breed true' between mothers and offspring. Of the 13 diagnostically interviewed schizophrenic offspring, only four were paranoid schizophrenics, and of these only one had a paranoid schizophrenic mother, compared with three among the remaining nine non-paranoid schizophrenic offspring (Fisher's Exact Probability = 1.00).

As mentioned above, maternal diagnosis is strongly related to age at first admission to hospital. We have previously shown early age of maternal admission to hospital to be predictive of poorer diagnostic outcome in offspring (Parnas *et al*, 1985). In order to explore the independence of the contributions of diagnostic subtype and age at admission to hospital, we have conducted a series of logistic regression analyses (Fleiss *et al*, 1986) employing diagnostic outcome in the offspring as a dependent variable, and maternal age at first admission to hospital and maternal diagnostic subtype (paranoid *v*. non-paranoid) as independent variables.

In the first analysis the offspring diagnoses were dichotomously grouped into schizophrenic plus borderline ($n = 44$) and other diagnoses ($n = 131$). In this analysis, both maternal age at first admission to hospital and maternal diagnosis subtype have significant chi-squared to-enter values ($\chi^2 = 6.77$, $P = 0.009$, and $\chi^2 = 10.13$, $P = 0.002$). Following forced entry, however, neither variable remained significant after the prior entry of the other. This indicates that the contributions of the variables are not statistically independent of each other.

In a second analysis we compared the contribution of the same two maternal independent variables with offspring diagnoses within the 'schizophrenia spectrum', by comparing the 15 schizophrenic offspring with the 29 borderline schizophrenic offspring. In this comparison neither predictor was significant.

Discussion

In the present study the rediagnoses of the mothers are based on hospital records; i.e. on information collected for clinical rather than research purposes. Hospital records are usually less structured and less comprehensive than information gathered for psychopathological research purposes. In fact, it is not infrequent that clinical information, including discharge diagnosis, is based on the immediate presenting picture at admission, rather than a thorough and comprehensive evaluation of the psychopathology (Parnas & Teasdale, 1987). This might call into question the reliability of the rediagnoses presented here.

Counter to these considerations, however, in 1962 the sample of 129 mothers was selected precisely because their hospital records contained sufficient information to confidently diagnose schizophrenia according to a rather narrow concept of the disease, with emphasis on chronicity. In addition, from 1962 to 1975 the great majority of mothers had several further admissions, often for long periods of time and to different psychiatric departments, yielding a fuller psychopathological picture.

For the diagnosis of schizophrenia, DSM–III requires that the patient's age at onset should be less than 45 years and that the duration of the illness should be more than six months. Estimation of disease onset and duration is necessarily made retrospectively. In the present study, however, no seriously problematic cases were encountered. The very large majority of the mothers were admitted to hospital with a schizophrenic breakdown before the age of 45, and the disease had a chronic course. In view of the potential diagnostic difficulties to be encountered by longitudinal high-risk studies (Strauss, 1984), it is reassuring to find that so high a proportion of the mothers meet the DSM–III criteria now widely used for research. Indeed, among the 21 who did not do so, ten fell only on the criterion of latest age of onset (i.e. 45 years). In the proposed revision of DSM–III, this limit has been raised to 50 years, which would have included nine of these ten mothers. The high level of agreement between the diagnoses made 24 years ago and the present-day DSM–III diagnoses is probably to be attributed to the joint emphasis placed by both diagnostic procedures on both psychopathological and social functioning criteria.

Epidemiological evidence suggests that paranoid schizophrenia is less common than non-paranoid forms (Fuller Torrey, 1981). There may be two reasons why, in the present study, almost three-quarters of the mothers were of the paranoid subtype. Firstly, perhaps because of the later age of onset, the reproductive capacity of this subtype is higher than for all other subtypes, except the schizoaffective (Reisby, 1967; Larson & Nyman, 1973; Erlenmeyer-Kimling, 1978). Consequently, in a sample of schizophrenics selected as being mothers, the paranoid subtype would be particularly well represented. Secondly, the diagnosis of schizophrenia based on hospital records might favour the selection of patients with reliably identifiable productive paranoid symptoms.

Our difficulty in distinguishing the relative influences on offspring outcome of maternal schizophrenia subtype and age at onset arises from the well-established strong association between these two. It is conceivable that the disintegration and disruption of psychic structures seen in non-paranoid schizophrenics (Meissner, 1981) arise precisely because the schizophrenic process afflicts the individual early in his or her life.

The disparity in the prevalence of schizophrenia among the offspring of non-paranoid early-onset mothers versus late-onset paranoid mothers – 29% versus 5% – is surprisingly high compared with that reported elsewhere (Kendler & Davis, 1981). The offspring were diagnosed at a mean age of 24 years, which is rather early in the risk period for schizophrenia, and further schizophrenic cases are to be expected. However, it seems unlikely that such a disparity will be evened out, given that the overall morbidity risk for schizophrenia in the offspring of a schizophrenic parent is estimated to be 10–15% (Gottesman & Shields, 1982).

Based upon earlier findings from our study, we suggest that the disparity could be due to: (a) a greater specific genetic loading for schizophrenia in the offspring of early-onset non-paranoid mothers (Parnas *et al*, 1985), further compounded by assortative mating with schizotypal fathers (Parnas, 1985), and (b) both non-specific genetic and environmental factors effecting the penetrance of the specific genetic loading; for example, birth complications (Parnas *et al*, 1982), early rearing experiences (Parnas *et al*, 1985), and other maternal personality and social characteristics (Talovic *et al*, 1980).

Familial homotypy for schizophrenia subtype has been found by Kallmann (1938), Ödegaard (1963), Farmer *et al* (1983), and Scharfetter & Nüsperli (1980), although not by Tsuang *et al* (1980). Irrespective of how many more schizophrenia cases ultimately manifest themselves in our study in future follow-ups, we anticipate that some degree of homotypy will emerge and that those with paranoid schizophrenic mothers will themselves principally develop paranoid schizophrenia.

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A Long-term Follow-up Study of Schizophrenia in Japan – with Special Reference to the Course of Social Adjustment

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A 21–27-year follow-up was conducted on 140 schizophrenic patients consecutively discharged from the Department of Neuropsychiatry, Gunma University Hospital, between 1958 and 1962, and 130 patients (93%) were successfully evaluated. Of these patients, 105 were still living, and the longitudinal courses could be fully observed in 98. With regard to the social outcome of the 105 patients, 47% were assessed as 'self-supportive' whereas 31% were 'hospitalised'. As for psychopathological outcome, 31% were 'recovered', 46% were 'improved', and 23% were 'unimproved'. In the studies of the longitudinal courses of the 98 patients, fluctuating courses of social adjustment were the most numerous in the early stages, whereas in the later stages many of the patients showed differentiation in one of two directions, namely the 'stable self-supportive' state and the 'chronic institutionalised' state. However, this form of differentiation was not revealed in the distribution of psychopathological 'end states'.

This paper reports a longitudinal study of schizophrenic patients which was conducted over a period exceeding 20 years.

In 1958, a five-year project aimed at preventing the relapse of schizophrenic patients was initiated at the Department of Neuropsychiatry, Gunma University Hospital, Japan by the professor at that time, H. Utena. The subjects were 140 schizophrenic patients consecutively discharged from the hospital between 1958 and 1962. The purpose of the project was to clarify prospectively the degree to which relapse could be prevented by placing the patients under what were then the latest therapies, such as neuroleptic drugs, the open-door system, and intensive aftercare. The results of the project, which was carried out until 1967, were reported by Eguma (1962), Utena (1965), and Yuasa *et al* (1969). These reports revealed that the social adjustment of the subjects had clearly been improved. However, as far as relapse was concerned, the results were found to be rather unfavourable. During the period of the project, the guiding principle of rehabilitational activity known in Japan as *Seikatsu-rinsho* was formulated by Eguma, Yuasa and others (Katoh *et al*, 1966; Tajima *et al*, 1967). Literally, this means 'clinical work in a patient's everyday life', but the term 'living skills development procedure' seems to express the meaning more accurately, and it seems to correspond roughly to a combination of individual counselling and social casework in Western methods of management.

The subjects of the project were subsequently followed up and their courses and social outcomes over a period of 16–22 years were reported by Miya *et al* (1984).

The present paper deals with the long-term courses and social and psychopathological outcomes of the same subjects over a period of 21–27 years.

Method

Subjects

The subjects were 140 schizophrenic patients (67 males, 73 females) consecutively discharged from the Department of Neuropsychiatry, Gunma University Hospital, between 1958 and 1962.

Classifying the subjects at the present time according to the ICD-9 (World Health Organisation, 1978), 84 cases are 295.1 (hebephrenic type), 20 are 295.2 (catatonic type), and 36 are 295.3 (paranoid type). As revealed above, the original selection of the subjects at the time of discharge was based on the narrower concept of schizophrenia.

Of the 140 subjects, 81% were under 30 years old, 79% were first admissions, 91% were prescribed neuroleptic drugs during the period of index admission, 87% had terms of index admission of less than six months, and 82% were 'recovered' or 'improved' at the time of leaving the hospital. Also, almost all the subjects were living in or close to a provincial city, Maebashi, about 100 km north-west of Tokyo. Almost all belonged to the middle social class. Population mobility during the follow-up period was not remarkable.

TABLE I
Eguma's Social Adjustment Scale (ESAS)

Self-supportive
1. Has returned to a level of social functioning similar to that prior to onset of illness
2. Maintains an independent social life with or without asking any advice from psychiatrists or acquaintances
3. Maintains a normal family life (housewife, for example)

Semi-self-supportive
1. Displays vocational ability, with some occasional failures
2. Maintains a positive attitude towards work, but needs supervision and guidance
3. Maintains a normal life at home, but hesitates to return to the job held prior to onset of illness

Socially adjusted to family or community
1. Works when encouraged with continuous significant support from others
2. Needs more time before being ready to return to previously held job
3. Able to work continuously if the work is kept at a simple level

Maladjusted
Social adjustment not possible
1. Non-productive life (able to be cared for at home)
2. Anti-social (admission to psychiatric hospital necessary)

Hospitalised
In psychiatric hospital

Assessment

The investigation of the course and outcome of the subjects was concentrated mainly on the aspect of social adjustment in the same way as previous reports on the same subjects. However, this investigation also covered the aspect of psychopathology.

For the assessment of social adjustment, we used Eguma's Social Adjustment Scale ESAS; (Eguma, 1962) (Table I), which we had used in previous studies. We assessed the course of social adjustment of each case, month by month, throughout the whole term.

For the psychopathological assessment of the outcome, we used the criteria of 'recovered', 'improved' and 'unimproved' which had been used in assessing the outcome at the time of the patients' leaving hospital. For the psychopathological assessment of the course, especially in the last five years, we used the category of 'end state' (Bleuler, 1972). In the criteria mentioned above, 'recovered' means a state without positive schizophrenic symptoms, 'unimproved' refers to a state with remarkable positive and/or negative symptoms, and 'improved' means a state with mild positive and/or negative symptoms.

Information was collected in the following ways. We used data which had already been accumulated in previous studies; the results of analysis of clinical charts and nurses' records from the medical institutions involved; information from doctors, nurses, public health nurses working in the community, and caseworkers; interviews with patients' families, relatives, and acquaintances; and data provided by administrative agencies. Also, interviews were held with as many patients as possible (80%).

Our last follow-up in this study covered the period up to the end of January 1984.

Results

The follow-up was completed in 130 (93%) of the 140 patients (62 males, 68 females). Of the 130 patients, 105 (48 males, 57 females) were still living at the time of the study, and in 98 of these (45 males, 53 females), longitudinal courses were fully observed.

The observation period for the 105 living patients was in the range 21.1–27.2 years, the average being 23.6 years (the duration of illness was 21.3–47.0 years, the average being 26.4 years). The mean age of the patients was 48 years (range 36–70 years) at the end of the follow-up period.

Twenty-five patients (14 males, 11 females) had died during the follow-up period. Suicides accounted for more than half of the deaths (14 patients). Of the other deceased patients, six had died suddenly of unknown cause, one had died of acute cor pulmonale, one of general prostration, one of asphyxia, and we were unable to obtain information concerning the cause of death of the remaining two.

The following results are limited to the living subjects only.

Outcome

Social outcome

The social outcome at the end of the follow-up period, as assessed on ESAS, is shown in Table II. The outcome was regarded as favourable in 'self-supportive' cases, which constituted 47% of the 105 living subjects, and unfavourable in 'hospitalised' and 'maladjusted' cases, which totalled 34%. The cases in the middle grades of ESAS, namely 'semi-self-supportive' and 'socially adjusted to family or community', amounted to only 19%. It was found that many of the cases had become polarised into 'self-supportive' and 'hospitalised'.

TABLE II
Social outcome

Social adjustment	*Male*	*Female*	*Total (%)*
Self-supportive	24	25	49 (47)
Semi-self-supportive	4	4	8 (8)
Socially adjusted to family or community	5	7	12 (11)
Maladjusted	0	3	3 (3)
Hospitalised	15	18	33 (31)
Total	48	57	105 (100)

When the above outcome was substantiated from the aspects of occupational, marital, and residential status, the results were as follows. As to occupational status, 78 (74%) of the 105 patients were fully productive or productive to some extent. Of these 78 patients, 49 were 'self-supportive', 20 were outside a hospital although not 'self-supportive', and 9 were still 'hospitalised', in the sense that they were attending on a 'night-hospital' basis. Such 'hospitalised' patients accounted for 27% of all 'hospitalised' subjects. This was because of the difficulty in discharging these patients, who were not acceptable to their families or to alternative accommodations, of which there are few in the part of the country covered by the study.

As to marital status, of the 105 patients, 47 (45%) were married, 11 (10%) were divorced and 47 (45%) were unmarried. Among the 'self-supportive' cases, 82% were married; among the 'semi-self-supportive' and 'socially adjusted to family or community' cases, 15% were married; and among the 'maladjusted' and 'hospitalised' cases, only 8% were married.

As to residential status, of the 105 patients, 52 (50%) were living in their own homes, 20 (19%) were with parents or other relatives, and 33 (31%) were in psychiatric hospitals. Among the 'self-supportive' cases, 96% were living in their own homes, while among the 'semi-self-supportive', 'socially adjusted to family or community', and 'maladjusted' cases, only 22% were living in their own homes.

Psychopathological outcome

The psychopathological outcome at the end of the follow-up period is shown in Table III according to the criteria mentioned above. There were 31% 'recovered', 46% 'improved', and 23% 'unimproved'. The number of middle-grade cases was the greatest, and the polarisation to two extremes as observed in the social outcome was not seen here.

TABLE III
Psychopathological outcome

Psychiatric state	*Male*	*Female*	*Total (%)*
Recovered	19	14	33 (31)
Improved	23	25	48 (46)
Unimproved	6	18	24 (23)
Total	48	57	105 (100)

Table IV shows the correlation of the psychopathological outcome with the social outcome. It can be seen that the 'improved' patients showed degrees of social adjustment ranging from 'self-supportive' to 'hospitalised', and that they accounted for one-third of the 'self-supportive' and one third of the 'hospitalised' patients.

Treatment

Of the patients alive at the end of the follow-up period, 69 patients (66%) were under psychiatric care. This high percentage may be due to the fact that there were many patients in whom relapse had occurred, resulting in resumed, and subsequently prolonged, treatment; 78% of patients had been readmitted one or more times. Almost all the patients (96%) under psychiatric care were taking neuroleptic drugs.

Even among the 49 'self-supportive' cases, 20 (41%) were under psychiatric care. Many of these cases had mild psychiatric symptoms. Of the 'semi-self-supportive', 'socially adjusted to family or community', and 'maladjusted' cases (23 in all), 16 (70%) were under psychiatric care, and 7 had dropped out of psychiatric care.

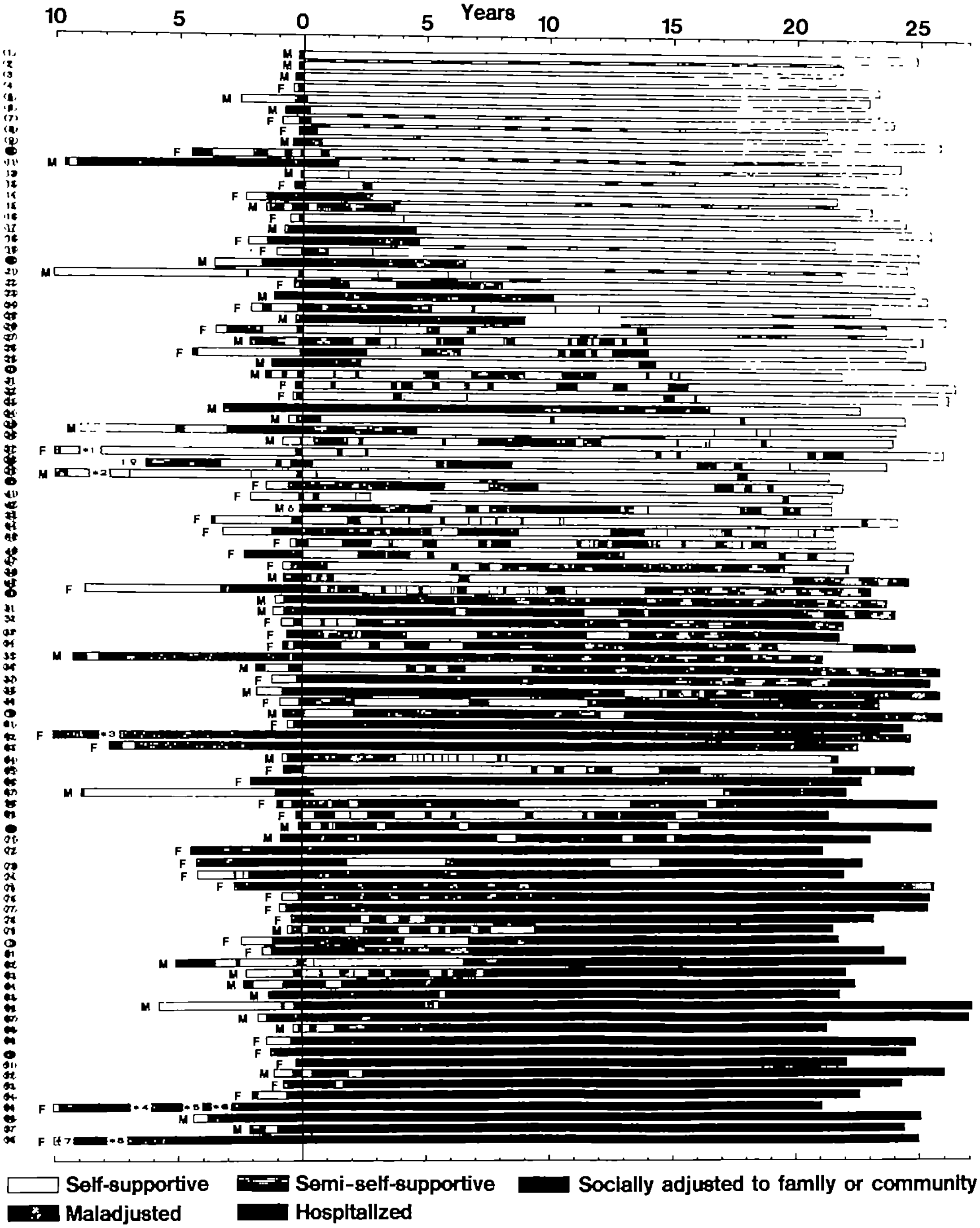
Course

Course of social adjustment

In 98 patients, the longitudinal courses were assessed and illustrated month by month using the five ESAS grades, as shown in Fig. 1, in which the passage of time is plotted

TABLE IV
Correlation between psychopathological outcome and social outcome

Social adjustment	*Psychiatric state*			*Total*
	Recovered	*Improved*	*Unimproved*	
Self-supportive	33	16	0	49
Semi-self-supportive	0	8	0	8
Socially adjusted to family or community	0	11	1	12
Maladjusted	0	2	1	3
Hospitalised	0	11	22	33
Total	33	48	24	105



*1-8: Abbreviations

Cases 19, 25, 41: The blank signifies an unidentified grade of ESAS

FIG. 1 The course of social adjustment.

TABLE V
Correlation between 'end states' and social course types

	Stable self-supportive		*Fluctuating and stable middle*		*Chronic institutionalised*		*Total*	
Subjects	35		32		31		98	
'End states': number of subjects	33	(94%)	9	(28%)	29	(94%)	71	(72%)
Recovery	23		0		0		23	
Mild chronic	10		4		4		18	
Moderately severe chronic	0		5		13		18	
Severe chronic	0		0		12		12	

horizontally, and the origin point is the start of follow-up. The illustrated courses for each patient were arranged in order from good to bad in terms of social adjustment as follows:

(a) 'Stable self-supportive' patients, who were in a long-term 'self-supportive' state for at least 5 years immediately prior to the end of the follow-up period, were arranged from the top in the order in which they reached this state.

(b) 'Chronic institutionalised' patients, who were in a long-term 'hospitalised' or 'maladjusted' state for at least 5 years immediately prior to the end of the follow-up period, were arranged from the bottom in a similar manner.

(c) The remaining patients were arranged below the category of 'stable self-supportive' in order from good to bad in terms of the social adjustment outcome at end of the follow-up period.

In Fig. 1, it can be seen that in the early stages of the long-term course, fluctuating courses were the most numerous, while in the later stages these courses gradually decreased in number and many of them differentiated in one of two directions, namely to the 'stable self-supportive' state and to the 'chronic institutionalised' state; out of the 98 patients, there were 35 of the former and 31 of the latter. The remaining 32 patients were termed 'fluctuating and stable middle' types; 25 of these were 'fluctuating' patients and 7 were 'stable middle' patients, who were in either of two long-term middle states in the ESAS for at least 5 years immediately prior to the end of the follow-up period.

Fig. 1 shows that the two curves obtained by plotting the initial points at which individual patients attained 'stable self-supportive' states and 'chronic institutionalised' states, respectively, were smooth and unbroken and in addition formed a symmetrical pattern.

This phenomenon was found to be the same even if the origin of the time plot in Fig. 1 was changed to the time of onset of illness or to the time of first admission. Therefore we called this phenomenon, in which fluctuating courses gradually decreased in number, and many differentiated in one or the other of two directions, 'the scissors phenomenon', as it resembled a pair of scissors with the blades open and facing in two directions.

Psychopathological course, especially 'end state'

For the psychopathological assessment of the course, especially the last 5 years, we used the category of 'end state' (Bleuler, 1972). Table V shows the result, and its correlation with the course of social adjustment. It can be seen that the proportion of patients who attained an 'end state' amounted to 72% (71 of 98). This proportion coincides closely with that (74%) of patients in a socially stable state (the 'stable self-supportive' state, the 'chronic institutionalised' state, or the 'stable middle' state).

As to the proportion of patients who attained an 'end state' in each social course classification, this amounted to 94% among the 'stable self-supportive' patients; and among the 'chronic institutionalised' patients, the same high proportion was observed. Among the 'fluctuating and stable middle' patients, nine (28%) attained an 'end state'. Seven of these nine were 'stable middle' patients, and only two had a fluctuating social course.

From these results, it was found that there is a close correlation between the 'end state' and the socially stable state.

The 'end states' were distributed as follows. In the 'stable self-supportive' type, most patients (70%) had recovered, and mild chronic 'end states' also occurred (30%). In the 'chronic institutionalised' type, 41% were severe chronic 'end states', but more (45%) were moderately severe chronic 'end states', and mild chronic 'end states' also occurred (14%). Overall figures were: recovery 32%, mild chronic 'end states' 25%, moderately severe chronic 'end states' 25%, and severe chronic 'end states' 17%. In the distribution of 'end states', the differentiation in two directions which was found in social adjustment did not occur.

Discussion

Our results on the outcome for schizophrenic patients showed that 47% were 'self-supportive' with regard to social adjustment, whereas 31% were 'recovered' with regard to psychopathological outcome. Of the 'self-supportive' patients, all were fully productive, almost all (96%) were living in their own homes, and the majority (82%) were married. But even among the 'self-supportive', 41% of the patients were under psychiatric care.

The results of earlier studies on the long-term outcome of schizophrenia in Japan indicated that drug therapy had no effect on the percentage of patients who recovered completely, although it

TABLE VI
Proportion and distribution of 'end states'

	Bleuler (1972)	*Ciompi & Müller (1976)*	*Huber* et al *(1979)*	*Present report*
Average follow-up term: years	22	36.9	22.4	23.6
Subjects	208	289	502	98
'End states': number of subjects	152 (73%)	262 (91%)	367 (73%)	71 (72%)
Recovery: percentage of end states	20	29	26	32
Mild chronic: percentage of end states	33	24	31	25
Moderately severe chronic: percentage of end states	24	26	29	25
Severe chronic: percentage of end states	24	20	14	17

reduced the number of patients with severe defects and increased the number with slight or moderate defects (Gotoh, 1971; Murakami, 1971; Shimazono, 1974; Aritome, 1978). However, as to the social outcome, a trend towards improvement in outcome has been noted; that is, the percentage of 'self-supportive' cases has increased as a result of intensive aftercare, including drug therapy (Yuasa, 1984). These findings are confirmed by results of our present study, which is a longer follow-up study than those mentioned above.

In other countries, several reports of follow-up studies of schizophrenia exceeding 20 years were published in the 1970s (Bleuler, 1972; Ciompi & Müller, 1976; Huber *et al*, 1979; Tsuang *et al*, 1979). Although it is difficult to make an exact comparison between these studies and our own, we have tried to do so on a limited basis, especially with regard to the percentages of cases in the 'self-supportive' state and those that recovered completely.

Bleuler (1972) performed a very careful follow-up study of 208 schizophrenics for more than 20 years and, according to his results, a social outcome of 'employable, outside a clinic', which seems equivalent to a status of 'self-supportive' in our study, accounted for 34% of the probands. This proportion was greater than the proportion (22%) of complete recoveries (the "undulating courses types progressing to recovery"). Studies by Huber *et al* (1979, 1980) and Tsuang *et al* (1979) produced similar results to those of Bleuler; in the study of Huber *et al*, the 'social remission degree 0' cases accounted for 39%, while the proportion of complete remissions was 22%; in the study of Tsuang *et al*, those having a rating of 'good' in 'occupational status' made up 35%, while 'good' patients with regard to 'psychiatric symptoms' accounted for 20%. The results of the study by Ciompi & Müller (1976, 1980) differed from these results in that only 15% of patients were 'good' in 'social adaptation', while 20% achieved 'total remission'. This seems to have been chiefly due to the advanced age of the probands (the average age was 74 years at follow-up). The above results from three studies, except those of Ciompi & Müller, are in agreement with our results in that the number of cases equivalent to 'self-supportive' was greater than the number of cases that recovered completely. However, in our results the percentage of 'self-supportive' patients is higher than that in the other three studies. This seems to be due to the fact that almost all our subjects had been under intensive aftercare, including neuroleptic medication from the early stages of the illness, whereas in the other three studies there were many subjects treated before the era of drug therapy. In other words, it is thought that neuroleptic medication, when used in conjunction with intensive aftercare, improves the long-term social outcome of patients with schizophrenia.

As to the course of social adjustment, our results showed that various courses were observed, but that overall, the characteristic pattern which we called 'the scissors phenomenon' occurred. The features of 'the scissors phenomenon' are:

(a) Transition from fluctuating courses to stable courses.
(b) Differentiation of the transition in two directions (one to the 'stable self-supportive' state and the other to the 'chronic institutionalised' state).
(c) Smooth, unbroken continuity of the transition.

Although the classification of the course types in schizophrenia has already been reported in many studies (the eight types of Bleuler, the eight types of Ciompi & Müller, and the 12 types of Huber *et al*, etc.), a fine, clear-cut distinction between subtypes was difficult to achieve in the present study.

Table VI shows the results for the 'end state' in the above three studies and our study. It is seen that as to the percentage of patients who attained an 'end state', our results coincide closely with those of Bleuler and of Huber *et al.* However, the percentage reported by Ciompi & Müller is higher than those in the other three studies. This seems to be due to the difference in the length of the follow-up term: the percentage of patients attaining an 'end state' seems to become higher when the course of the illness is longer and the age of each patient is higher. However, the difference in the length of the follow-up term does not influence the distribution of 'end states'. In particular, the results of all four studies are consistent in that the percentage of the middle grades (mild chronic and moderately severe chronic) of 'end state' is 50–60%. This differs remarkably from the distribution of socially stable states, in which differentiation in two directions was observed in the course of social adjustment.

We shall now deal with the relationship between 'the scissors phenomenon' and each course. It is necessary to pay attention to the fact that the cases in the 'stable self-supportive' state and in the 'chronic institutionalised' state, which increase in number gradually in the later stages of the course, change places to some extent with the cases in the 'fluctuating and stable middle' states. Although the 'stable self-supportive' and 'chronic institutionalised' states are not easy to change, they are not altogether unchangeable. For instance, in the last 5 years of the course, 6 (9%) of 64 patients in the 'stable self-supportive' and 'chronic institutionalised' states changed to the 'fluctuating and stable middle' states. However, in the same period, 12 (32%) of 38 patients in the 'fluctuating and stable middle' states changed to the 'stable self-supportive' or 'chronic institutionalised' state. Consequently, the number of cases in the 'stable self-supportive' and 'chronic institutionalised' states increases and 'the scissors phenomenon' progresses.

The present report deals with the course and outcome of schizophrenia as a whole. We are now analysing the other important issues involved in the prognosis of schizophrenia.

Conclusions

In the era of use of neuroleptic drugs, a trend towards improvement in the social outcome of schizophrenia has been recorded. This seems to be due to the effect of intensive aftercare, including neuroleptic medication from the early stages of the illness.

The percentage of complete recoveries with regard to psychopathological outcome seems to have remained at a similar level to that existing before the era of use of neuroleptic drugs. This shows the limit of these treatments. Furthermore, the percentage of cases with a favourable outcome without need of care still remains at a low level, and the length of the term spent under care tends to have been increased. These problems remain to be solved.

During the course of social adjustment, fluctuating courses in the early stages change on the whole to stable courses in the later stages. This is correlated with the increase in the number of patients who attain an 'end state' in the later stages.

The differentiation in two directions observed in the course of social adjustment is not seen in the distribution of 'end states' in psychopathology. This is characteristic of the course of social adjustment.

As to the 'chronic institutionalised' state, which is one of the two directions taken, this has again been brought to the fore as a problem to be solved, especially for those patients who remain in a psychiatric hospital even though their psychiatric condition is not very serious, making it apparent that social support systems should be established immediately in Japan.

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Mode of Inheritance as Indicated by One-sided and Two-sided Familial Taint: With an Application to Schizophrenia

ERIK ESSEN-MÖLLER and JAN LANKE

Among his various approaches to an understanding of schizophrenic inheritance, whether dominant or recessive, Rüdin (1916) also compared the respective frequencies of one-sided and two-sided familial taint. Essen-Möller (1949) tried the idea on another sample, but neither of us arrived at a definite choice. Later on Slater (1966) surprisingly demonstrated that unilaterality is typical not only of dominant but also of polygenic transmission. In the present paper we adjust Slater's computation and, in so doing, accentuate his result. We also find a similar preponderance of unilaterality on a recessive model, and offer an explanation for this. The findings substantially reduce the efficacy of laterality of taint as an indicator of mode of inheritance.

In a way this paper had its origin more than 40 years ago, when the families of 73 schizophrenic patients (see Appendix 1) were investigated by the first author (EEM) (who is sole author of this historical section). The purpose was to contribute to the contemporary discussion of whether schizophrenia, seen as a homogeneous genetic disease, was inherited according to a dominant or a recessive model.

The method consisted in studying the distribution of one-sided and two-sided familial taint. With dominant transmission, the illness was expected to occur, in most cases, on just one of the two ancestral sides; that is, either on the maternal or on the paternal side but not on both. Recessivity would imply more cases where the illness occurred on both ancestral sides.

The first to apply this line of thought was probably Rüdin (1916), in his classical work on the genetics of dementia praecox. Actually he found a moderate preponderance of unilaterality, but less so when a wider definition of the illness was applied. The preponderance was thought to be possibly due to incompleteness of his sample (which may not have been collected primarily with the problem of laterality in mind). So with due reservation Rüdin was inclined to assume recessivity, an opinion supported by conventional studies irrespective of side (Lundborg, 1913; Rüdin, 1916).

Some 25 years later the perspective had changed, and for various reasons it had become natural for me (EEM) to believe chiefly in dominant transmission.

For one thing, the concept of reduced penetrance had made the exact proportions within separate types of relatives less informative, and had shifted the interest to a comparison between the types. Now the children of schizophrenics were found to run approximately the same risk as did the sibs, a finding in favour of dominance (Lenz, 1921, 1937). Secondly, the exceptionally low incidence in parents of schizophrenics, for a long time a major argument for recessivity (Luxenburger, 1937, 1939), could now be explained as resulting from a reduced marriage rate in affected individuals, and such reduction was shown to occur even premorbidly (Essen-Möller, 1935, 1936; Lenz, 1937). Thirdly, it was recognised that the pattern according to which affected children of schizophrenics were scattered over families would require, on a recessive model, a much higher frequency of gene carriers among the probands' mates than was reasonable or could be verified by any substantial increase of taint in the families of mates (Schulz, 1938; Leistenschneider, 1938; Koller, 1939; Egger, 1942; Früh, 1943).

Accordingly, it was a surprise to me when my own sample, which had been collected expressly for a study of laterality, appeared to present a bilateral distribution and thus to suggest recessivity. I doubted my method of calculation, and after some time submitted the problem to an international congress of geneticists, asking for suggestions and advice (Essen-Möller, 1949). Eliot Slater, who was present, agreed that the method was probably too primitive for the purpose. Much like Rüdin, I had simply compared the morbidity in relatives of non-psychotic parents according to whether or not the other parent was psychotic. However, no better method was suggested, the problem remained unsolved, and my sample was laid to one side (although used in other connections).

The next opening came when a third model, the polygenic, caught interest within medical genetics.

To be sure, this model had already become accepted for certain normal traits with a quantitative variation, like stature and intelligence. The gradation of such traits from slight to pronounced was assumed to depend not on which particular polygenes were present, but rather how many. Depending on their contents of relevant polygenes, 'the genetic liability', the separate individuals were conceived to be distributed symmetrically around the population mean according to the well-known normal or Gaussian curve. In relatives pertaining to a defined proband, the liabilities would likewise be distributed normally, but their mean would be dislocated toward the proband in proportion to the degree of relationship.

Now several authors (e.g. Carter, 1958; Edwards, 1960; Crittenden, 1961; Falconer, 1965; Gottesman & Shields, 1967) suggested that qualitative and abnormal traits too, such as diabetes or schizophrenia, might be interpreted as polygenic provided there was a threshold, a critical number of abnormal polygenes, that had to be exceeded for the trait to have a chance of showing up, other conditions ('penetrance') permitting. Because of their dislocated distribution, the relatives of probands would surpass the threshold and get affected more frequently than do random individuals.

Although the polygenic threshold model might represent an adequate solution to various intricate problems, doubts were put forward regarding its generality (Morton, 1967), and specifically its application to schizophrenia (Shields & Slater, 1967; Dalén 1972; Essen-Möller, 1977, 1979). Thus even the polygenic model proved difficult to distinguish from dominance by means of conventional methods. So far the situation was reminiscent of the time when the alternative to dominance was recessivity. As once Rüdin (1916), so now Slater (1966) had the idea of probing whether a study of laterality of taint might help to elucidate the problem. To this end he developed a sophisticated procedure of his own, and subsequently quite a number of psychiatrists applied his results to their samples of functional disorder (Slater & Tsuang, 1968; Perris, 1971; Tsuang, 1971; Slater *et al*, 1971; McCabe *et al*, 1972; Mendlewicz *et al*, 1973; James & Chapman, 1975; Johnson & Leeman, 1978; Pauls *et al*, 1979; Baron, 1980; Rinieris *et al*, 1981). So I could not resist taking up the topic once more and reviving my hibernated sample.

To that end I associated with Jan Lanke, mathematician–statistician. He suggested a number of adjustments and improvements on Slater's strategy, and will give an expert presentation elsewhere; he also planned and carried through the extensive computations required for the data analysis.

Fortunately we had notified Eliot Slater about our project in the year prior to his death, and had his agreement in principle in two most encouraging and amiable letters. The preceding notes largely comply with his wish for a presentation of "the true history of the idea".

Theoretical considerations

Slater's strategy for predicting laterality of taint on a polygenic threshold model

Clearly there is a difficulty in analysing simultaneously both ancestral sides of all families in an entire sample of probands. This is so because of the varying number of relatives on either side and the varying number of affected individuals among them. At this point Slater had the idea of confining his computations to families with two affected relatives on the two sides taken together. When more than two affected individuals were present, he regrouped them into sets of two, in every possible combination, and subsequently compensated for the excess weight that this policy entailed in such families (Slater & Tsuang, 1968).

By this arrangement the number of affected individuals on either side could be only zero, one, or two. Out of the resulting nine combinations, only three would add up to the stipulated number of two affected relatives on the two sides taken together; namely, (a) no maternal and two paternal; (b) one on each side; (c) two maternal and no paternal. The first and the third of these three crucial combinations are seen to represent unilaterality, and the second, bilaterality.

Admittedly this formulation of the problem reduced substantially the number of families available for calculation. In exchange, it implied a striking simplification as compared with earlier attempts, where families with a single affected relative were also included.

With these ideas in mind, Slater started out by combining two Gaussian distributions, which were taken to represent the two sexes of an unselected population mating at random. This combined distribution is again of the Gaussian type, to be visualised now as a symmetrical hill. The pairs of mates were grouped together into numerous classes or cells, corresponding to the deviations of the two mates from their population means in units of standard deviations. The expected frequency (probability) of any cell is the product of the probabilities of the mates.

The mid-parent value of each pair of mates is also, according to the rules of population genetics, the mean liability of the children of those mates, or parents. The probability of such a child being affected is obtained as that proportion of the children's distribution which transgressed the threshold.

The three probabilities – one for each parent, one for the child – were multiplied together, and their product was stored.

Slater then proceeded, within every cell, from the mothers of affected children to the mothers' sibs, that is, to the maternal uncles and aunts of the affected children, which were now posited as fictitious probands. The distribution of these maternal uncles and aunts around their dislocated mean was computed, and so was the probability of their containing among themselves either none, one, or two affected individuals. The same procedure was accomplished on the paternal side.

Finally the probability for each one of the three crucial constellations of affected uncles and aunts pertaining to each cell (0+2), (1+1), (2+0) was weighted (multiplied) by the stored probability of the cell, and the products for all cells were then summed. The three resulting sums would constitute, according to Slater, the three requested odds (or probabilities) for uni- and bilaterality of the affected uncles and aunts on a polygenic threshold model.

Adjustments of Slater's procedure

Summation over cells. The summation over cells, described at the end of the previous section, was somewhat invalidated by a slip. From each of the three terms presented by Slater (1966, p. 160), a constant factor $n(1-p)^{n-2}(1-m)^{n-2}/2$ has been eliminated. When summing over all cells this does not work since the factor removed is different in different cells as soon as there are more than two relatives on each side. The error is easily corrected by not making the cancellation.

Standard deviations. While Slater correctly worked with dislocated means for the relatives of probands, he overlooked the fact that the standard deviation of relatives is smaller than that of the unselected population. In proportion to the degree of relationship (correlation) with the proband, this reduction in standard deviation slightly restricts the risk for a relative to transgress the threshold and get the disease. It is the joint effect of dislocation and reduction in standard deviation that has to be considered.

Dependence between relatives. Slater conceived the nine aforementioned combinations of uncles and aunts as the products of two binomial expansions, one for each side of the family. For instance, in the case of two maternal uncles, he would put the probability for both of them to be ill equal to the square of the probability that relates to just one of them, and correspondingly on the father's side. However, the probability of two ill on the same side is greater than that square, indeed considerably greater. This follows from the correlation which prevails between the relatives. The phenomenon is in principle the same as that referred to by Edwards (1960) when he says that the risk to the unborn increases with the number of relatives ill (cf. Ødegaard 1972; Essen-Möller 1977). Of course the correlation also affects the remaining terms of the presumed binomial expansions. The uncorrected binomials simply do not apply in the situation. For an adequate computation, we built the appropriate correlation into each of the two distributions.

The three adjustments combined. In his polygenic model, Slater posited the threshold at $+2\sigma$. At full penetrance, where phenotype equals genotype, this implies, according to tables of the normal distribution, that 2.28% of the population would be affected. When applied to families with exactly two affected uncles and aunts on the two sides taken together, and (about) ten uncles and aunts overall on each side, Slater computed from these assumptions the odds 4675:4193:4675 for the three crucial constellations of laterality. In small families (two members on each side) the proportion of the bilateral group was doubled. In terms of probabilities of unilaterality this equals 0.6904 in large families and 0.5272 in small ones. Making all three adjustments described above increases these probabilities to 0.7960 and 0.7796, respectively.

Probability of unilaterality on different genetic models

Slater and followers (see Slater & Tsuang, 1968) were prepared to decide in favour of dominance when the observed frequency of unilaterality was significantly higher than the calculated polygenic expectation. Now we supplement this indirect inference by introducing a direct comparison with calculated dominant expectation, and, for completeness, also with recessive expectation, still with 2.28% genotypical population frequency and full penetrance. The resulting probabilities in families of different size are presented in Table I.

As could be foreseen, the dominant model had the highest probabilities of unilaterality, the polygenic the lowest, while recessivity held an intermediate position.

TABLE I

Expected proportion of unilaterality in large and small families on different genetic models (genotypic population frequency 2.28%, penetrance 1.00)

	Polygenic	*Recessive*	*Dominant*
Large family (10+10)	0.7960	0.9820	1.0000
Small family (2+2)	0.7796	0.8156	0.9642

The span between the three models is strikingly narrow, and in view of the sample size required for a significant genetic differentiation, it appears doubtful whether the study of ancestral laterality will prove as helpful as had been hoped.

Unilaterality is seen to predominate throughout the table. It is unequivocally the most probable finding whatever the mode of transmission. This is in striking disagreement with the intuitive concepts with which Rüdin, Essen-Möller, and Slater started their investigations. It is true that certain corrections had to be performed with regard to Slater's computations, but this did not restore the bilateral distribution that Slater had anticipated with polygenic transmission; rather, we ended by supporting and accentuating Slater's discovery that unilaterality is typical not only of dominant but also of polygenic transmission, and we also added a similar finding for recessivity.

Obviously there is reason for looking closer into the nature of this apparent mystery. The following theoretical explanation is offered with regard to the recessive model.

Recessive unilaterality explained

The fact that the proband is homozygous implies that the abnormal gene must be present in both parents, and hence in one of the maternal and one of the paternal grandparents as well. Let us denote as 'loaded' the two grandparents that have provided the proband's parents (and subsequently the proband) with the gene in question; the two remaining grandparents may be characterised as 'free'. From the loaded grandparents the abnormal gene may spread also among the proband's uncles and aunts, and this will largely occur to the same extent on both ancestral sides. This is what might seem at first sight conducive to bilaterality.

However, one single recessive gene does not produce the illness. Should it so happen that the illness is actually displayed in an uncle or aunt of the proband, then a complementary additional gene of the same sort must be present in the free grandparent on the relevant side. This additional gene stems from the population, where it has a certain frequency. If the gene is a rare one, the chances are low that both of the free grandparents possess it, and bilateral illness among uncles and aunts is therefore infrequent. It is much more common that an additional gene occurs in just one or the other of the free grandparents, and this implies a comparatively high frequency of unilaterality among the affected uncles and aunts.

Let us put it this way. The proportion r of abnormal recessive genes among all genes at the same locus is the square root of the frequency r^2 of the homozygotically abnormal individuals in the population; for example, 10% if the population frequency is 1%. The frequency of the carriers of the abnormal gene (whether hetero- or homozygotic) is $2r(1-r)+r^2$; in this example 19%. Accordingly, the probability of the abnormal gene occurring bilaterally, that is in both of the free grandparents, is $0.19^2 = 0.0361$. In contrast, for the abnormal gene to occur unilaterally, that is either on the maternal or the paternal side but not on both, the probability is $2 \times 0.19(1-0.19) = 0.3078$. This means that the odds for unilaterality are about 8½ times those for bilaterality. With a genotypical population frequency of 2.28%, the odds for unilaterality would be about 5:1.

One might venture to speculate that a similar mechanism, if more complicated, is at play in polygenic transmission.

Empirical findings

Observed and expected unilaterality

Having dealt so far with computations in theoretical models, we shall now turn to the actual schizophrenic sample referred to in the historical section and described in Appendix 1.

For every observed family with two or more affected relatives on the two ancestral sides taken together, a hypothetical family with the same number of paternal and maternal members and the same number of affected individuals was constructed. Within this hypothetical family, the expected frequency of unilaterality was computed and compared with observation. The comparison was made both with and without regard to certain background variables of conceivable influence, such as sex, age and length of observation (see Appendix 1), and of course under various assumptions regarding genetic model, genotypic frequency, and degree of penetrance. The comparison was also

TABLE II

Observed laterality by diagnostic classification

Uncles and aunts			*Uncles, aunts and parents*		
Cumulated diagnosis	*Number of unilateral families*	*Total families*	*Cumulated diagnosis*	*Number of unilateral families*	*Total families*
D1	–	–	D1	3	3
D2	2	3	D2	6	8
D3	3	5	D3	9	12
D4	8	12	D4	13	20
D5	10	18	D5	13	25

performed with and without inclusion of the parents. The entire procedure was finally repeated within each of the five extensions of the diagnostic classification, as described in Appendix 1 and Table II. When, for instance, the D5 criteria were used, there are 18 families with two or more affected uncles and aunts and 25 families with two or more affected members including parents. In 10 and 13 of these families, respectively, the affected members are all on one side, and in the remainder they occur on both sides.

The voluminous crosswise tabulations cannot be presented here. A brief exemplification is offered in Tables III and IV. Table III shows, that, for instance, when the D3 diagnosis was applied, there were five families with two or more ill relatives, three of which were affected unilaterally and two bilaterally. The expected number of unilateral families was 3.74. Table IV gives the expected frequencies of unilateral disease (D3) for each of the three genetic models, when the genotypic population frequency is put at 5%, 2.5% and 1% and the penetrance at 0.2, 0.4 and 1.0, respectively.*

The closest *proximity* between observed and expected *frequency* of unilaterality was found, with rare exceptions, on the polygenic model. Recessivity came almost as close, while dominance was slightly more distant. Exclusion or inclusion of other variables, such as parents, age, and length of observation, did not appreciably influence this order of proximity between the models.

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*The combinations of genotypic population frequency and degree of penetrance were chosen so as to comply approximately with the commonly accepted 1% phenotypic incidence of classical schizophrenia. The combination 2.5% × 0.4 (which is close to Slater's assumption) would also go well with the gap between 25% for the genotypic expectation and 10% for the phenotypic observation in the sibs of schizophrenic probands on a recessive model. Similarly, the combination 5% × 0.2 might exemplify the corresponding gap in the proband sibs under dominant transmission (50% as against 10%).

TABLE III

Frequencies and likelihoods of unilateral and bilateral disease in a diagnostic subsample of five families of varying composition (Diagnosis D3; polygenic model; genotypic frequency 2.5%; penetrance 0.40 (sex, age, length of observation, and parents not considered))

	Number of uncles and aunts			*Unilateral sets of affected individuals*		
Family code	*Paternal*	*Maternal*	*Affected*	*Observed*	*Expected*	*Likelihood*
35	6	3	2	1	0.8114	0.8114
45	5	8	2	0	0.8080	0.1920
46	5	3	2	1	0.7996	0.7996
64	8	8	3[1]	1	0.7050	0.7050
73	8	8	4[1]	0	0.6186	0.3814
				3	3.7426	0.0335

1. Instead of regrouping into sets of two the families with more than two affected relatives, it was here preferred to keep together the total number of affected individuals, and to accept such a family as unilateral only when all of its affected members occurred on the same side. Obviously this method, too, complies with Slater's principle of three crucial and mutually exclusive alternatives. The difference is of no practical avail.

TABLE IV

Expectations of unilaterality and likelihoods of observed lateralities (same diagnostic subsample as in Table III; variable genetic model, genotypic frequency, and degree of penetrance)

	Assumed genotypic frequency and penetrance		
Genetic model	*5%, 0.20*	*2.5%, 0.40*	*1%, 1.00*
	Expected number of unilateral families[1]		
Polygenic	3.35	3.74	4.05
Recessive	3.29	3.81	4.63
Dominant	4.08	4.70	4.99
	Likelihoods		
Polygenic	0.0441	0.0335	0.0227
Recessive	0.0437	0.0305	0.0041
Dominant	0.0223	0.0028	0.0067

1. As against 3 observed.

Likelihoods. The likelihoods, as shown in the last column of Table III and in the bottom part of Table IV, are the cumulated products of such probabilities of uni- and bilaterality as were realised among the families of a subsample. With few exceptions, the maximal likelihoods were found to be associated with the polygenic model, although recessivity gave likelihoods not far from the maximal in most cases. Once again, the background variables influenced the results only marginally.

Conclusion

A high level of unilateral taint prevails among the uncles and aunts of affected probands, and this is typical not only of dominant but also of polygenic and recessive transmission. These results clearly have a bearing beyond the concept of schizophrenia – if the latter can, indeed, be regarded as a genetical unit.

Appendix 1 (EEM)

Psychiatric sample

The sample was drawn from the first admissions through years 1927–1941 to a new mental hospital from a defined rural area of South Sweden. About one fifth of the patients had been transferred from other mental hospitals or had a history of earlier episodes with or without hospital admission. In spring 1942, I (EEM) selected those patients whose record, either original or continued, seemed to justify a diagnosis of classical schizophrenia. In order to facilitate the collection of family data, the last third of the sample was restricted to patients born 1902 or later, and with both parents living at the time of the field work.

The field work took place in the summer of 1942 and 1943. I managed to get information regarding 68 families; five of them contained two probands each, and so there were 73 probands. The families were investigated in geographical order so as to avoid subjective selection. I used to visit as many informants as was necessary for feeling reasonably confident regarding the presence or absence of a history of mental illness or of a major personality disorder in any one of the 601 uncles and aunts and the 144 parents (two fathers were unknown). Altogether more than 400 individuals served as informants, ranging from 3 to 12 per proband. Most of them were relatives or relatives-in-law from both sides of the family, and quite frequently they proved informed also regarding the side opposite to their own. The informants were chosen largely by availability, and the visits were made without notice. Out of the 353 living uncles and aunts, 114 were among the informants, and out of the 97 living parents, 93. As usual in rural populations, collaboration was mostly good and engaged.

Classification

I am indebted to three colleagues and friends, Eliot Slater of London, Torsten Frey of Uppsala, and Eberhard Nyman of Lund, for giving (1977) each separately their classification of the parents. This was intended as a test of my own classification of parents, uncles and aunts.

A twofold classification was used, one of personality and the other of mental illness. Only the latter has been considered sufficiently reliable to be included in the present paper, as follows:

D1. Classical schizophrenic psychosis or defect.
D2. Other psychosis suggestive of genetical kinship with schizophrenia (on the whole more acute, confusional, or emotional).
D3. Other functional psychosis (e.g. depressive or unspecified, also suicide with no further information).
D4. Mild functional mental illness (with preserved reality testing, 'neurosis').
D5. Mental illness mild or severe, of obvious or probable organic origin (e.g. old age dementia, alcoholic delirium, or following infection or childbearing).

The diagnoses were cumulative in the sense that every diagnostic group was successively included in the next group. Within each cumulated group all affected individuals were treated as if they had the same diagnosis, the most peripheral one from point of view of the schizophrenic spectrum being chosen. Table II shows the number of unilateral out of total families in each group.

Morbid risk

For the calculation of morbid risk (morbidity), the start of individual observation was put at the 15-year birthday for all uncles and aunts. For parents, the start of observation was at the age when they became parents of the respective proband (Weinberg, 1913; Essen-Möller, 1955, 1963). In exceptional instances where the onset was prior to parenthood, the age at onset was for convenience 'postponed' correspondingly. Otherwise the parents were treated as were the uncles and aunts. The end of observation was when the individual was last heard of.

In every five-year age group of relatives, the number of first incidences (relapses were not considered) was divided by the total time under risk in that age group. The rates (R) were converted into probabilities (P) according to the formula $P = 1 - e^{-Rw}$ where e denotes the base of the natural logarithms (2.718) and where w is the width of the age interval. The complements of these probabilities were successively multiplied through the age groups, and, in turn, the complements of the products indicated the cumulated morbid risks up to the lifetime risk (see Hagnell *et al*, 1981).

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Depressive Syndromes in the Year following Onset of a First Schizophrenic Illness*

ALLAN HOUSE, JANET BOSTOCK and JOHN COOPER

A complete, unselected series of 68 patients who were seen during their first episode of an undoubtedly schizophrenic illness, and followed up one year later (for 56 patients) is described clinically. Depressive symptoms were common at onset, and 22% of patients could have been considered cases of depression from these symptoms alone. At follow-up, depressive symptoms had reduced in prevalence and only 7% of subjects were depressed cases. Only two cases of depression at follow-up had not been cases at onset. These changes could not be attributed to the use of antidepressants or ECT. Depressive syndromes could be distinguished from akinesia and the negative syndromes. The findings indicate that depression cannot be attributed solely to the administration of neuroleptics.

Depressed mood is a frequent finding in the aftermath of an acute schizophrenic episode, even in the absence of first-rank symptoms, a state often known as *post-psychotic depression* (McGlashan & Carpenter, 1976). Until recently, it has been widely accepted that neuroleptic medication is one important aetiological factor in what has come, therefore, to be known as *pharmacogenic depression*. It has been suggested that this 'post-treatment depression' may reflect a specific underlying pathophysiology in certain cases of schizophrenia (Galdi, 1983). Several detailed reviews of these issues have appeared recently in which the view of depression in schizophrenia as following the acute illness and being in some way tied up with its treatment or chronic sequelae has been challenged, most notably by Hirsch (Knights & Hirsch, 1981; Hirsch, 1982, 1984). These studies indicate that substantial levels of depression are found in first-onset acute schizophrenic illnesses and become less severe with the institution of neuroleptic medication. Relatively few patients have been shown to develop depressive syndromes *de novo* on neuroleptic medication.

The studies quoted by Hirsch are substantial, and their results are clear, but they can be criticised for the diagnostic criteria used, for relatively short follow-up periods, and most importantly for case selection. It may not be appropriate to consider relapses in chronic schizophrenia together with new acute schizophrenic episodes, or to take patients participating in a drug trial as representative of all schizophrenics.

Other problems in the phenomenology and classification of schizophrenia have been raised in the debate (Johnson, 1986). It has been suggested, for example, that the depressed mood is not strictly comparable to that found in the primary affective disorders but is really a variant of an extrapyramidal syndrome, so-called *akinetic depression* (Van Putten & May, 1978). The degree to which depression and akinesia can reliably be separated from each other or from the negative syndromes of slowing, affective blunting, social withdrawal and self-neglect is also a matter of some dispute.

It has been suggested that where insufficient attention is paid to diagnostic criteria there is over-inclusion of patients who would be better described as schizoaffective (Galdi, 1983), although this seems to beg the question of the nature of mood disorder in schizophrenia.

A recent review has emphasised the need for further research in all these areas (Johnson, 1986). The aim of the present study is to provide a detailed clinical description of an unusually complete group of patients seen during their first onset of an undoubtedly schizophrenic illness. Information obtained at onset and one-year follow-up is used to examine

(a) the prevalence of major mood disorder as part of the first illness in patients diagnosed as suffering from definite schizophrenia
(b) the natural history of any such mood disorder over the year of the follow-up, and in particular whether it is the same individuals who have mood symptoms at onset and at follow-up
(c) the degree to which depressive symptoms may be distinguished from akinesia and negative syndromes

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*A report from the UK Field Research Centre of the World Health Organization Collaborative Study on Determinants of Outcome of Severe Mental Disorders.

(d) the relationship between depressive symptoms and the administration of neuroleptic medication.

Method

The 68 subjects were derived from a larger series of 99 patients making their first contact with Mapperley Hospital for a psychotic disorder as defined for the WHO collaborative study on Determinants of Outcome of Severe Mental Disorder (Cooper *et al*, 1987). The aim of the study was to identify and assess, in each of the catchment areas for a two-year intake period, individuals who met the following criteria:

(a) age 14–54
(b) residence of at least six months in the area in the year preceding the initial examination
(c) evidence of presence, in the preceding 12 months, of *at least one* of the following overt psychotic symptoms; hallucinations or pseudohallucinations in any modality; delusions; qualitative thought or speech disorder; qualitative psychomotor disorder; gross behavioural abnormalities representing a break in the person's previous pattern; a definite change of personality and behaviour during the last year, manifest in any of the following: loss of interests, initiative and drive leading to deterioration of performance; onset of social withdrawal, episodic severe excitement, purposeless destructiveness or aggression; episodic or persistent states of overwhelming fear or anxiety; gross and persistent self-neglect
(d) absence of clinical evidence of: organic cerebral disorder; CNS damage due to alcohol or drug abuse, manifest in encephalopathy, with or without polyneuritis
(e) first-in-lifetime contact with the defined psychiatric services of Nottingham within the last three months, occasioned by problems associated with the symptoms and behaviours enumerated in (b).

All patients who fulfilled the entry criteria and made contact within a two-year period were seen and assessed using the following instruments:

(a) Present State Examination (ninth edition) (PSE-9)
(b) Psychological Impairments Rating Scale (PIRS); this is an expanded and improved version of the behavioural items from PSE-9; it contains items relevant to behaviour, speech and interaction, covering such areas as body language, affect display, and facial expression (Jablensky *et al*, 1980)
(c) Disability Assessment Schedule (DAS); this is a social disability schedule designed for use by a trained interviewer and completed with the help of a near relative (Jablensky *et al*, 1980)
(d) Psychiatric and Personal History Schedule (PPHS); on this schedule is recorded basic demographic data, details of the presenting history, duration of symptoms and past medical history.

All patients were seen within 14 days of contact for the index illness and, where possible, within four weeks of the one-year follow-up date. A clinical diagnosis was made for each patient at entry to the study, and a current 'cross-sectional' diagnosis was made at the time of each of the one-year and two-year follow-up assessments. At these times, the entry diagnosis was also reviewed, in the light of any extra information about the onset of the illness that might have come to light in the intervening period, and which should have been available to the project diagnosticians at the time of entry (only one change was made in the entry diagnoses as a result of this review for the 68 patients in this report). The diagnoses were consensus diagnoses made at meetings of two or more experienced psychiatrists using the criteria of ICD-9, and potentially included a main and a subsidiary diagnosis, and, where appropriate, an alternative diagnosis to the main one. All the survey schedules and the hospital case-notes were available for these meetings.

The presence of main, subsidiary and alternative diagnoses allows the project diagnosis of these patients to be expressed in several different degrees of confidence for schizophrenia. A 'schizophrenia confidence' code was devised as follows:

(a) Schizophrenia certain: the main diagnosis is any of the schizophrenic categories except schizoaffective type (295.0–295.9, except 295.7), and no alternative diagnosis is given.
(b) Schizophrenia very likely: the main diagnosis is as for (a), but an alternative diagnosis is also given (any code).
(c) Schizophrenia probable: the main diagnosis is schizoaffective psychosis (295.7) or a paranoid psychosis (including psychogenic) (i.e. 297.0–297.9, 298.3, 298.4).
(d) Schizophrenia possible: the alternative diagnosis is a schizophrenic or paranoid category as in (a)–(c), but the main diagnosis is not.
(e) Not schizophrenic: neither main nor alternative diagnosis is any of the schizophrenic or paranoid categories.

For the purpose of this report, the first two categories of 'certain' and 'very likely' are combined, to give a group of 68 patients; for convenience, they are referred to here as having a diagnosis of 'definite schizophrenia'. The selection of these two groups means that the six patients with a diagnosis of schizoaffective schizophrenia are not included in this report.

A number of scores were derived from the PSE and PIRS:

(a) Four subscores were derived from the PSE: delusional and hallucinatory syndromes (DAH); behaviour, speech and other syndromes (BSO); specific neurotic syndromes (SNR); and non-specific neurotic syndromes (NSN), as in the CATEGO program. Table I indicates the constituent syndromes for these subscores.
(b) A 'total depression score' was defined as the sum of scores for the three PSE depressive syndromes: simple depression (SD); special features of depression (ED); and other symptoms of depression (OD).
(c) A 'clinical poverty score' was defined as the sum of the scores on the following syndromes: catatonic

TABLE I
PSE syndromes and subscores[1]

Abbreviation	*Definition*
DAH	*Delusional and hallucinatory syndromes*
NS	Nuclear syndrome
DD	Depressve delusions and hallucinations
AH	Auditory hallucinations
PE	Delusions of persecution
RE	Delusions of reference
GR	Grandiose and religious delusions
SF	Sexual and fantastic delusions
VH	Visual hallucinations
OH	Olfactory hallucinations
SC	Subcultural delusions and hallucinations
BSO	*Behaviour, speech and other syndromes*
CS	Catatonic syndrome
IS	Incoherence of speech
RS	Residual syndrome
AF	Affective flattening
HM	Hypomania
OV	Overactivity
SL	Motor slowness
NP	Non-specific psychotic syndrome
AG	Agitation
NG	Self-neglect
SNR	*Specific neurotic syndromes*
SD	Simple depression
ON	Obsessional syndrome
GA	General anxiety
SA	Situational anxiety
HT	Hysteria
ED	Special features of depression
NSN	*Non-specific neurotic syndromes*
DE	Depersonalisation
IR	Ideas of reference
TE	Tension
LE	Lack of energy
WO	Worrying etc.
IT	Irritability
SU	Social unease
IC	Loss of interest and concentration
HY	Hypochondriasis
OD	Somatic symptoms of depression

1. The 36 Present State Examination (PSE) syndromes are summed in four groups to derive components of the total score. The symptoms that make up the syndromes are listed by Wing *et al* (1974).

syndrome (CS); affective flattening (AF); slowness (SL); and self-neglect (NG).

(d) A syndrome of akinesia was derived from the PIRS. The syndrome comprised ten items related to psychic tempo, communication skills and speech. Details are available from the authors.

(e) The presence of extrapyramidal movements was also rated from the PIRS.

For further details of the symptoms that make up these syndromes and the methods of scoring the PSE, see Wing *et al* (1974).

Cut-off points for three of these scores were defined as follows.

(a) 'Clinical depression' was diagnosed if the total depression score was more than 5 in the presence of depressed mood (PSE symptoms 23 or 121).

(b) 'Clinical poverty' was diagnosed if the total poverty score was more than 2.

(c) Akinesia was diagnosed if the PIRS akinesia score was more than 2.

The 'clinical depression' definition corresponds to the checklist definition of depressed caseness derived by Finlay-Jones *et al* (1980). It was chosen because patients meeting its criteria would be considered to be sufficiently depressed to warrant description as cases in that respect alone regardless of any schizophrenic symptoms.

No such criteria exist for the definition of 'cases' of akinesia or clinical poverty, and we therefore took as the threshold the mean score for the group of patients who scored at all in the relevant syndrome groupings.

At one-year follow-up interview the same assessments were made. In addition, information on the medication that the patients had been prescribed in each month of the follow-up year was obtained from the PPHS.

Results

Onset data

There were 45 men and 23 women in the group. Nearly half of them were aged between 15 and 24 years (49%), 17 patients (25%) were aged between 25 and 34 years, and the rest were aged up to 55 years. Predictably, therefore, many of the patients were unmarried (68%), and 53% lived at home with their parents. The social class of the household head was defined according to the Registrar General's definition, and 18% of the group were in social classes I, II, and III (non-manual), while the rest were in skilled and unskilled manual categories of social class. At entry to the study, 75% of the group were in-patients.

A number of social and demographic factors were checked for significant association with any of the PSE syndromes, the PSE-derived subscores, or the PIRS-derived akinesia score. There was no association between any of these measures of psychiatric status and the patients' age, sex, marital status, social class, or occupation. As far as the illness itself was concerned, there was no association between any of the PSE or PIRS measures and the acuteness of onset as assessed on the PPHS, or the subtype of schizophrenia as defined using ICD-9 criteria.

The differences in initial presentation were sought between those eventually diagnosed as definite schizophrenics and others. PSE syndrome profiles for the definite schizophrenics and the other diagnostic groups are shown in Fig. 1. PSE and PIRS syndromes not part of the delusional and hallucinatory subgroup (DAH) were tested individually for their association with a final diagnosis of 'definite schizophrenia' or 'any other diagnoses' using corrected χ^2. The principal finding was that hypomania

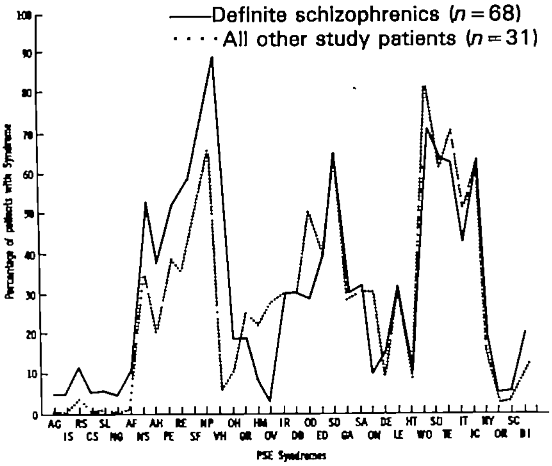

Fig. 1 PSE syndrome profiles (percent prevalence) at onset.

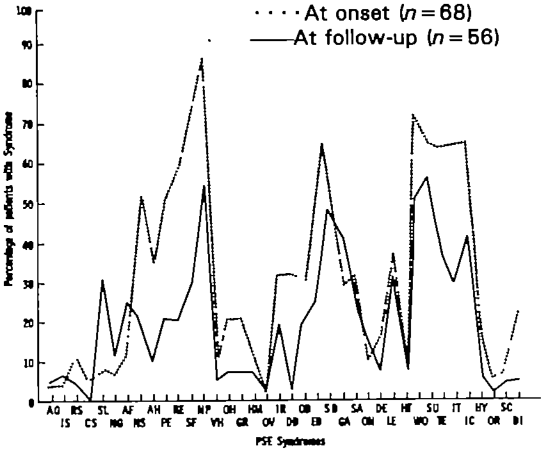

Fig. 2 PSE syndrome profiles (percent prevalence) for definite schizophrenics.

(HM) and somatic symptoms of depression (OD) were associated with all diagnoses except definite schizophrenia ($P<0.05$). Other mood symptoms were unrelated to diagnosis, while affective flattening (AF), residual syndrome (RS) and akinesia were associated with a final diagnosis of definite schizophrenia ($P<0.05$).

Non-psychotic syndromes at onset

The onset data were examined to determine the degree to which the depressive syndromes, negative syndromes and akinesia could be distinguished from each other, by testing the correlations between all these PSE syndromes for the 68 patients. The specific depressive syndromes and the non-specific dysphoric syndromes tended to be significantly associated with each other, as shown in Table II. None of these depressive or dysphoric syndromes was correlated with any of the negative syndromes or akinesia, at the 5% significance level.

At onset, 15 of the 68 definite schizophrenic patients (22%) were defined as depressed cases by the criterion adopted here. In comparison, 23 patients (34%) fulfilled Hirsch's criterion of a score on at least three of the PSE depressive syndromes (a broader definition because depressed mood itself was not a prerequisite). Twenty-one patients (31%) had some score on the syndrome 'other features of depression' (OD), which comprises the biological symptoms of depression.

One-year follow-up data

Of the original 68 'definite schizophrenics', 56 were available for follow-up interview at one year. Their PSE syndrome profiles are shown in Fig. 2. The 12 patients unavailable for PSE interview at follow-up did not differ substantially from the overall group with regard to their syndrome profiles or other characteristics at onset.

The prevalence of delusional and hallucinatory syndromes (DAH) and of behaviour and speech disorder (BSO) was reduced in the follow-up period, such that for the group as a whole there was no association between onset and follow-up scores for DAH and BSO.

Similarly there was a decrease in prevalence both of depressive and of other neurotic syndromes. In particular, only five patients at follow-up were considered depressed cases; three of these had been definite and one a borderline case at onset (see Table III). The number of patients with any biological symptoms of depression had fallen from 21/68 (31%) to 11/56 (20%).

Three subjects who were diagnosed as depressed cases at onset could not be interviewed at follow-up; two had left the area and lost contact and one was in prison, reportedly still in a depressed, withdrawn state and receiving antidepressant medication. Of the other nine subjects not available for PSE interview at follow-up, three had left the area, five declined interview and one was in prison. In other words, there is no good evidence that the prevalence of depression fell at follow-up because of failure to interview the most depressed subjects.

By contrast, both clinical poverty and akinesia became more prevalent in the group. Numbers of patients with a clinical poverty score >2 had increased from three to nine and with an akinesia score >2 from 31 to 38.

Symptoms and medication

Any association between clinical picture at follow-up and recent medication was sought by comparing those patients who had been prescribed neuroleptics consistently throughout the second six months of the follow-up period with those who had not. Complete information on neuroleptic medication was only obtainable for 46 of the follow-up group. The numbers involved were too small to assess the relationship between depression or clinical poverty and neuroleptic medication. However, as shown in Table IV, both akinesia and the subscore BSO were associated with neuroleptics.

Of the 68 definite schizophrenic patients, 11 patients received a course of antidepressant medication for three consecutive months at some time during the follow-up year.

TABLE II

Pearson product–moment correlation coefficient matrix of significantly associated ($P<0.05$) PSE/PIRS dysphoric syndromes (definite schizophrenics, onset data)

	SD	*OD*	*ED*	*SA*	*GA*	*IR*	*TE*	*WO*	*IT*
Simple depression (SD)									
Somatic symptoms of depression (OD)	0.58								
Special features of depression (ED)	0.37	0.45							
Situational anxiety (SA)	0.34	0.29	NS						
General anxiety (GA)	0.26	0.40	NS	0.27					
Ideas of reference (IR)	0.36	0.24	NS	0.28	NS				
Tension (TE)	0.44	0.35	NS	0.28	0.37	NS			
Worrying etc (WO)	0.42	0.32	0.35	NS	NS	0.42	0.52		
Irritability (IT)	0.31	0.24	NS	NS	NS	0.29	0.23	0.32	
Obsessional syndrome (ON)	0.49	0.53	0.35	0.29	0.26	0.29	0.49	0.32	NS

TABLE III

Relationship between depressive caseness[1] and total neurotic score at onset and follow-up for definite schizophrenics

Case	*Onset: depression caseness*[2]	*Onset: total neurotic score (NSN+SNR)*	*Follow-up: depression caseness*[2]	*Follow-up: total neurotic score (NSN+SNR)*
004	Y	22	N	11
006	Y	23	N	3
012	Y	28	U	—
013	Y	23	U	—
014	Y	27	N	13
015	Y	29	U	—
017	Y	36	B	21
029	Y	10	N	2
037	N	12	Y	21
045	Y	25	N	1
047	Y	27	N	7
051	Y	23	Y	30
052	B	15	Y	28
058	Y	12	N	4
068	Y	26	N	7
084	Y	23	Y	23
086	Y	14	N	0
097	Y	11	N	7
099	Y	42	Y	40

1. Depressed caseness as defined by Finlay-Jones checklist criteria; see text for details
2. Y, yes; N, no; B, borderline; U, unavailable

TABLE IV

Relationship of neuroleptic medication to clinical picture[1] at follow-up

	Patients on neuroleptics for all 6 months prior to one-year follow-up interview ($n=28$)	*Patients not on neuroleptics for all 6 months prior to one-year follow-up interview ($n=28$)*	
Depression			
Caseness	3	1	NS
Poverty score			
>2	3	1	NS
Akinesia			
>2	10	2	$P=0.02$
<2	18	16	($\chi^2=7.885$, d.f.$=2$)
BSO			
1+	23	9	$P=0.03$
0	5	9	($\chi^2=7.018$, d.f.$=2$)

1. See text for definitions of clinical scores

Of these 11, only five were depressed cases at onset (subjects 012, 037, 047, 097, 099), and two remained depressed at follow-up. None of the four patients who received ECT during the year were depressed cases at onset or follow-up; two received treatment for symptoms of withdrawal and neglect with little overt depression, one for a state of catatonic excitement, and one for depressive symptoms of non-caseness severity occurring in the puerperium. All four received ECT within two months of onset. In other words, the reduction in prevalence of depression at follow-up could not be attributed solely to specific treatments for depressive illness.

Discussion

Discussions about the meaning of mood disorder in schizophrenia have been hampered by uncertainties about diagnostic validity or selection bias in the group studied. The present group of patients represents an unselected, consecutive series of contacts with Mapperley Hospital, which serves a diverse, mainly urban population. All contacts were for the first onset of psychotic disorder: they were not acute relapses of previously known patients. Diagnostic uncertainty was kept to a minimum by the two-year follow-up period and the use of consensus meetings of experienced psychiatrists to arrive at clinical diagnoses based on the operational criteria of a standard classification. This diagnostic process led to exclusion from the group of 'definite schizophrenia' of patients with major mood disorders at onset. For example, syndrome profiles show that the presence of hypomania (HM) or biological symptoms of depression (OD) was significantly less common in the 'definite schizophrenia' group than in those with any other diagnosis. The frequency of both these syndromes in the group was less than that noted for a population diagnosed as schizophrenic using CATEGO and on the basis only of onset of mental state (Knights & Hirsch, 1981).

Despite this proviso, patients with a diagnosis of definite schizophrenia had a wide range of dysphoric symptoms with an essentially similar distribution and frequency to those without such a diagnosis. Despite differences in case selection and diagnostic criteria, the clinical syndrome profile of our patients was similar to that of those described by Knights & Hirsch (1981).

At one-year follow-up there was a substantial reduction in the specific and non-specific dysphoric syndromes, so that relatively few patients continued to display a clinically significant depressive disorder. Nonetheless, minor dysphoric symptoms remained common. The time interval between the two interviews does not preclude the possibility of a brief but major depressive episode emerging in the months after acute onset and resolving spontaneously or with phenothiazines (Mandel *et al*, 1982).

The negative syndromes present a more complicated picture. They were present at first onset in some patients, so they cannot be considered solely as a consequence of chronicity or of prolonged neuroleptic medication. Nonetheless, their prevalence increased at follow-up, when there was a significant association with neuroleptic medication even in the absence of Parkinsonian symptoms. This suggests one of the following: (a) that the negative syndromes are drug-induced in some patients, (b) that they cannot always be reliably separated from drug-induced akinesia, or (c) that patients who are more prone to develop these syndromes have other features which make it more likely that they will be prescribed neuroleptics. The observational nature of the present study did not allow a further examination of these possibilities.

The question remains as to what part neuroleptic medication has to play in depression. There is no evidence from our data that depressive syndromes were related to concurrent neuroleptic medication. This finding is in keeping with ideas of revealed or co-existent depression (Knights & Hirsch, 1981; Ndetei & Singh, 1982) and provides no support for the concept of pharmacogenic depression.

In summary, these findings demonstrate that the depressive disorders so commonly seen in the follow-up of schizophrenic patients are present at first onset, and that those patients who are depressed at follow-up are the same individuals who were depressed at onset. Few new cases of depression emerge at follow-up, and in fact the prevalence of depression reduces with time. The depressive syndromes can be reliably separated from akinesia and clinical poverty, which by contrast increase in prevalence at follow-up and are associated with the taking of neuroleptics. The purpose of the study at the time these ratings were made was not specifically to examine depressive or negative syndromes in schizophrenia, and therefore this differential behaviour of the two syndrome groups is unlikely to have been due to rater bias.

Taken together, these findings argue against the suggestion that neuroleptic medication is important in causing depression in schizophrenia, either in a manifest form or in a disguised form such as akinetic depression.

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Prediction of Outcome in Depressed Patients by Weekly Monitoring with the Dexamethasone Suppression Test

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Forty-three depressed patients in hospital were studied with weekly dexamethasone suppression tests (DSTs) and were followed as out-patients for at least three months after discharge. The detection rate of patients with LHPA axis dysfunction increased from 41% with a single DST to 59% with serial DSTs. There was a poor correlation between weekly post-dexamethasone cortisol levels and Hamilton depression rating scores. In patients with evidence of LHPA axis dysfunction, a DST at discharge discriminated effectively between a good and a poor outcome group; persistent non-suppression was strongly linked with a relapse of depression in the first three months after discharge. In general, our results support previous claims that the DST is a state marker for depressive illness.

Extensive research which originated in the 1960s (Carroll *et al*, 1968; Carroll & Davies, 1970; Sachar, 1967) has resulted in the finding that approximately 50% of patients suffering from a major depressive illness have an associated abnormality of functioning of the limbic–hypothalamic–pituitary–adrenal (LHPA) axis. Evidence, including recent studies with the corticotropin releasing factor stimulation test, suggests that the dysfunction originates from centres at or above the hypothalamus (Holsboer *et al*, 1984; Gold & Chrousos, 1985). The outcome of these studies has been the widespread clinical application of the dexamethasone suppression test (DST) as standardised by Carroll *et al* (1981). The popularity of the DST as a measure of LHPA axis dysfunction in depression has resulted from its simplicity and ease of application. Recently, a greater awareness has grown (Berger *et al*, 1982; Hirschfeld *et al*, 1983; Braddock, 1986) of several intervening variables which may cause difficulties in interpretation of results. These variables, such as false positives and negatives, dexamethasone concentrations, and drug withdrawal, have imposed limitations on the DST's principal clinical use as a diagnostic tool.

The DST has also been used to monitor and predict treatment response; however, this aspect of the DST has not been extensively investigated. Carroll *et al* (1976*a*,*b*) reported three cases of normalisation of the DST response following recovery from depression, and this observation has been repeated in several studies (Albala & Greden, 1980; Greden *et al*, 1980; Albala *et al*, 1981). Non-suppression of the DST at discharge has been linked with a poor response to treatment, increased likelihood of a relapse of symptoms following discharge, and increased suicide risk (Goldberg, 1980*a*,*b*; Yerevanian *et al*, 1984; Nemeroff & Evans, 1984; Targum, 1984).

There have been four detailed reports in which depressed patients were systematically monitored while in hospital with serial (mostly weekly) DSTs (Holsboer *et al*, 1982; Greden *et al*, 1983; Bowie & Beaini, 1985; Gerken *et al*, 1985). All concluded that normalisation of the DST response was linked with good treatment outcome. Greden *et al* (1983) reported a correlation between decreasing post-dexamethasone cortisol levels and decreasing ratings of severity of depression. These findings suggest that the DST may be a biological state marker for the episode of a depressive illness in the monitoring of treatment response. In the capacity of monitoring patient progress, the previously mentioned limitations of the test are less crucial, especially if intervening variables remain stable.

In this report we present data on 43 depressed patients who had weekly DSTs while in hospital. As our aim was to investigate the prognostic implications of changes in DST status, we have included follow-up, post-discharge data. In this study, treatment and discharge decisions were made solely on clinical grounds, without reference to DST results.

Method

Patients

Over a one-year period, 43 patients admitted to the Psychiatric Unit of the Royal Melbourne Hospital with a primary diagnosis of Major Depressive Disorder were studied. All patients were assessed by two senior psychiatrists using DSM-III (American Psychiatric Association, 1980) diagnostic criteria. Patients had a detailed physical examination and laboratory screening tests to exclude serious physical disorder. Carroll's exclusion criteria (Carroll *et al*, 1981) were applied. Only those patients who completed at least three weeks of treatment have been included. Although not all patients studied were initially medication-free, none were receiving drugs which are known to influence dexamethasone kinetics. Informed consent was obtained from all patients.

DST

During the first week (but not before the third day following admission) all patients received a standard DST as specified by Carroll *et al* (1981), with 1 mg of dexamethasone being administered at 23:00 and blood samples for plasma cortisol being taken the same and the following day at 16:00. Plasma cortisol was analysed in the Department of Biochemistry (Royal Melbourne Hospital) using the routine assay (Amerlex; Amersham, UK). The DST was repeated at approximately weekly intervals while the patients were in hospital.

Definition of non-suppression/suppression status

In this report we have based our definition of cortisol non-suppression on the result of a previous study in this department of 100 normal volunteers (Tiller *et al*, in preparation). Volunteers received a standard DST and the criterion value was determined using Carroll's (1985) recommendation that 5% of a normal population are taken to be non-suppressors. The derived criterion value was 170 nmol/l. In the region around the criterion value, the confidence with which an individual can be classified as a suppressor or non-suppressor is low. Such an interval was defined using the coefficient of variation for cortisol at 170 nmol/l, which was approximately 5% for the Amersham kit used in this study. Therefore, we have classified post-dexamethasone plasma cortisol values above 187 nmol/l as non-suppression and values below 153 nmol/l as suppression, with at least 95% confidence. Plasma cortisol values between these limits have been excluded from analysis.

Clinical ratings

Patients received weekly assessments on the 17-item Hamilton depression observer-rating scale (HDRS) and the Carroll depression self-rating scale. Assessors were blind to the laboratory DST results. All patients scored 17 or higher on the HDRS for admission into the study.

Patient case records were studied for post-discharge follow-up data and whenever possible this was supplemented by consultation with the treating psychiatrist. We defined relapse as a recurrence of those symptoms that had led to the patient's original admission; the symptoms were judged to be of such severity to warrant either readmission to hospital or an increase in dose or change of medication and closer out-patient supervision.

Weight assessment

A careful history of recent weight loss was recorded at admission. All patients were weighed and their heights measured at admission, and they were weighed weekly while in hospital.

Treatment

All patients received standard antidepressant treatment; that is, electroconvulsive therapy (ECT) or antidepressant medication. Courses of ECT ranged from 6–15 applications. All patients were maintained on antidepressant medication (lithium carbonate, tricyclics or monoamineoxidase inhibitors) during the out-patient follow-up period. All treatment decisions were made by the treating clinician.

Data analysis

Variables were tested for normality using the Kolmogorov–Smirnov one-sample test, and parametric or non-parametric tests were used where appropriate. Independent *t*-tests were used for group comparisons, and Pearson's product moment correlations for testing significance of relationships between variables. The significance level was set at $P<0.05$ for comparisons and at $P<0.01$ for correlations.

Results

Categorisation of patients

On the basis of the initial DST result, of the 43 patients studied, 24 patients had a suppressor response and 17 a non-suppressor response. Two patients had post-dexamethasone plasma cortisols within the region of unreliability and were excluded from the analysis.

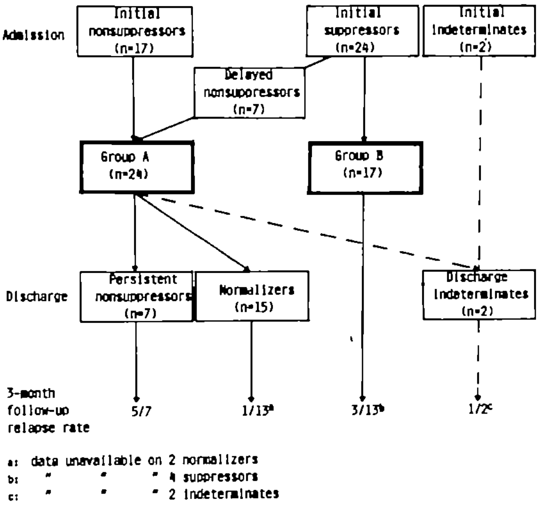
Admission
Initial nonsuppressors (n=17)
Initial suppressors (n=24)
Initial indeterminates (n=2)
Delayed nonsuppressors (n=7)
Group A (n=24)
Group B (n=17)
Discharge
Persistent nonsuppressors (n=7)
Normalizers (n=15)
Discharge indeterminates (n=2)
3-month follow-up relapse rate
5/7
1/13ᵃ
3/13ᵇ
1/2ᶜ
a: data unavailable on 2 normalizers
b: " " " 4 suppressors
c: " " " 2 indeterminates

FIG. 1. Categorisation of patients.

When serial DST responses were considered, there were seven patients with initial suppressor responses which were followed on subsequent weeks with at least two consecutive non-suppressor responses. As this indicated LHPA axis dysfunction, we have consequently studied these delayed non-suppressors with the initial non-suppressors (Fig. 1). Recategorisation gave 24 patients with evidence of LHPA axis dysfunction (group A) and 17 without such evidence (group B). Therefore, the detection rate of depressed patients with an abnormality of LHPA axis functioning was increased from 41% with a single DST to 59% with serial DSTs.

Group A and B categories did not differ significantly in age, baseline severity of depression as reflected by HDRS, weight, or history of recent weight loss (Table I).

TABLE I

Comparison of group A and group B patients

	Group A ($n=24$)		*Group B* ($n=17$)		*P*
	Mean	*s.d.*	*Mean*	*s.d.*	
Age: years	62.0	12.2	59.0	14.3	NS
Weight: kg	59.3	10.8	63.1	16.8	NS
Weight loss (pre-admission): kg	2.4	2.3	1.8	3.4	NS
HDRS	27.6	5.7	27.5	4.5	NS
Pre-dexamethasone cortisol: nmol/l	453	143	338	140	0.016

There was no relationship between the patients' sex and group A or B status. Seven group A and five group B patients were male ($\chi^2=0.389$, NS). There was no difference in choice of treatment between the two groups, A patients being no more likely to receive ECT than B patients, 14 As and 11 Bs receiving ECT ($\chi^2=1.42$, NS). The median stay in hospital was five weeks for both groups.

Comparisons of post-dexamethasone cortisols and HDRS

The relationship between mean post-dexamethasone plasma cortisols and mean HDRS scores over four consecutive weeks was examined in the 16 group A patients who had complete records. There was a tendency for cortisol and HDRS scores to fall together, but this did not reach statistical significance ($r=0.832$, $P=0.08$).

There were no significant correlations between post-dexamethasone plasma cortisols and HDRS scores at weeks 1–4 for group A or B patients; for example, group A at week 1, $r=0.083$, $P=0.351$. Similarly, there were no significant lag correlations between the variables over the four weeks, correlations ranging from $r=-0.131$ ($P=0.314$) to $r=0.234$ ($P=0.191$) for group A and from $r=-0.198$ ($P=0.313$) to $r=0.218$ ($P=0.286$) for group B.

Normalisation of the DST response in group A patients

At discharge, 15 of the 24 patients with evidence of LHPA axis dysfunction (group A) were suppressing cortisol normally (normalisers), while 7 had a non-suppressor response (persistent non-suppressors). Two patients had intermediate post-dexamethasone plasma cortisol levels at discharge and were not subclassified (Fig. 1). The mean HDRS of the persistent non-suppressors (13 ± 7) tended to be higher than that of the normalisers (11 ± 6), but the difference did not reach statistical significance. Discharge weights and mean hospital stay were similar in these two subcategories. Also, there had been no difference in choice of treatment during hospital stay: four persistent non-suppressors and eight normalisers received ECT ($\chi^2=0.082$, NS).

Three-month post-discharge follow-up

Follow-up post-discharge data were available on 13 group B and 20 group A patients. A relapse of the depressive illness occurred in six group A and three group B patients; this result was non-significant ($\chi^2=0.016$, NS).

However, in patients with evidence of LHPA axis dysfunction (group A subjects), serial DSTs discriminated between those who were likely and those not likely to suffer a relapse of depression. Only one of 13 normalisers relapsed, while five of the seven persistent non-suppressors suffered a relapse. This result is highly significant (Fisher's exact: $P=0.006$). There was no significant difference in mode of out-patient treatment between the two subcategories; neither normalisers nor persistent non-suppressors were more likely to receive lithium carbonate or other antidepressant therapy. Three of the 13 normalisers and two of the seven persistent non-suppressors were prescribed lithium carbonate ($\chi^2=0.073$, NS).

Discussion

In this report we present data on 43 depressed patients who were followed with serial DSTs while in hospital. Our results support previous work which concludes that the DST is a biological state marker for some patients who have a depressive illness.

In our patient sample, a DST shortly prior to discharge was able to effectively distinguish a poor outcome from a good outcome group. For patients who were persistent non-suppressors there was a significantly increased likelihood of relapse in the first three months following discharge. Most patients (92%) whose DST response normalised while they were in hospital progressed well after leaving hospital. These findings are in keeping with previous work, such as that of Yerevanian *et al* (1983), who reported on ten patients who were non-suppressors at discharge. Nine of the ten, in their report, did poorly following discharge, with three patients committing suicide. Our study has provided further evidence for the predictive value of the DST by including a comparison outcome group of normalisers, thereby establishing the highly significant difference in outcome between normalisers and persistent non-suppressors.

The weight of current evidence suggests that a non-suppressor response at discharge indicates inadequate or incomplete treatment of the depressive illness. These patients are a high-risk group and we recommend either further in-patient assessment or careful out-patient supervision, such as weekly appointments. To further investigate these conclusions we suggest that there is an important need for outcome research where such patients are followed prospectively with out-patient DSTs.

It is pertinent to mention those few patients whose clinical course was inconsistent with the above findings. There were two persistent non-suppressors who remained well following discharge. In both of these cases, reductions in post-dexamethasone cortisol levels of over 50% had in fact occurred during their hospital stay. It is possible that their DST responses may have normalised shortly after discharge, but we do not have data to support this contention. The one normaliser who relapsed did so near the end of her three-months follow-up.

A principal purpose of a state marker in medicine is to monitor improvement in the underlying pathophysiological process of a particular condition (Galen & Gambino, 1975). Clinical symptomatic status has limitations in following response to treatment because of its subjectivity and vulnerability to influence from a multiplicity of factors. Consequently, serial testing has widespread application in medicine, monitoring recovery in conditions such as anaemia, myocardial infarction, diabetes, and uraemia. In depressed patients with evidence of LHPA axis dysfunction, there is a general group trend for normalisation of the DST response and clinical improvement to parallel each other during hospital stay; however, there is no clear relationship when patients are considered on an individual basis. This finding is consistent with previous work (Gerken *et al*, 1985; Bowie & Beaini, 1985). For some depressed patients, the non-specific aspects of being in hospital and the psychosocial interventions may result in a temporary amelioration of symptoms. Therefore the relatively poor correlation between HDRS (an essentially clinical assessment) and post-dexamethasone cortisol does not in fact detract from the DST's usefulness in monitoring response to treatment. Our results suggest that if a non-suppressor response persists at discharge in a patient with major depression, the biological aspect of the illness has been inadequately treated.

In this study we found that the detection rate of depressed patients with an abnormality of LHPA axis functioning increased from 41% with a single DST to 59% with serial DSTs. Greden *et al* (1983) reclassified three of 31 subjects from suppressor to non-suppressor status on the basis of a repeat DST. They attributed the change in DST status to anti-depressant withdrawal, regarding these patients as having been partially treated. It is likely that this was a factor with at least some of our reclassified patients. The DST is a sensitive, dynamic test, primarily of the feedback mechanism of the LHPA axis. The DST is vulnerable to a variety of influences, including technical errors and possibly unknown factors, which may cause both false positive and false negative results. The need to perform repeated tests is well recognised in certain medical conditions. For instance, several blood tests are often required to confirm a diagnosis of hypercalcaemia (Potts, 1983). The application of serial DSTs described here, for improved detection of depressed patients with LHPA axis dysfunction, has not been previously emphasised and needs further investigation.

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The minimum recommended starting dose is 100mg given in the evening. A total daily dosage in
of 100mg should be given in divided doses. Elderly: The same dosage regimen may be utilised in the
as in the general population. Children: There are insufficient data to recommend the use of Faverin in
. **Contra-indications, Warnings, etc:** Faverin should not be given together with or within two
of terminating treatment with monoamine-oxidase inhibitors. Patients with hepatic or renal
iency should begin treatment with a low dose and be carefully monitored. Faverin has no effect on
notor skills associated with driving and operating machinery. However, in combination with alcohol
npairment may be seen. Use in pregnancy and lactation: There has been little human use but there
en no adverse animal evidence. Avoid in pregnancy and in nursing mothers if a better known and safer
ive is available, although safety in pregnancy is likely. Interactions with other drugs: In interaction
studies increased plasma levels of propranolol and warfarin were seen during concurrent adminis
Faverin. Care should be taken when Faverin is prescribed to patients already receiving propra
warfarin as the dose of these drugs may need to be lowered. No interactions were seen with digox
atenolol. **Side-effects:** The commonest symptom observed with Faverin administration is naus
usually diminishes early during the course of treatment. Other treatment emergent symptoms
during clinical studies were somnolence, constipation, agitation, anorexia and tremor. Other report
are often associated with the illness and their incidence in controlled trials is comparabl
experienced with placebo. Faverin may cause a decrease in heart rate. During treatment, limited
repolarization were observed in the ECG, but no causal relationship could be demonstrated. The in
postural hypotension was similar to that seen in patients treated with placebo. Treatment of overd
specific antidote is known. The stomach should be emptied as soon as possible after tablet inge
symptomatic treatment should be given. The use of medicinal charcoal is also recommended.
Quantities 50mg tablets: available in packs of 60 (3 strips of 20 blister-packed tablets). **Product**
Number 50mg tablets: 0512/0070. Further information is available from: Duphar Laboratorie
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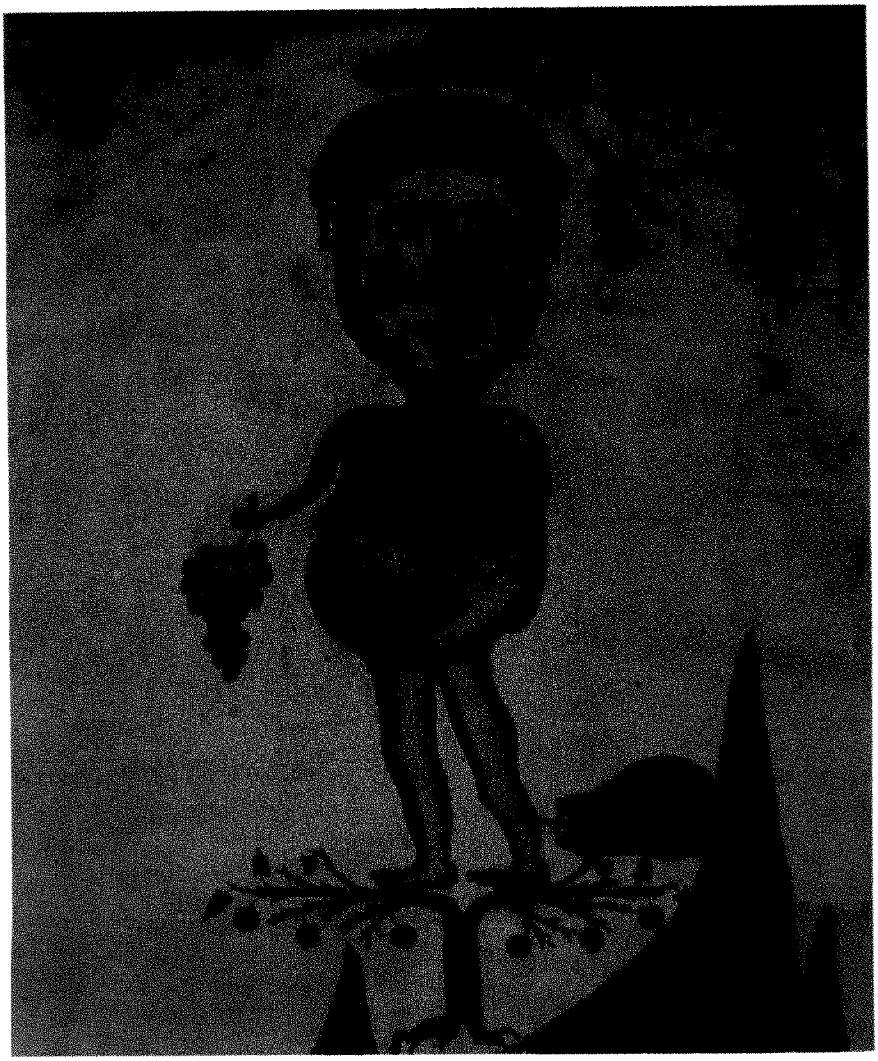

A TREATMENT.

Haldol* Decanoate administered as a single deep intra-muscular injection provides a consistent 4 week duration of action.

An initial dose of 50mg every four weeks is recommended, increasing if necessary after two weeks by 50mg increments to 300mg every four weeks. The four weekly dose should reflect the total dose given in the first four weeks of treatment. In patients with severe symptomatology or in those who required large oral doses for maintenance medication, higher doses of Haldol* Decanoate may be required.

The schizophrenic patient can benefit from a therapy with little incidence of sedation[1] and a reduced requirement for antiparkinsonian medication[2] leaving him/her better able to participate in other forms of therapy.

For example when 35 patients receiving fluphenazine decanoate 3-weekly were given instead Haldol* Decanoate 4-weekly: "A total of 18 patients could successfully be withdrawn from previously used antiparkinsonian medication . . . without experiencing an exacerbation of extrapyramidal side-effects and their psychotic symptoms continued to be well controlled."[3]

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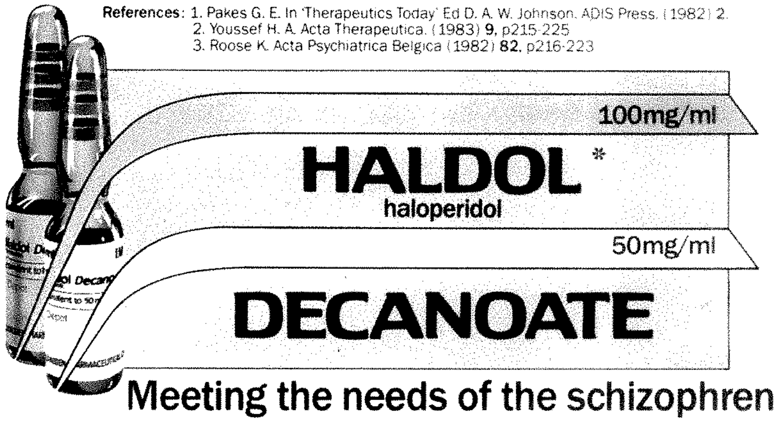

Meeting the needs of the schizophrenic.

Presentation: Straw coloured, viscous solution presented in 1ml brown glass ampoules equivalent to 100mg/ml haloperidol (as decanoate ester) and 1ml brown glass ampoules equivalent to 50mg/ml haloperidol (as decanoate ester) both in packs of 5. **Uses**: As a depot injectable form of haloperidol indicated for adults where long term maintenance treatment with a neuroleptic is required, for example in schizophrenia, other psychoses (especially paranoid) and other mental or behavioural problems where maintenance treatment is clearly indicated. **Side effects, precautions, contra-indications**: Haloperidol is not recommended during lactation. Use in Pregnancy: The safety of haloperidol in human pregnancy has not been established. Precautions: Caution in liver disease, Parkinson's disease, phaeochromocytoma, thyrotoxicosis, epilepsy and conditions predisposing to epilepsy. Antagonism of adrenaline, guanethidine, phenindione, anti-Parkinson effects of levodopa and impairment of metabolism of tricyclic anti-depressants have been reported. Haloperidol may increase the effects of CNS depressant drugs, and enhanced CNS effects when combined with methyldopa have been reported. Neurotoxic reactions to combined treatment with lithium and haloperidol have been reported. Side effects: In common with all neuroleptics, the following side effects may be observed, sedation, extra-pyramidal symptoms, tardive dyskinesia, mental dullness, dizziness, headache, excitement, agitation, insomnia, hyperprolactinaemia, galactorrhoea, gynaecomastia, oligo- or amenorrhoea, hypotension. Gastro-intestinal symptoms and weight changes have been reported. Anti-Parkinson agents should only be given as required. The elderly are more susceptible to sedative/hypotensive effects. Occasional local reactions such as erythema, swelling or tender lumps have been reported. In common with other anti psychotics, haloperidol has been associated with rare cases of neuroleptic malignant syndrome. **Dosage**: Haldol* Decanoate provides one month's therapy following a single deep intra-muscular injection in the gluteal region. An initial dose of 50mg every 4 weeks is recommended increasing if necessary after 2 weeks by 50mg increments to 300mg every 4 weeks. The dosage should be tailored to suit the individual patient and to reflect previous oral dosage. In adults 100mg Haldol* Decanoate is approximately equal to 500mg chlorpromazine (or equivalent) per day. Caution is required with the elderly, starting doses of 12.5mg to 50mg monthly should be tried first. **Product Licence Numbers**: 0242/0095, 0242/0094 **Basic NHS Cost**: 1ml x 100mg/ml x 5 £24.00 1ml x 50mg/ml x 5 £15.60. (Correct at time of printing). Further information is available on request from: **Janssen Pharmaceutical Limited**, Grove, Wantage, Oxon OX12 0DQ.

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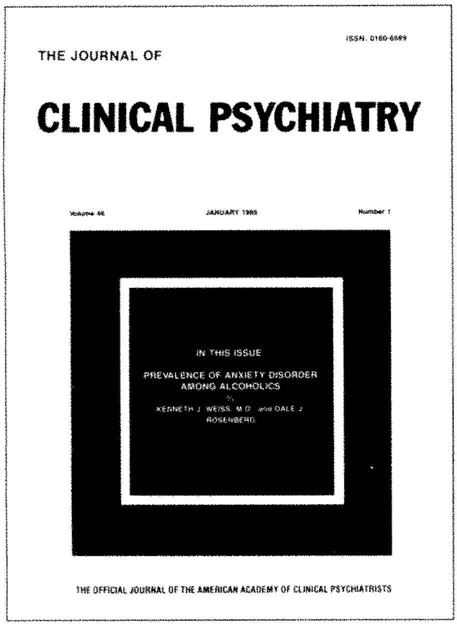

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IN THIS ISSUE

PREVALENCE OF ANXIETY DISORDER AMONG ALCOHOLICS

by

KENNETH J. WEISS, M.D. and DALE J. ROSENBERG

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Major Affective Disorder in Anorexia Nervosa and Bulimia

A Descriptive Diagnostic Study

REINHOLD G. LAESSLE, SUSANNE KITTL, MANFRED M. FICHTER,
HANS-ULRICH WITTCHEN and KARL M. PIRKE

DSM–III lifetime diagnoses were assessed in 52 patients with a lifetime history of anorexia nervosa or bulimia by means of a standardised diagnostic interview. It was found that 44.2% had a lifetime diagnosis of DSM–III major affective disorder, with abstaining anorectics having a lower rate of depression than those with bulimic symptoms. In the great majority of cases, the onset of affective disorder post-dated the onset of the eating disorder by at least one year. In patients whose eating disorder was in remission, the rate of depressive symptoms was lower than in those in the acute stage of their illness. These findings, combined with recent studies on biological changes in eating disorders, and psychological theories of depression, suggest that in most cases in which the two conditions are associated, the depression is secondary to the eating disorder.

The possibility of a close relationship between the two eating disorders and major affective illness has been the subject of considerable recent interest (e.g. Swift *et al*, 1986), and the hypothesis that anorexia nervosa and bulimia are each a variant of affective disorder has been suggested by several authors (Cantwell *et al*, 1977; Hudson *et al*, 1983). Evidence for this association is provided by neuroendocrine similarities, the response of eating disorder patients to antidepressant medication, family studies, and psychopathological observations of the frequent occurrence of depressive symptoms in the acute stage of an eating disorder. However, the conclusion that eating disorders are a variant of or secondary to affective disorders has been questioned. Neuroendocrine abnormalities both in major depression and eating disorders may be due to various behavioural factors such as the quantity, quality and temporal ordering of nutrition (Pirke *et al*, 1985), exercise (Bullen *et al*, 1985), or stress (Rose, 1984). Other authors have shown that only a small proportion of patients with anorexia nervosa or bulimia respond to antidepressant medication (Sabine *et al*, 1983; Fairburn, 1984), and a recent well-controlled family study found rates of affective disorder in relatives of eating disorder patients to be no higher than those in relatives of normal controls (Stern *et al*, 1985).

Data on psychopathological relationships have to be interpreted with care. In most studies, 'depression' in eating disorder patients has been assessed by means of self-report scales like the Beck Depression Inventory or MMPI subscales, which do not allow syndromal diagnosis of affective disorder. Studies using operational diagnostic criteria have shown great variability in rates of major affective disorder in acute eating disorder patients (for review see Strober & Katz, 1986), and the nature of the relationship between the two illnesses remains unclear. The available data could well be interpreted as indicating that the depression is a consequence of or reaction to the psychopathological and biological characteristics of the eating disorder. Clarification necessitates consideration of the rate of affective disorder not only in the acute stage of eating disorder, but also at other times during the patient's life. Attention must also be paid to the temporal relationship between onset of the eating disorder and the onset of affective illness. Furthermore, patients should be studied for the persistence of depressive symptoms after successful treatment of their eating disorder. The aim of this study is to investigate the psychopathological relationship of affective disorder to eating disorders using standardised diagnostic assessment and DSM–III diagnostic criteria.

The following questions are dealt with: (a) How large are the lifetime rates of major affective disorders in patients with different types of eating disorder? (b) Does the onset of major affective disorder precede the onset of eating disorder? (c) Is the rate of major affective disorders lower in patients after treatment of their eating disorder than before?

Method

Subjects were 52 consecutive individuals who met DSM–III criteria (American Psychiatric Association, 1980) for anorexia nervosa, bulimia or both disorders at any point in their lifetime. Characteristics of the total sample and

TABLE I
Characteristics of 52 patients with eating disorders

	AN (anorexia only)	*AN/B (anorexia and bulimia)*	*B (bulimia only)*	*Total sample*
	(n = 13)	*(n = 26)*	*(n = 13)*	*(n = 52)*
Age: years				
Mean	21.5	22.1	24.9	22.7
Standard deviation	3.0	3.1	5.7	4.0
Duration of illness: years				
Mean	2.7	6.0	6.3	5.3
Standard deviation	1.6	3.5	5.2	3.9
Weight maximum: % IBW[1]				
Mean	97.5	111.9	117.0	106.3
Standard deviation	12.6	17.2	15.5	23.4
Weight minimum: % IBW				
Mean	64.8	66.3	89.1	69.3
Standard deviation	9.1	6.9	9.6	16.4

1. IBW = Ideal Body Weight according to tables of Metropolitan Life Insurance Company, 1959.

subgroups are shown in Table I. All but one patient in the group with anorexia nervosa plus bulimia were female. Thirty-five of the patients were interviewed for the study as in-patients within the first two weeks of admission, the remaining 17 as out-patients at a follow-up one year after discharge from hospital. Patients were treated on open psychiatric wards at either the Max Planck Institute of Psychiatry or the Psychiatric University Hospital in Munich. Diagnostic assessment was made using the German version of the Composite International Diagnostic Interview (CIDI), which is an expanded form of the National Institute of Mental Health Diagnostic Interview Schedule (DIS) (Robins *et al*, 1981). The CIDI/DIS is a standardised instrument of demonstrated reliability (Semler *et al*, 1987) and enables computerised DSM-III diagnoses. All patients were assessed for eating disorder by two psychometric questionnaires, the Eating Disorder Inventory (EDI; Garner *et al*, 1983) and the Eating Attitudes Test (EAT; Garner *et al*, 1982). The CIDI/DIS interviews were conducted by one of the first two authors (RGL, SK). There were no significant differences between the two interviewers in the rate of diagnosing any DSM-III disorder.

A control group for lifetime frequency of depressive symptoms was formed of 47 patients (all female; mean age 27.3 years, s.d. 3.8) with a DSM-III diagnosis of major depression, but without a lifetime diagnosis of an eating disorder, who had been treated at the Max Planck Institute of Psychiatry and who were also interviewed with the CIDI/DIS.

Relative frequencies were compared for statistical significance using Fisher's exact test with Tocher's modification. Significance levels are one-tailed. To obtain correct levels of significance in the case of multiple tests between groups, alpha was adjusted according to the Bonferroni–Holm method. The eight subscales of the EDI and the three subscales of the EAT, respectively, were compared simultaneously between in-patients and out-patients by Multivariate Analysis of Variance (MANOVA).

Results

DSM-III diagnoses found in the groups studied are shown in Table II. Significant differences between subjects with anorexia nervosa only and those with anorexia plus bulimia were found in lifetime rates of major affective disorder ($P<0.02$) and total affective disorder ($P<0.03$). There was a significant difference between pure anorexics and pure bulimics in the lifetime rate of substance use disorders ($P<0.05$). The highest lifetime rates of major affective disorder were found in subjects with anorexia plus bulimia, the lowest in patients with anorexia nervosa only. Comparison of lifetime frequency of depressive symptoms for the three eating disorder groups revealed statistically significant differences (adjusted $P<0.05$) in symptom frequency for only two symptom areas: patients with only anorexia nervosa had a lower frequency of "feelings of worthlessness, self-reproach or guilt" (15.4%) than patients with anorexia plus bulimia (69.2%) and those with bulimia only (69.2%); they also had a lower frequency of "wishes to be dead" (7.7%) than patients with anorexia plus bulimia (57.7%). In general there was a tendency towards higher symptom rates for patients with anorexia nervosa plus bulimia than in the other two groups. Statistical comparison of all three eating disorder groups to the control group with only major depression showed significantly lower frequencies (adjusted $P<0.05$) for the eating disorder group in four areas: "loss of appetite" (eating disorders 34.6%, major depression 66.0%), "loss of energy" (42.3% *v.* 85.1%), "slowed thinking" (34.6% *v.* 72.3%) and "suicide attempts" (21.1% *v.* 65.2%).

Among the 23 patients with a lifetime diagnosis of major affective disorder, the onset of the affective disorder post-dated the onset of the eating disorder by at least one year in 16 cases (69.6%) and occurred within the same year in six (26.1%). Only one patient fulfilled criteria for major depression prior to onset of the eating disorder. This distribution is significantly different ($P<0.01$) from equal cell frequencies which are expected by chance. The mean age of onset of anorexia nervosa was 16.3 years (s.d. 4.4), of bulimia 17.9 years (s.d. 3.3), and of major affective disorder 19.8 years (s.d. 4.7). Rates of current DSM-III diagnoses in patients with active anorexia nervosa or bulimia at admission were 25.7% for major affective disorders, 34.3% for anxiety disorders, and 8.6% for substance use disorders. There were no significant differences either in rates of major affective disorder or in rates of anxiety or substance use disorders between the subgroups of eating disorders.

A trend was observed ($P<0.10$) towards a lower rate of major affective disorder in those patients who were interviewed as out-patients one year after hospital treatment and whose eating disorder was in remission. These patients had significantly lower scores in the EDI (MANOVA: $P<0.04$) and the EAT (MANOVA: $P<0.03$) than the in-patients, and only one met DSM-III criteria for major depression at the time of interview.

TABLE II
Lifetime DSM-III diagnoses in 52 patients with eating disorders

	AN *(anorexia only)*		*AN/B* *(anorexia and bulimia)*		*B* *(bulimia only)*		*Total*	
	(n = 13)		*(n = 26)*		*(n = 13)*		*(n = 52)*	
DSM-III diagnosis[1]	*n*	%	*n*	%	*n*	%	*n*	%
Affective disorders								
Major depression	2	15	12	46	6	46	20	39
Bipolar[3]	0		3	12	0		3	6
Total major affective disorders	2	15	15	58	6	46	23	44
Dysthymic	4	31	11	42	4	31	19	37
Atypical	2	15	0		1	8	3	6
Total[2]	4	31	18	69	7	54	29	56
Anxiety disorders								
Agoraphobia	0		4	15	1	8	5	10
Simple phobia	2	15	5	19	6	46	13	25
Social phobia	3	23	13	50	4	31	20	39
Obsessive-compulsive	2	15	4	15	1	8	7	13
Total[2]	4	31	14	54	8	62	26	50
Substance use disorders								
Alcohol abuse or dependence	0		4	15	2	15	6	12
Amphetamine abuse or dependence	1	8	3	12	2	15	6	12
Other substance use	0		1	4	1	8	2	4
Total[2]	1	8	7	27	5	38	13	25

1. Not using DSM-III exclusion criteria.
2. Number of patients who had at least one diagnosis within the group of disorders.
3. All three patients fulfilled criteria for depressed type of bipolar disorder.

Discussion

Other studies using operational diagnostic criteria have found somewhat higher lifetime rates of major affective disorders in anorexia nervosa (Hudson *et al*, 1983; Herzog, 1984; Piran *et al*, 1985) and in bulimia (Hudson et al, 1983; Walsh *et al*, 1985) than those presented here.

In contrast to previous reports (Hudson *et al*, 1983; Piran *et al*, 1985), the lifetime rate for major affective disorder in patients with anorexia nervosa only was significantly lower than in patients with anorexia plus bulimia, and also lower than in patients with bulimia only. This may be due to differences in duration of illness at the time of interview: the anorexic group differed significantly from the other two groups in this respect. Nevertheless, the data suggest an association of affective disturbance in patients with bulimic symptomatology.

Surprisingly, our sample also yielded high lifetime rates of anxiety disorders, the highest being in patients with bulimia only. These results are consistent with the findings of Hudson *et al* (1983) and other studies reporting a high level of anxiety symptoms, especially in patients with bulimia (Pyle *et al*, 1981).

Lifetime rates of depressive symptoms in our patients were generally comparable with those in patients with major depression. This, in combination with our data on depressive syndromes, leads one to assume that while eating disorder patients experience nearly the whole spectrum of depressive symptoms during their lifetime, half of them do not show enough concurrent symptoms to fulfil DSM-III criteria for major affective disorder.

How can these results be explained? Our finding that nearly half of the patients with eating disorders had displayed lifetime major affective disorder would coincide with the assumption that a substantial proportion of eating disorders might be primary affective disorders (Hudson *et al*, 1983). However, our results on onset of major affective disorder in eating disorders and the low rate of depression at follow-up do not support this hypothesis. Considering studies that have found improvement of depressive symptomatology parallel to improvement of eating disorder (Eckert *et al*, 1982; Fairburn, 1984), we propose a model consisting of several biological and psychological risk-factors for developing a diagnosable depressive syndrome in patients with an eating disorder.

First, specific symptoms (e.g. insomnia, concentration difficulties) which are used to diagnose 'depression' might be directly related to starvation or weight loss (Keys *et al*, 1950; Crisp *et al*, 1971). Secondly, specific abnormal eating behaviour or adaptation to starvation, which is not necessarily dependent on significant weight loss (Pirke *et al*, 1985), may cause changes in neurotransmitter systems that may be responsible for depressive symptoms (Wurtman, 1982). For instance, indications of long-lasting impairment of norepinephrine and serotonin turnover have been described (Kaye *et al*, 1985).

Thirdly, there are three psychological theories of depression that may explain part of depressive symptomatology as a consequence of eating disorder. Patients with long-lasting anorexia nervosa or bulimia usually show marked reduction of social activities (Garfinkel & Garner, 1982; Johnson *et al*, 1984): according to the theory of Lewinsohn (1974), this is related to a significant loss of reinforcers in important life areas and ultimately leads to depression. Anorexic and bulimic patients often feel out of control, ineffective, or helpless (Bruch, 1984; Johnson *et al*, 1984): in many patients those feelings might have been generalised to "learned helplessness" (Seligman, 1975), which is closely related to depressive symptomatology. The third psychological explanation is based on the cognitive theory of depression (Beck, 1976), which assumes that irrational beliefs and cognitive distortions may lead to depression. In fact, such beliefs and distortions have frequently been found in patients with anorexia nervosa (Garner & Bemis, 1982) and bulimia (Fairburn, 1984).

We hold none of these theories alone to be sufficient explanation of major depressive illness in eating disorders, but one might assume that in a substantial subgroup of patients, especially those with bulimic symptoms, these factors interact and lead to depressive symptomatology sufficient to warrant the diagnosis of a major affective disorder according to the DSM-III criteria.

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Prescriptive *v.* Exploratory Psychotherapy

Outcomes of the Sheffield Psychotherapy Project

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Prior research suggests that psychotherapeutic techniques which differ in their contents are quite similar in their outcomes. Outcome data are reported from a study designed to maximise sensitivity to technique effects on outcome in a clinically realistic setting, and to permit detailed analysis of the relations between content, immediate impact, and outcome of therapy. Forty professional and managerial workers with depression or anxiety received eight sessions of Prescriptive (cognitive/behavioural) and eight sessions of Exploratory (relationship-oriented) therapy in a crossover design, with each client seeing the same therapist throughout. Outcome was assessed by standard interview and questionnaire methods. The results favoured Prescriptive therapy, although this difference was of moderate extent. The outcome was largely unaffected by the order in which the two methods were offered.

A striking paradox dominates psychotherapy research. On the one hand, practitioners' allegiances to diverse methods are seemingly supported by the results of process research documenting substantial technical diversity among treatments (Strupp, 1955, 1957; Gomes-Schwartz, 1978; Brunink & Schroeder, 1979; Hill *et al*, 1979; Stiles, 1979; DeRubeis *et al*, 1982; Luborsky *et al*, 1982). On the other hand, most major reviews of psychotherapy outcomes have returned the "Dodo bird verdict" (Luborsky *et al*, 1975) that psychotherapy is effective, but that no substantial *differential* effectiveness has been demonstrated (Meltzoff & Kornreich, 1970; Roback, 1971; Smith & Glass, 1977; Smith *et al* 1980; Bergin & Lambert, 1978; Bergin & Suinn, 1975; Beutler, 1979; Frank, 1979). This paradox presents a dilemma: technical diversity despite equivalence of outcomes suggests that therapeutic techniques are irrelevant to outcome (Stiles *et al*, 1986).

Current studies of psychotherapy may be conceptualised in terms of the proposed solutions to this dilemma underlying the various research designs employed (Stiles *et al*, 1986). Within outcome research, the predominant set of solutions attributes the Dodo verdict to poorly conducted outcome research, and advocates increasing the precision with which patient, treatment, and outcome variables are measured and controlled (Paul, 1967; Kazdin, 1980; Rachman & Wilson, 1980). This approach confronts the conflicting requirements of clinical realism *v.* tight control over treatment and extraneous variables. Most of the well-controlled studies are 'analogue research' conducted with non-clinical populations, such as undergraduate students participating in trials of very brief treatments for minor and circumscribed problems, and receiving course credits for their participation (Shapiro & Shapiro, 1983). The art of outcome-research design is the art of compromise (Waskow, 1979). In particular, new ways must be found to approximate in realistic clinical settings the degree of experimental control readily attained in analogue research.

The first substantive issue addressed in the present study concerns the comparative efficacy of dynamic and humanistic therapies *v.* behavioural and cognitive therapies. Meta-analytic reviews of prior research (Smith *et al*, 1980; Shapiro & Shapiro, 1982; Shapiro, 1985) suggest a slight superiority of behavioural and cognitive therapies over dynamic and humanistic therapies, although the latter approaches are clearly at a disadvantage in terms of the quality and quantity of research conducted by proponents. These treatments often appear as quasi-control conditions which the investigators do not expect to be particularly effective, and the behavioural and cognitive orientations of most investigators are a likely source of bias, in view of the correlation obtained by Smith *et al* (1980) between outcome and the allegiance of the investigator inferred from the text of the report. Another limitation is that most of the better designed studies are analogue experiments with clinically unrepresentative samples (Shapiro & Shapiro, 1983).

The conclusions from meta-analysis correspond quite closely to those of the most substantial clinical studies of this comparison with depressed and anxious out-patients. For example, Sloane *et al* (1975) found that neurotic out-patients made similar gains with short-term, analytically oriented psychotherapy and behavioural therapy, with each treatment conducted by experienced practitioners, although ratings of global improvement and of social functioning based on a structured interview slightly favoured the behavioural-therapy group. Klein *et al* (1983) found essentially no difference in the treatment of phobias between behavioural therapy and dynamically oriented, supportive psychotherapy. However, McLean & Hakstian (1979) found greater improvement among depressive out-patients with behavioural therapy than with dynamically oriented psychotherapy, although the dependability of this finding is compromised by sole reliance upon mailed questionnaires.

This pattern of findings suggests that the behavioural and cognitive therapies may enjoy some superiority in the out-patient treatment of depression and anxiety, but that this finding is not unequivocal, and is subject to the qualification that the available data may be somewhat biased against dynamic and humanistic therapies. The Sheffield Psychotherapy Project was therefore designed to ensure maximal control over patient, therapist, and 'non-specific' factors which may account for the variability between previous findings, and to give both treatment approaches an equal chance of demonstrating their efficacy in the treatment of the depressed and/or anxious out-patients who are the primary recipients of psychological treatment.

Our Prescriptive treatment was designed to represent the cognitive and multimodal behavioural therapies (Kanfer & Goldstein, 1975) which emerged from the Shapiro & Shapiro (1982) meta-analysis of outcome research as particularly promising. This method incorporates anxiety-control training (Snaith, 1974), self-management (Goldfried & Merbaum, 1973), cognitive restructuring (Meichenbaum, 1977; Beck *et al*, 1980) and a work-oriented package derived from management-training literature to suit professional and managerial employees (Hackman & Suttle, 1977; Taylor, 1979; Ferner, 1980).

The dynamic and humanistic therapies are represented by Exploratory therapy, based upon Hobson's (1985) Conversational Model. This method synthesises interpersonal and client-centred concepts and focuses upon the use of the therapeutic relationship as a vehicle for the revelation and resolution of interpersonal difficulties and conflicts. Prior research (Goldberg *et al*, 1984; Maguire *et al*, 1984) has specified constituent techniques within this method, and shown that these can be taught to psychiatrists in training. A videotape microteaching package and brief manual are available (Margison & Hobson, 1983).

Our objectives in designing this study were to maximise its sensitivity to treatment effects and permit high-power examination of correlations between process, impact, and outcome variables (to be reported subsequently). We selected a cross-over design with each patient seen by the same experienced therapist in both periods, to maximise control over patient and therapist variables. Since our primary interest was in comparing the processes and outcomes of different therapies, and in view of evidence for the overall efficacy of psychological treatments (Smith *et al*, 1980) and the logistic and ethical difficulties associated with placebo or no-treatment controls in a realistic clinical setting, no such control groups were employed.

A second substantive question addressed by this study concerned sequence effects within eclectic therapy. Several authors have described the complementary use of behavioural and psychodynamic therapies (Birk & Brinkley-Birk, 1974; Wachtel, 1977; Birk, 1979; Bernstein, 1984). Most authors have used one treatment adventitiously when progress in the other appears impeded in some way (Brady, 1968), but it has been argued (Luborsky, 1984) that purity of modality is an essential feature of eclectic therapy; that is, one type of treatment should be completed and then followed by another according to plan, rather than the therapist being allowed to respond to day-to-day expressions of client needs, which can permit avoidance of difficult issues.

If eclectic treatment packages are to be constructed along these lines, the question arises as to which therapy should be offered first. When behavioural techniques precede psychodynamic, this may allow behavioural work to deal with immediate crises prior to psychodynamic exploration of the interpersonal issues which may have originally precipitated the crisis (Bernstein, 1984), or behavioural techniques may be used to bring into the open anxieties or conflicts and thereby facilitate the psychodynamic work which follows (Levin *et al*, 1968). Conversely, brief psychodynamic work may be usefully followed by practice and rehearsal of new adaptive techniques and their generalisation (Marmor, 1979).

In this context, it is of interest to use a cross-over design to address the question of which of the two orders, Exploratory followed by Prescriptive or Prescriptive followed by Exploratory, is the more effective.

Method

Measures

Our approach to outcome assessment was based upon the recommendations of the National Institute of Mental

Health review of psychotherapy change measures (Waskow & Parloff, 1975), adapted to reflect prevailing practice in the UK. Measures used included substantial interviews assessing psychiatric symptomatology and social role performance, the Present State Examination (PSE; Wing *et al*, 1974), and the Social Adjustment Scale (SAS; Weissman & Paykel, 1974); and four self-report questionnaires: the Beck Depression Inventory (BDI: Beck *et al*, 1961), a standard symptom checklist, the SCL–90 (Derogatis *et al*, 1973) and the O'Malley & Bachman (1979) eight-item revision of the Rosenberg (1965) self-esteem scale, presented in a seven-point format (SE). The PSE yielded a total symptom score suitable for parametric analysis, obtained by summing the scores obtained for each item. The Index of Definition (Wing *et al*, 1974) was obtained from the ID-CATEGO program at the Institute of Psychiatry. The SAS was represented by the grand mean of all items on every scale. On each occasion, patients were asked to consider the month prior to assessment when responding to the PSE and SAS questions. At intake, all patients were required to score at least 8 on the 30-item version of the General Health Questionnaire (GHQ; Goldberg, 1972), and to attain a total symptom score of at least 14 on the PSE, although those with obsessional or psychotic symptoms on the latter instrument were excluded as unsuitable for brief, out-patient psychotherapy.

Recruitment of patients

Patients were referred by general practitioners, psychiatrists, and company doctors, who were posted information publicising our specialist clinic offering psychological therapy to professional and managerial workers suffering from depression and/or anxiety. Many patients, eventually referred by general practitioners, made their initial contact with our clinic in response to publicity materials distributed in appropriate employment settings by welfare and personnel staff. Initial screening criteria excluded people not complaining that their work was affected by their problems, those with a continuous history of psychiatric disorder extending more than two years prior to the time of referral, and those who had during that period undergone psychological treatment sufficiently similar to pre-empt either of our two treatment modalities. We also excluded those who had undergone a significant change in psychotropic medication during the three months prior to referral. Such significant changes included commencing or finishing a course of treatment, or switching from one type of medication to another (such as from benzodiazepines to antidepressants).

Potential patients were mailed the GHQ and SCL–90. On receipt of the completed questionnaires, the intake assessment was scheduled with a research assistant, who administered the PSE and SAS, together with questionnaires including BDI and SE. Patients were excluded at this stage when interviews revealed psychotic or obsessional symptoms sufficiently severe to be scored 1 or more on the PSE, or insufficient PSE symptomatology (total score below 14). The objectives and requirements of the research programme were set out in a three-page outline handed to accepted patients after the intake assessment. This presented the goals of the project as being to "find out more about which of two well-established methods is more helpful to what kinds of people", and covered the structuring of treatment into two distinct phases, the audio-recording of all sessions, and the completion of impact and outcome measures.

In all, 108 individuals were referred to or approached the clinic during the period of recruitment to the study. Of these, 75 completed the GHQ and SCL–90, and 55 attended for intake assessment. Forty-eight were randomised, having met the inclusion criteria. Two of these (one assigned to each order of treatments) decided against therapy. A further six (three assigned to each order) failed to complete treatment, four terminating during Prescriptive and two during Exploratory therapy. Three of these six completed one eight-session phase of treatment and showed typical gains over that phase, but did not wish to continue treatment after 1–5 sessions of the second phase. The remaining three discontinued after 3–6 sessions of the first phase; these patients did not appear to form a commitment to therapy. The present analyses concern 40 patients completing therapy, whose mean age was 40.7 years (s.d. 8.4 years).

Intake and outcome assessments

There were four assessments: intake, mid-therapy (between the two treatment phases), end of therapy, and a three-month follow-up. Interviews were conducted by social science graduates who had undergone training in the PSE and SAS. The BDI, SCL–90 and SE were administered in a single package by the interviewer, except that the intake SCL–90 scores were based on the mailed questionnaire. In most cases, patients were interviewed by the same research assistant at each of their four assessments. Where this was not possible, data were based upon ratings of all four assessments by the same interviewer (who listened to tapes of assessments carried out by her colleague), so as to reduce error due to variations between assessors. Assessors were not informed of the order in which each patient was assigned to treatment.

Patients were informed by both assessor and therapist that outcome data were not made available to the therapist until after the completion of treatment. All patients were offered a single consultation with their therapist after the follow-up assessment, at which their progress could be reviewed and they could receive advice concerning the appropriateness and availability of further treatment.

Assignment to therapist and order of treatment

Patients were assigned to therapists in accordance with scheduling and workload constraints. As far as practicable, we balanced the numbers of each sex seen by each therapist. Other commitments entailed that 34 patients be shared equally between the two authors, with the other two therapists contributing four and two patients. All therapists were full-time research clinical psychologists, selected for an eclectic orientation valuing both treatment modalities. Their initial professional training (at UK master's level) represents a level of experience intermediate between master's and doctoral levels in the USA. At the outset of the project, the first author had ten years' post-doctoral research and clinical experience. The second author began pilot work for the project upon completion of her master's training, and had had experience in counselling prior to that training. The other two project therapists had some three years' clinical experience and training in psychotherapy after their initial professional training. All therapists had been trained in both behavioural and relationship-oriented therapies prior to taking part in the study.

Assignment to order of the two treatments was determined by the toss of a coin for each alternate patient of a given sex assigned to each therapist, with the next patient of that sex seen by that therapist automatically assigned to the reverse order. This procedure counterbalanced any maturational changes in therapist behaviours over the course of the study. An assignment error by the first author resulted in an imbalance between the groups; 19 patients received Exploratory therapy first, and 21 received Prescriptive therapy first.

Scheduling of treatment and assessment sessions

Treatment began approximately three weeks after the intake assessment. Each treatment phase comprised eight weekly 1-h sessions. The mid-treatment assessment occurred 1-3 weeks after the eighth treatment session, and the second phase of treatment began 1-3 weeks later. The post-treatment assessment was scheduled 1-3 weeks after the last session, and the follow-up assessment three months later.

Treatments

The design of each treatment in the form of an eight-session package was evolved in preliminary pilot work, in the context of a review of the above-cited clinical texts. Fifteen-page manuals were written to define the objectives, strategy and techniques of each treatment. A summary of the contents of each follows.

Prescriptive therapy. This treatment emphasises the provision by the therapist of self-management strategies for application by the patient. The focus of therapy is upon the patient's behaviour outside the sessions. A wide range of techniques is outlined in the manual, divided into four areas: (a) anxiety-control training, including relaxation, anxiety management and graded exposure to anxiety-provoking situations both in imagination and *in vivo*; (b) self-management procedures, including contracting, behavioural tasks, guidance on changing the environment (e.g. via timetabling), self-monitoring (e.g. via diaries), and assertion training; (c) cognitive restructuring, including systematic rational restructuring, engendering more realistic goals and norms, engendering expectations of controllability, and challenging dysfunctional attributions; (d) a job-strain package, including analysis and reduction of friction at work, cognitive and thought-stopping methods of countering worry, time management and cognitive restructuring of work attitudes, delegation training, and functional analysis and remediation of concentration and memory problems. With each patient, elements are drawn from each of the four areas; there is no expectation that every technique can be included within eight sessions. Maintenance strategies are emphasised throughout, with the explicit rationale that therapy provides the patient with resources which he or she must continue to apply and develop after the completion of treatment.

Exploratory therapy. Key features of this treatment include the following: (a) the assumption that the patient's presenting problems arise from disturbances of significant personal relationships; (b) the creation within the therapy situation itself of a relationship in which the patient's interpersonal problems are revealed, explored, understood and modified by the testing-out of possible solutions which are generated in the dialogue (this contrasts with the prescriptive treatment's assumption that the therapeutic relationship is primarily a vehicle for the transmission from therapist to patient of knowledge, skills and principles to assist the patient in his life outside therapy); (c) the adoption by the therapist of conversational strategies designed to promote the process defined in (b) above, including *negotiation*, a language of *mutuality*, the use of *metaphor* to enhance the immediacy and 'wholeness' of experienced affect, *focusing* on the here and now experience of the patient in the interview, and the offering of *hypotheses* ranging from *understanding* hypotheses expressing the therapist's view of the patient's experience, through *linking* hypotheses suggesting connections between different experiences of the patient's, to *explanatory* hypotheses suggesting causes or reasons for behaviour and experience; (d) emphasis upon interpersonal problems arising within professional and managerial employment. In relation to maintenance, the explicit rationale of this method is to furnish links between the therapeutic relationship and other relationships so that insights and new solutions to interpersonal problems will generalise outwards from therapy.

Implementation

Therapy took place within our research clinic, in a city-centre university setting. The project therapists met at least once weekly for peer supervision, with every case presented after the second session within each of the two treatment modalities. Expert practitioners of the two modes took part in a total of some 60 supervision sessions during the course of the study. The work of the supervision sessions was directed toward the delivery of clinically representative and effective therapy, as well as to the maintenance of therapists' adherence to the treatment manuals. Audio recordings were made of every session for research and supervision purposes. Therapists made process notes for each session, using forms designed for the two treatment modalities. These notes were taken during the Prescriptive sessions, and after each Exploratory session.

At the first treatment session, the therapist reminded the patient of the research requirements, particularly of the structuring of treatment into two distinct phases, and outlined once more the rationales of the two methods. Although the great majority of problems were tackled in both phases, therapists were during the first phase at liberty explicitly to defer work distinctly appropriate to the second phase. Efforts were also made to shape the first phase as a distinct entity with goals to be attained within the eight-session span. Thus, as the eighth session approached, there was an increasing emphasis upon maintenance strategies in Prescriptive therapy and upon termination work in Exploratory therapy (expressed in terms of the loss of the therapeutic relationship). Particular attention was paid to the negotiation of the transition between treatment modalities. The final session of treatment necessarily involved some review of the work of both treatment modalities. We present elsewhere a discussion of these and other aspects of the implementation of the study design from a practitioner's perspective (Firth *et al*, 1986).

Manipulation check

Adherence to the treatment protocols was confirmed via a manipulation check of the verbal-response mode usage of therapists (Shapiro *et al*, 1984) in the two treatments. These data, reported fully elsewhere (Hardy & Shapiro, 1985), show substantial and predicted differences in the interpersonal intents or functions of therapist utterances as between the two treatment modalities. Excerpts selected at random from the treatment of patients included in the present study revealed greater use of interpretation, exploration, and process advice during exploratory therapy, and greater use of question, information, and general advisement during prescriptive therapy. Despite the control for individual therapist factors secured by the use of the same therapist in both treatments with each patient, differences in the percentage of therapist responses in a given category ranged from more than twofold to over sixfold.

Results

Initial comparability of groups

Table I shows the means and standard deviations obtained on each outcome measure at each of the four assessments. For SCL-90 and SE, the groups were essentially equivalent at intake ($t<1$ in each case). In terms of the PSE Index of Definition (ID) at intake, all but one patient in each group was at the 'threshold' level of caseness ($\mathrm{ID}=5$) or above. Despite random assignment, however, the Exploratory–Prescriptive (EP) group was somewhat more disturbed at intake on the SAS ($t(38)=2.07$, $P<0.05$) and marginally more symptomatic on the PSE ($t(38)=1.67$, $P=0.10$) and BDI ($t(38)=1.43$, $P=0.16$). The two groups were of similar age ($t(38)<1$). The EP group comprised 11 males and 8 females, while the Prescriptive–Exploratory (PE) group comprised 12 males and 9 females. Educationally, the EP group comprised 10 with university or college education, 4 with professional or other post-school qualifications, and 5 with no such qualifications; the PE group comprised 13 with university or college education, 2 with professional or other post-school qualifications, and 6 with no such qualifications. In terms of jobs held, the EP group comprised 4 teachers, 6 managerial/financial workers, 6 helping professionals, 2 technical/engineering workers, and 1 civil servant; the PE group comprised 7 teachers, 5 managerial/financial workers, 4 helping professionals, 2 technical/engineering workers, and 3 civil servants.

Clinical severity at intake and clinical significance of change

Table I shows that, at intake, this was a population with mean levels of symptomatology and distress well into the clinical range of severity. In terms of PSE ID level, 7 patients were clear 'cases' ($\mathrm{ID}=6$) and 31 were at the 'case threshold' ($\mathrm{ID}=5$). Social adjustment appeared less severely disturbed, however, reflecting the restriction to those currently employed in professional and managerial jobs. The most rigorous of the criteria discussed by Jacobson *et al* (1984) for the evaluation of the clinical significance of change occurring in psychotherapy research studies is based on the proportion of patients attaining end-of-treatment scores two or more standard deviations beyond the intake mean. The implication of such end-of-treatment scores is that individuals attaining them cannot be reasonably viewed as members of the intake population, irrespective of the degree of elevation of that population's

TABLE I
Means and standard deviations at each assessment

Order	*n*	*Assessment*	*Measure*									
			PSE		*BDI*		*SCL-90*		*SAS*		*SE*	
			Mean	*s.d.*	*Mean*	*s.d.*	*Mean*	*s.d.*	*Mean*	*s.d.*	*Mean*	*s.d*
EP	19	Intake	23.3	6.2	22.0	7.3	131.7	42.2	2.30	0.28	20.1	10.1
		Mid	16.2	6.7	14.2	7.9	97.6	46.1	2.02	0.32	24.4	10.2
		End	12.4	7.3	8.6	6.1	57.5	47.1	1.75	0.30	32.2	10.2
		Follow-up	11.1	7.3	7.9	6.1	43.5	28.9	1.71	0.38	32.4	9.5
PE	21	Intake	20.5	4.0	19.0	6.1	122.9	43.9	2.09	0.36	21.7	12.6
		Mid	11.1	3.6	9.3	5.5	67.7	21.3	1.78	0.39	28.0	9.9
		End	9.1	4.7	8.2	5.1	56.9	32.5	1.64	0.31	31.5	8.2
		Follow-up	9.4	7.0	8.6	8.2	60.0	42.3	1.59	0.39	32.2	10.0

TABLE II
Percentage of patients recovered after treatment[1]

Assessment	*Measure*				
	PSE	*BDI*	*SCL–90*	*SAS*	*SE*
Mid	38	32	13	18	2
End	56	37	47	32	10
Follow-up	65	54	43	41	10

[1] Recovery is defined as two or more standard deviations beyond pretreatment mean.

scores relative to a normal population. Table II shows the percentages of patients meeting this criterion of recovery on each measure at mid-treatment, end of treatment, and follow-up. On this demanding criterion, around half the patients improved on the symptomatic measures (PSE, BDI and SCL–90), somewhat fewer improved on the SAS, but few changed with respect to self-esteem. In terms of PSE 'caseness', by the end of treatment only 12 patients were at the threshold (ID = 5) and none was above this value. Follow-up data showed essentially no change from end of treatment.

Analysis of treatment effects

To evaluate the differential effects of treatments and of their order upon outcome in the context of the above-described differences between the two groups at intake, two alternative methods of analysis were employed. Residual gain scores, from which the effects of each individual's intake score are statistically removed, are often favoured (e.g. Cronbach & Furby, 1970; Mintz *et al*, 1979). However, residual gain scores only correct for initial differences to the extent permitted by the size of the correlation between scores obtained at the different assessments. In the present data, correlations between intake and mid-treatment scores ranged from 0.32 for the BDI to 0.61 for the SE measure; correlations between mid-treatment and end-of-treatment scores ranged from 0.39 for the BDI to 0.80 for the SAS; correlations between intake and end-of-treatment scores ranged from 0.20 for the SCL–90 to 0.46 for the SAS; and correlations between intake and follow-up scores ranged from 0.14 for the SCL–90 to 0.39 for the SAS. The modest size of most of these correlations suggested that simple change scores, which by definition correct fully for the effects of earlier measures, should be considered alongside residual gain scores. All analyses of treatment effects were therefore performed twice, using both residual gain and simple change (arithmetic difference) scores.

The combination of data from the two periods of a cross-over design is legitimate only in the absence of interaction between treatment and period (Hills & Armitage, 1979; Armitage & Hills, 1982). Such an interaction is identical with the between-groups difference in change over the two periods combined (from intake to end of treatment). This between-groups difference was also of interest in relation to our examination of order effects in eclectic therapy. The first step in the analysis of each outcome measure, therefore, was the comparison of residual gain scores for the two groups, comprising the end-of-treatment scores expressed as residuals from the values predicted from the intake scores. The parallel analysis of simple change scores compared end-of-treatment minus intake scores in the two groups.

As a longer-term test of the effect of the order of treatments upon outcome, residual gain scores comprising the follow-up data expressed as residuals from the values predicted from intake scores were compared for the two groups of patients. Similarly, simple change scores comprising follow-up minus intake scores were compared in the two groups.

Changes occurring during the two treatments were compared for each period separately, using residual gain scores comprising the mid-treatment values adjusted for intake scores to represent change during the first period, and residual gain scores comprising the end-of-treatment values adjusted for mid-treatment scores to represent the second period. Similarly, simple change scores comprising mid-treatment minus intake scores, and end-of-treatment minus mid-treatment scores, were compared to assess change in each treatment for each period in turn.

Before combining the two periods of the cross-over, a liberal significance level of 0.10 was set for rejection of the null hypothesis of no interaction between treatment and period. In the absence of interaction in the residual gain scores, residual gain scores for both periods (with mid-treatment scores controlled for intake scores and end-of-treatment scores controlled for mid-treatment scores) were analysed following the ANOVA method of Hills & Armitage (1979). In the absence of interaction in simple change scores, these were analysed similarly.

Effect of treatment order on end-of-treatment scores

Analysis of residual gain scores based on the end-of-treatment scores expressed as residuals from the values predicted from intake scores revealed no difference in overall change between patients assigned to the two orders of treatment. With respect to the BDI, SCL–90, SAS, and SE measures, the t value for this comparison was less than 1. The remaining measure, the PSE, also showed no significant difference between the groups ($t(37) = 1.13$, $P > 0.25$). In the analysis of simple change scores, the PSE, SCL–90, and SE scale yielded t values less than 1. The BDI and SAS yielded t values of 1.40 ($P = 0.17$) and 1.17 ($P = 0.25$) respectively. Of the ten comparisons yielded by the five measures and two methods of analysis, the arithmetic values of six favoured the EP group and four the PE group. These findings show an absence of interactions between treatment and period indicative of carryover effects. It was therefore appropriate to test for treatment effects over both periods of the study via within-subjects analysis of the crossover for all five outcome measures, and for both residual gain and change scores.

Effect of treatment order on follow-up scores

Analysis of residual gain scores comprising the follow-up scores expressed as residuals from the values predicted from intake scores revealed no difference in

TABLE III
Residual gains during Prescriptive and Exploratory therapies

Period	*Treatment*	*Measure*				
		PSE	*BDI*	*SCL–90*	*SAS*	*SE*
First	Prescriptive	1.70	1.86	12.64	0.04	1.30
	Exploratory	−1.88	−2.05	−13.98	−0.05	−1.44
	t	2.35**	1.90*	2.62**	1.04	1.08
Second	Prescriptive	−0.20	0.68	9.17	0.03	1.56
	Exploratory	0.17	−0.55	−7.43	−0.03	−1.33
	t	<1	<1	1.53	<1	1.32
Combined	t	1.35	1.97*	2.41**	1.38	1.75*

$^{*}P<0.10$; $^{**}P<0.05$; $^{***}P<0.01$.

overall change from intake to follow-up between patients assigned to the two treatment orders with respect to the PSE, BDI, SAS, or SE measures ($t<1$ in each case). For the SCL–90, however, there was a non-significant trend towards greater improvement at follow-up for the EP group ($M=9.47$) than for the PE group ($M=-8.12$; $t(37)=1.51$, $P=0.14$). In the analysis of change scores from intake to follow-up, the PSE, SAS, and SE all yielded t values less than 1, while the BDI and SCL–90 both yielded non-significant advantages to the EP group, with t values of 1.38 ($P=0.17$) and 1.51 ($P=0.14$) respectively. The arithmetic values obtained for seven of the ten comparisons favoured the EP over the PE group. Notwithstanding these non-significant trends in favour of the Exploratory–Prescriptive order, both end-of-treatment and follow-up results suggest that an eclectic package combining Prescriptive and Exploratory therapies is almost equally effective irrespective of the order in which the two treatments are offered.

Comparative outcomes of Prescriptive and Exploratory therapies

Table III shows the mean residual gains made by patients undergoing each treatment in each period of the study, together with the means of the two periods for each treatment. By definition, the mean residual gain across all 40 patients on any measure for either period is equal to zero; a positive value in Table III denotes a group whose scores at the end of the relevant period of treatment are more favourable than would be predicted from knowledge of their scores at the beginning of that period, while a negative value denotes scores less favourable than so predicted. The difference between each pair of means was evaluated by a t-test, the results of which are also presented in Table III. The within-subjects analysis of the two periods combined is represented by the final row of t values in the table. The pattern of these data clearly favours Prescriptive therapy, whose residual gains were positive in all but one of the ten comparisons. In the first period, gains were significantly greater for Prescriptive therapy on the PSE and SCL–90, and marginally greater on the BDI, although no treatment effects were significant in the second period. Data for the two periods combined indicated a significant advantage to Prescriptive therapy on the SCL–90, accompanied by marginally significant differences in the same direction on the BDI and SE measures.

The corresponding analysis of simple change scores is presented in Table IV. For each measure, a larger score means greater improvement. All ten comparisons show

TABLE IV
Change scores during Prescriptive and Exploratory therapies

Period	*Treatment*	*Measure*				
		PSE	*BDI*	*SCL–90*	*SAS*	*SE*
First	Prescriptive	9.38	9.67	55.19	0.31	6.33
	Exploratory	7.05	7.79	34.64	0.28	4.32
	t	1.32	<1	1.56	<1	<1
Second	Prescriptive	3.72	6.00	40.71	0.29	8.65
	Exploratory	2.05	1.09	10.81	0.14	4.40
	t	<1	2.26**	2.67**	2.11**	1.72
Combined	t	1.36	1.68*	2.76***	1.40	1.50

$^{*}P<0.10$; $^{**}P<0.05$; $^{***}P<0.01$.

arithmetic differences in favour of Prescriptive therapy, which attain significance during the second period of the study for the BDI, SCL-90 and SAS. The within-subjects analysis of effects for the two periods combined shows a significant advantage to Prescriptive therapy on the SCL-90, and a marginally significant difference in the same direction on the BDI. Although residual gain scores suggest stronger effects in the first period, while simple change scores do so only in the second, the results presented in Tables III and IV show a consistent difference in favour of the Prescriptive treatment.

In view of the similarly high proportion of patients in the two groups who were at or above the threshold of psychiatric caseness at intake, an additional measure of treatment effect was obtainable from the proportions of patients in each group who remained cases (ID = 5 or 6) at mid-treatment. Nine patients were still cases after exploratory therapy, while seven were still cases after prescriptive therapy; this difference was not statistically significant (chi-square < 1). A similar analysis of change during the second period was equally inconclusive: seven of the PE group, and five of the EP group, were threshold cases at the end of treatment.

Discussion

Before considering the answers to our substantive questions suggested by these results, it is worth noting the absolute magnitude of change observed during therapy. As shown in Tables I and II, this was at intake a clinically distressed sample, and large changes occurred, particularly on the symptomatic measures. For example, around 11 PSE symptoms were lost by the average patient in the study. Many patients made sufficient gains to be deemed 'recovered' (i.e. outside the range of intake scores) by the end of treatment. In terms of the PSE ID criterion, nearly all the patients were at or above threshold at the outset of treatment, but only a quarter remained so after treatment. These gains were maintained at three-month follow-up. Although these absolute changes reflect non-specific factors and 'spontaneous' change in addition to effects attributable to specific treatment techniques, they suggest that 'real' therapy was taking place, despite research constraints (Firth *et al*, 1986).

Overall, our findings suggest that Prescriptive therapy was somewhat more effective than Exploratory. This difference was most consistent and substantial over the study as a whole for the SCL-90, where more than twice as much improvement was observed during Prescriptive than during Exploratory therapy. Analysis of residual gains on the PSE and BDI showed greater reduction of psychiatric symptoms in Prescriptive than in Exploratory therapy during the first period of the cross-over, while simple change scores on these measures were greater during Prescriptive than Exploratory therapy during the second period of the study. The two remaining outcome measures (SAS and SE) showed trends in the direction of greater efficacy for Prescriptive therapy.

These results are evidence against the view that all therapies have equivalent outcomes. They are consistent with the suggestion that findings of equivalent outcomes may be due to poor control over those extraneous therapist, technique, and patient variables which were well controlled by our design. This control was achieved by the use of the cross-over design with the same therapist for each patient throughout; the manualisation, weekly peer supervision and monitoring of technique; and the socioeconomic homogeneity of our patient population. All these design features enhanced the study's sensitivity to treatment differences.

On the other hand, our findings as a whole do not imply a massive difference between the treatments. The PSE data, for example, show a difference around 2 points, equivalent to two symptoms falling from marked (2) to moderate (1) or dropping below the level required for a rating of 1, in the context of the 11-point overall improvement during treatment. The clinically meaningful caseness criterion of the PSE ID did not differentiate the two treatments. BDI changes differed by about 3 points, while overall changes during treatment averaged around 12 points. Even allowing for maturational and non-specific contributions to the substantial overall changes, these data attest only to a modest treatment effect, particularly in view of the study's sensitivity to it.

Advocates of Exploratory therapy could argue that our design was disadvantageous to that treatment. Eight sessions may be too few for this method to be effective, with existing defences unsettled but too little time in which to establish new ways of coping. In addition, it is possible that a delayed measure of effectiveness, unconfounded with any alternate treatment, would be more favourable to Exploratory therapy. The measures used may be insufficiently sensitive to intrapsychic change, although it is noteworthy that even SE data tended to favour Prescriptive therapy. On the other hand, any investigator bias was more likely toward rather than against Exploratory therapy, given our research team's concern to redress the balance of opinion within a national professional culture which tends to favour behavioural over non-behavioural methods (Shapiro & Shapiro, 1977).

With respect to the ordering of treatments within an eclectic package, our findings comparing the gains made by patients receiving the two treatments in different orders were almost uniformly equivocal.

Of 20 tests of this order effect, none attained statistical significance, and 13 yielded arithmetic values favouring the EP order, as against 7 favouring the PE order. Overall, these data do not support one ordering as more favourable than the other.

Although these findings can be usefully viewed as complementary to previous studies suggesting that cognitive and behavioural therapies may be a little more effective than are dynamic and humanistic treatments, it would be inappropriate to generalise from the present comparison between two specific treatment models to all treatments within these two broad classes.

Our two parallel analyses, of residual gain scores and of simple change scores, yielded findings which differed in detail but were consistent in favouring Prescriptive over Exploratory therapy. As far as residual gain scores are concerned, the treatment differences at mid-treatment are presumably in part a reflection of the intake differences, which were incompletely controlled by the residualising procedure, given the modest correlations between measures obtained at successive outcome assessments. On the other hand, no such reservation qualifies the interpretation of the treatment effects on simple change scores during the second period of the study.

In the light of the findings obtained, the choice of a cross-over design may be seen to have somewhat enhanced the study's detection of treatment effects. The inclusion of the second period, in which patients received the alternate treatment, allowed detection of a significant treatment effect on the SAS, and confirmation of an effect on the BDI which was only marginally significant during the first period. Furthermore, the effects of treatment order within the combined eclectic package can only be ascertained by such a design.

In addition, a cross-over design makes available invaluable process data in which patients are their own controls. Although this paper has focused exclusively upon the outcomes of our two treatments, presenting the project as a 'horse race' between them, the full benefits of our research strategy will be realised when these outcome data are linked with further measures concurrently obtained on these patients, including verbal response modes utilised during sessions by therapists and patients (Stiles & Sultan, 1979; Stiles, 1984), evaluations of the immediate impact of each session (Llewelyn & Hume, 1979; Stiles, 1980; Elliott *et al*, 1985), and week-to-week changes in self-rated target problems (Mulhall, 1976). To the extent that our outcome findings imply differential effectiveness of the two treatments, these process measures will contribute to our search for active ingredients to account for this difference. Process analyses will also permit evaluation of contrasting mechanisms of change postulated for the two treatments, whereby divergent pathways may lead to comparable final outcomes.

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Explaining and Understanding in Psychopathology

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Psychoanalytical methodology has been described as causal explanation or hermeneutic understanding. This methodological dichotomy has been introduced into psychopathology by Karl Jaspers. Contemporary authors' contributions in the area are discussed. Although these authors accept a role for both methods, they agree with Jaspers that psychoanalysis should be subjected to the logical limitations of hermeneutic analysis. A logical framework for the interaction of explaining with understanding is presented and discussed in relation to psychiatric research.

In a recent critical analysis, Gellner (1985) suggested that a "biohermeneutic fusion" of concepts – that is, the conflation of meaningful and causal connections (see below) – has helped psychoanalysis to assert and maintain its role as a healing discipline. Understanding meaningful connections by itself, he argues, is not sufficient to capture the imagination of the sufferers. After all, meanings can be found without professional help. A purely biological theory, on the other hand, demeans man by reducing him to a "bundle of animal drives". Mixing both elements gives meanings increased depth and authority (by virtue of being linked to biological forces) and helps patients to retain their dignity in the face of biological determinism (thanks to the meaning which has to be analysed on a personal level). Thus, "bio-hermeneutics is simultaneously reductive (thereby giving control) and restorative (dignity-preserving)" (Gellner, 1985).

The interplay of causes and meanings, or explaining and understanding, in psychoanalysis has been noted earlier: the first discussion of psychoanalytic methodology under these headings was carried out by Karl Jaspers in his *General Psychopathology* (Jaspers, 1963).

This paper will examine some of the limitations of explaining and understanding, as conceived by Jaspers, as methods of psychopathological investigation. Contemporary definitions of psychoanalysis as a discipline which uses both explaining *and* understanding will be discussed in an attempt to define the methodological constraints imposed upon psychoanalytic theory and practice. A logical framework for the interaction of both methods in psychopathology will be proposed.

Jaspers: Psychoanalysis is an hermeneutic discipline mistaking itself for a causal science

Jaspers' concept of *causal analysis* is derived from Hume's (1962) regularity model of causality: the discovery of regular temporal conjunctions between two kinds of events leads to the postulation of a general law, which *explains* this conjunction. The general law, in turn, can be used to predict future conjunctions of events of the same kind.

Empathic understanding is a method modelled by Jaspers on the hermeneutic disciplines, philology and history. *Hermeneutics* (literally, the art of interpretation) always starts with a preliminary understanding. A facial expression with closed eyes and tears rolling down the cheeks is recognised as crying. This momentary facial expression, however, can have many, sometimes contradictory, meanings. It makes a difference whether someone has just received news of the death of a close relative, has been told a joke, or has been peeling onions; an examination of the context of the expression is necessary to define it more precisely. Knowing that a patient has a hysterical personality shows the crying in a different light than would the detection of organic brain disease, like pseudo-bulbar palsy. Conversely, the context is modified by appreciation of the detail. For example, crocodile tears which can be wiped away as soon as their purpose is achieved might point towards a particular kind of personality.

Hermeneutic analysis, therefore, follows a movement rather like ascending a spiral staircase. The preliminary understanding is modified by a successive appreciation and reappreciation of details and context. If new observational material is gathered at each stage, an infertile circle is avoided, and more and more precise understanding can be achieved. This procession from the part to the whole and back can never be complete. Understanding can, therefore, never be final. Consequently, contrary interpretations are often equally plausible. One can understand how a handicap could lead to withdrawal from competition, but one can equally understand that a handicapped person might try to make up for disadvantages by developing his remaining capabilities and skills even to the degree of surpassing his healthy peers. Although meaningful connections can be found in the analysis of human expressions and artifacts, understanding has its limits: one cannot *understand* empathically why a patient with pseudo-bulbar palsy cries or laughs. The finding that other patients with similar brain lesions show the same behaviour, offers a causal *explanation*.

It follows that both explaining and understanding are applicable in the human sciences. Where understanding ends, explanations are still possible, and where understanding is possible using objective data this might point towards a causal connection. In order to establish a causal law, however, external data will have to be adduced to avoid circular arguments. Violent behaviour cannot be explained in terms of an explosive personality if this personality is diagnosed by the observation of just this violent behaviour. An observation external to the process of understanding is necessary for a causal explanation, as for example the detection of EEG abnormalities in this kind of personality disorder (Williams, 1969).

Understanding by means of conjectured mechanisms like the ego, the unconscious, or libido, is, in Jaspers' terminology, 'as if' understanding. These mechanisms have to be postulated in order to explain non-understandable observations like conversion symptoms or dissociative states. They lack, however, externality and independence from the process of empathic understanding and, therefore, lead to circular rather than causal statements (Nagel, 1959). Jaspers comes to the conclusion that psychoanalysis is an understanding psychology, which frequently uses 'as if' understanding, but is presented by Freud as a causal science similar to the natural sciences.

Explaining and understanding in contemporary descriptions of psychoanalysis

More recently, psychoanalysis has been described as either a causal science (Hartmann, 1964; Rubinstein, 1973) or as a hermeneutic discipline (Rycroft, 1966; Lacan, 1977). Other authors have commented on the interaction between explaining and understanding in psychoanalytic theory and practice. Three of the latter, Ricoeur, Habermas, and Bhaskar, will now be discussed in more detail.

Jaspers argued for a total disjunction between causal and meaningful connections in psychopathology (Jaspers, 1963), which led to his definition of psychoanalysis as a hermeneutic discipline. Ricoeur's refusal to accept this disjunction becomes the basis of his interpretation of Freudian theory (Ricoeur, 1970). He identifies two elements in Freudian theory: libido theory, which uses dynamic explanations, modelled on a physical theory of hydraulics; and the interpretation of dreams, parapraxes, and the like. Ricoeur's central question is how a dynamic explanation can be involved in an interpretation dealing with meanings, and conversely, how an interpretation can be an aspect of a dynamic explanation. If psychoanalysis was made an operational science like academic psychology, it would lose its distinctive character as an interpretative enterprise dealing with the symbolic relationships between substitute objects and the primordial (and lost) instinctual objects. On the other hand, the understanding of human motives alone is unable to account for Freudian concepts like cathexis, where the language of intention and meaning is replaced by energy metaphors. A distortion between literal and intended meaning evokes mechanisms like dreamwork, displacement and condensation. Jaspers' verdict that these mechanisms only promote 'as if' understanding is judged to be too harsh, because their reality is deducible from the manifestation of object relations, which reappear and can be examined in the transference relationship.

The weakness of Ricoeur's argument lies in his acceptance of Freud's dynamic mechanisms. Even if one accepted that these mechanisms correctly took over where empathic understanding was at an end, and that they were providing a plausible and coherent explanation of the observed phenomena, Jaspers' criticism of 'as if' understanding still holds. Psychodynamic mechanisms, as long as they are only based on the *failure to understand* a patient's behaviour by examining all overt clues, are circular and fanciful constructs. In order to become valid explanatory constructs they must be identifiable independently of the psychotherapeutic dialogue (Nagel, 1959).

Habermas, a contemporary representative of the Frankfurt School of Critical Theory, has developed a theory of knowledge in which psychoanalysis occupies a special place (Habermas, 1978). He acknowledges the difference in methods between causal sciences and hermeneutic disciplines, but gives it a novel meaning. Categories like space, time, substance, and causality are, according to Kant (1931), implied with the perception of any object, independently of its particular impression on our senses. In a similar fashion Habermas postulates constitutive interests in knowledge which underlie scientific activities. He recognises a technical interest to manipulate and predict the environment. Its method is causal analysis, and its field, the natural sciences. Similarly, the interest of interpersonal communication underlies the understanding of meaningful connections in the human sciences. Habermas criticises the extension of the technical interest into the human sciences: sociology and psychology, whenever they try to establish laws in order to predict human behaviour, introduce an unjustified sense of necessity. They tend to maintain the status quo, and can act as 'false consciousness' or ideology that prevents the emancipation of human beings or groups.

Habermas takes Freud's statement, "where there was Id, there shall be Ego", and defines the task of psychoanalysis as the shifting of the barrier between explaining and understanding by self-reflection. Seemingly unavoidable acts like compulsions or conversion symptoms are brought back into the realm of intentional, freely willed actions by the uncovering of their meaning. Habermas, indeed, suggests that the understandability of seemingly causal connections points towards the possibility of emancipation from their false power.

Habermas' theory relies on a cognitive account of psychoanalysis. It hinges on the questionable efficacy of insight in bringing about the emancipation from symptoms. Similarly, Habermas' strict insistence on a division between the methods for the natural and the human sciences is open to criticism: it implies a division of the world into a natural world subjected to causal necessity and human sphere where causal constraints on free will and agency can only be temporary (Keat, 1981). It would go beyond the limitations of this paper to give a discussion of determinacy and free will; suffice it to say that without the causal efficacy of human motives or decisions on their implied acts, free will could never express itself (Bhaskar, 1979).

Another author who examines the link between explaining and understanding in the human sciences is Bhaskar (1979). His realist theory of science rejects the Humean regularity model of causality. Regularities, according to Bhaskar, can only be observed in controlled experiments in closed systems. In particular, falsification as a criterion for the rejection of hypotheses cannot be used in open systems, since the constellation of frame-conditions only allows for the expression of causal mechanisms on special occasions. This observation leads Bhaskar to base his theory of science on the assumption that causal generative mechanisms are really existing in the world around us. Since falsification cannot be a criterion for rejecting their existence, they cannot necessarily be used for predicting events.

Bhaskar's theory of knowledge (Bhaskar, 1975) has been applied to psychoanalysis and psychiatry by Will (1980, 1983, 1984). Will's conclusion for the methodology of these disciplines is that the search for explanations with the help of generative mechanisms makes hermeneutic approaches unnecessary and undesirable. Bhaskar himself seems to suggest otherwise (Bhaskar, 1979). He argues that the natural sciences arrive at the knowledge of generative mechanisms through their direct observation or the observation of their effects, as in the case of electrical or gravitational fields. The human sciences need a hermeneutic approach for the *definition* of their generative mechanisms. The differentiation of, for instance, causally effective reasons from mere rationalisations, or lies, is possible by comparing the stated reason with its context; that is, the situational and personal circumstances. Naturally, the statement of a reason by itself might change our appreciation of somebody's personality – we encounter here, again, the hermeneutic circle. Bhaskar thus accepts understanding as a legitimate method for arriving at generative mechanisms in the human sciences. He fails to discuss, however, the logical relationship between hermeneutics and the testing of hypotheses.

A logical framework of the interaction between understanding and explaining

The strength of causal analysis is most obvious where predictions can be made, or where technical control of the environment can be achieved. The logical structure of explanations has been examined by Hempel & Oppenheim (1948); both Nagel (1959) and Popper (1969) have described criteria of adequacy for explanations in general, and for psychoanalysis in particular. Stockman (1983) has delineated the controversies between realist and Popperian approaches to causal explanations, and has suggested that these are at present not decidable (Stockman, 1985).

According to Jaspers (1963), understanding derives its validity from two sources. On the data side, *objectively observable behaviour* or artifacts like letters, drawings and the like can be analysed by several examiners. Their understanding of the data within their context can be compared. Jaspers calls the understanding of objective data interpretation, a choice of terminology which is compatible with everyday language.

Understanding, as long as it is based on objectively observable data, requires, in addition, the experience of plausibility in the observer. This experience of plausibility, or *evidency* in Jaspers' terminology, does not depend on concurrent empirical validation. It is plausible, for example, that someone who has been disappointed in relationships might consequently be suspicious about the motives of others. Where does this experience of plausibility or evidency come from? A necessary condition for the possibility of understanding is that human beings can empathise with each other. There are several levels on which this can be illustrated. As social beings humans use an array of inborn gestures and expressive utterances. For example, the ability to laugh and cry is retained in congenitally deaf and blind people, and these expressions are understandable independently from cultural background and personal experience. A shared physical and social environment facilitates understanding, but even foreign cultures can be understood by learning the rules of their language and their social institutions (Malinowski, 1922). Since the rules linking code signs to coded message are arbitrary (Saussure, 1974), the breaking of a code consists of an attempt to unravel internally the meanings of signs, relating them reciprocally to the whole message. Wherever human cultures have created systems of signs or symbols, the hermeneutic method will, therefore, be the appropriate method of analysis.

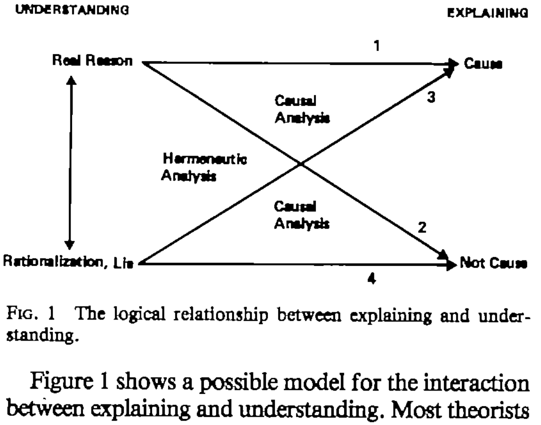

FIG. 1 The logical relationship between explaining and understanding.

Figure 1 shows a possible model for the interaction between explaining and understanding. Most theorists agree that causal explanations, before they become established, have to go through a stage of being tested as hypotheses. How hypotheses are arrived at in the first place often remains obscure. A common approach in psychopathology is to start with understandable connections, to make their components operational, and to test them in order to arrive at a correlation and, hopefully, a causal connection. It is understandable, for example, that a single mother without support and with many children to look after might easily slip into a depressive state after a serious loss, particularly if she has been sensitised to loss by a previous important bereavement, or if her mother, the traditional source of help, is not available (Young & Willmott, 1962; Brown & Harris, 1978). This understandable connection can be transformed into a hypothesis to be tested by empirical means (Fig. 1, arrow 1). It is essential to differentiate between the claim for truth of the understandable connection and that of the causal hypothesis. The former depends mainly on the psychological plausibility of depression developing from loss under certain circumstances. The latter hinges on the statistical persuasiveness of empirical results.

It is conceivable, in contrast, that an understandable connection, for instance between the loss of an organ and depression, is not borne out in causal analysis (Fig. 1, arrow 2). Although a causal relationship is not confirmed by the empirical data, a particular case can still be legitimately understood, if the connection is psychologically plausible. Although hysterectomy might not lead to an increased incidence of depression (Gath & Day, 1982), a case of depression after hysterectomy can still be understood on the basis of this connection.

Researchers are less likely to derive hypotheses from plainly disingenuous reasons (Fig. 1, arrows 3 and 4). A case could easily be construed, however, of a patient with occult carcinoma of the pancreas who claimed that he was depressed *because* he was terminally ill. Empirical testing of this psychologically clearly absurd hypothesis (which might have been classified as a nihilistic delusion by the clinical observer) could, however, lead to a medical and statistical confirmation (Fras *et al*, 1967; Fig. 1, arrow 3). Similar examples could theoretically be adduced for implausible rationalisations which prove to be empirically false if adopted as hypotheses (Fig. 1, arrow 4).

If the decision whether some factor is causally relevant or not lies with the empirical testing of hypotheses and is logically independent from the psychological plausibility of the postulated connection, it could be argued that the understanding of meaningful connections has nothing to contribute as a method of enquiry to the final outcome of scientific investigation (Will, 1983). At best it provides a heuristic tool for the formation of hypotheses. As long as no technological paradigms are being exploited, like imaging techniques or hormone assays, scientific investigation in psychiatry will naturally fall back on understandable connections for the production of causal hypotheses. In addition, the hermeneutic method has remained a part of diagnostic instruments in clinical research, like the PSE (Wing *et al*, 1984), which continually requires the examiner to decide whether symptoms or distress exceed the degree understandable from the patient's actual experiences and background. Here, and even more in clinical psychiatric practice, a wholly objective, operational system of diagnosis and assessment, although conceivable, has not yet been achieved.

Understandable connections can immunise the corresponding causal hypotheses against rejections (Kuhn, 1962). It is plausible, for instance, that middle-class psychiatrists find it easier to empathise with patients of their own class and, therefore, are less likely to classify their beliefs as delusional and to make a diagnosis of schizophrenia than with working-class patients. With seemingly supportive empirical results (Hollingshead & Redlich, 1958), a labelling theory of schizophrenia was quickly established. It needed much clinical awareness and research stamina to reject this theory (Goldberg & Morrison, 1963). Even the pseudo-plausibility of psychoanalytic 'as if' understanding was able to sustain, for example, the idea of schizophrenogenic mothers (Fromm-Reichmann, 1948) against mounting empirical evidence, an idea which was only disbanded after a considerable research effort (Leff, 1985).

Because of the central role understanding plays in psychiatry, it has to be recognised when it occurs. Its inner logic and limitations have to be appreciated, so that no unjustified claims are made (Storr, 1983). There is a moral obligation to use and improve understanding both in medicine and in human relations in general. The failure to do so is amply and painfully documented in the abuse of medicine and psychiatry under authoritarian regimes. Since both instrumental intelligence and empathy have survival value for a species, it can only be hoped that man's ability to find working explanations which lead to advanced technology is matched and tempered by his ability to empathise with others' way of thinking and feeling. In order to avoid pseudo-scientific claims, however, the fusion of biological and hermeneutic concepts, to use Gellner's terms, should be recognised and rejected.

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The Evaluation of a Hostel Ward

A Controlled Study using Modified Cost–Benefit Analysis

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A controlled modified cost–benefit evaluation of a hostel ward caring for new long-stay patients is described and results are presented for the first two years. In some respects the residents of the hostel ward had fewer psychotic impairments than those remaining on the wards of the district general hospital, mainly because the latter seem to continue to acquire such defects, while the former have remained relatively unchanged. The hostel ward residents also develop superior domestic skills, use more facilities in the community, and are more likely to be engaged in constructive activities than controls. These advantages were not purchased at a price, since the cost of providing this form of care for these patients has cost less than care provided by the district general hospital.

In spite of the current policy of shifting the emphasis of psychiatric care from hospital to the community, mental hospitals and psychiatric units of district general hospitals (DGHs) have continued to accumulate a number of long-stay patients who require continuous nursing care (Mann & Cree, 1976; Bewley *et al*, 1981; McCreadie *et al*, 1983; Levene *et al*, 1985). Even when long-stay patients have been discharged, there is evidence to suggest that few achieve full independence from hospital services (Howat & Kontny, 1982). Further, studies on community facilities such as hostels, small hotels and group homes have reported on the selection policies they may operate, which exclude the more severely disabled or disturbed patients (Hewett *et al*, 1975; Hewett, 1979; Ryan, 1979; Ryan & Wing, 1979).

Remaining in hospital for long periods may give rise to both clinical and social disadvantages for patients with chronic psychotic illness. The social milieu, particularly on an acute admission ward, may become too emotionally arousing and cause them to relapse into florid symptoms. The needs of these patients may be taken for granted by the staff when their attention is distracted by the demands of floridly disturbed and newly admitted patients. Further, it may progressively disable a person for life in the outside community when the social milieu and management practices of a ward prevent patients from being responsible for domestic tasks such as cleaning, cooking, and laundry, as well as outside activities such as shopping and gardening.

In view of these disadvantages, other forms of care for long-stay patients have been considered which may be more appropriate for meeting both their care and accommodation needs, and for helping them to become more equipped to cope with the demands of life outside in the community. Wykes (1982) and Wykes & Wing (1982) reported preliminary evidence which indicated that the care of these patients – excluding the elderly and those with organic brain syndromes – may be better in a hostel ward rather than in hospital; this study also compared revenue costs of a hostel ward with care in a mental hospital.

The purpose of this paper is to present details of a controlled modified cost–benefit evaluation (Glass & Goldberg, 1977; Goldberg & Jones, 1980), in which the care of long-stay patients in a hostel ward – called Douglas House – is compared with the care provided by the psychiatric unit of the DGH.

The Department of Psychiatry at the University Hospital of South Manchester was first opened in 1970. It is a psychiatric unit in a teaching DGH and has a full catchment area responsibility for approximately 230 000 people. The unit does not have access to any long-stay beds in a large mental hospital. Despite an active rehabilitation programme, use of local authority hostels and care by community psychiatric nurses, since 1970 we have accumulated a number of patients under the age of 65 years who have needed to remain in hospital for longer than six months. They are our most disabled patients in that they have resisted all attempts to resettle them in the community: their common characteristic is that they need intensive nursing care.

Douglas House is a ward of Withington Hospital, and its residents are legally in-patients who are in receipt of 24-h nursing care, albeit in a Victorian house just over a mile from the main hospital. It has been described in detail by Goldberg *et al* (1985). During the period of the evaluation, the number of patients living there has never been more than ten. We first carried out an overall comparison of non-financial effects ('soft' costs and benefits), and then considered what each service costs ('hard' costs).

Hypotheses

At the beginning of the evaluation, the following hypotheses were formulated about the non-financial effects of being cared for in Douglas House compared with the care being provided by the DGH unit (Goldberg, 1983):

(a) that there would be a reduction in problem behaviours and impairments displayed in the residents;
(b) that residents would have fewer episodes of acute psychosis than hospital patients;
(c) that residents would have significantly more socially adaptive behaviours;
(d) that residents would spend significantly more time than patients in the DGH doing socially useful activities;
(e) that residents would be more satisfied with their living conditions, and would not wish to return to the hospital.

Method

Patients

Before the start of the study, all patients in the Psychiatric Unit of Withington Hospital who had been in hospital for at least six months, were in the age range 16–65 years, were in need of 24-h nursing care, and were not thought to constitute a serious hazard to public safety were identified. Only 20 patients satisfied these criteria; of these, 18 patients were eligible and consented to be in the study while a further 2 refused to co-operate. Subjects were approximately matched in pairs in terms of their potential for rehabilitation based on duration of illness, hospital diagnosis, extent of psychological impairments and extent of remediable problem behaviours as measured at our baseline assessments.

It required considerable ingenuity to produce two groups of nine patients each of whom matched a corresponding subject in all these respects. Although no attempt was made to match the patients for age, sex, or research diagnosis, the two groups turned out to be matched in these respects as well. The index group to be transferred to Douglas House was chosen by the toss of a coin.

When places subsequently became available at Douglas House, further eligible patients were identified and introduced into the study in pairs. As we aimed to ensure that the two groups remained broadly comparable with one another, we examined the effects of the overall matching of the new pair being offered first one way round, then the other. It turned out that the overall matching was always preserved, so the allocation of each member of a pair was randomised. We are reporting results for the first 22 patients (11 pairs) to complete two years in the study.

The two groups

In the index group, the management of each resident is individually formulated and structured by a rehabilitation programme which aims to help the resident to improve and maintain his or her self-care, domestic, and community skills as well as to change specific problem behaviours. Nursing is organised on behavioural lines under supervision of the clinical psychologist, with a reward system of points which are exchanged for money on a weekly basis. Patients are encouraged to follow their own leisure and social pursuits and they invariably take part in group activities that they arrange for themselves with the staff, such as going to the cinema, theatre, pubs, or restaurants. Patients who had to be returned to the hospital are still considered to be within the index group, both for non-monetary and monetary effects of the analysis.

In the control group, patients have access to comprehensive facilities of the Occupational Therapy, Industrial Therapy, and social and recreational departments of the hospital and they are able to use community amenities as they wish. Patients who were discharged into the community are still considered within the control group.

Measures

Non-financial effects ('soft' costs and benefits)

Problem behaviours and impairments were measured by the Psychological Impairment Rating Scale (PIRS) (Jablensky *et al*, 1980) and the Remedial Problem Behaviours scale developed at the Maudsley hostel ward (Wykes, 1982). We were informed that ten factor scores can be computed from the PIRS which account for over 90% of the variance (Jablensky, personal communication). Of these, we predicted that our treatment programmes might have beneficial effects on four: psychomotor inhibition, non-assertiveness, parathymia, and indifference. We also examined the 'overall impression score' and the total item score.

The clinical state of the patients was measured by the Present State Examination – PSE (Wing *et al*, 1974), the Mini-Mental State – MMS (Folstein *et al*, 1975) and the Krawiecka scales (Krawiecka *et al*, 1977).

Socially adaptive behaviours and socially useful behaviours were measured by use of a social facilities checklist (Wykes, 1982) and a television substudy. Each patient was interviewed by a social worker in a television studio for 15 min on a topic concerned with how the patient spent his or her leisure time. During this they were offered coffee and biscuits and the studio was so arranged that the patient had to pour out the coffee and pass this and biscuits to the interviewer. By using a split-screen technique, details of social and eating behaviours were recorded. These were later rated by raters who were blind to which group the patients were in and again by two speech therapists who made ratings on clarity of speech and communication skills.

Use of time was assessed with a time budget. This consisted of standardised observations of what the patients in each group were doing with their time between 0800 h and 1030 h on three consecutive weekdays and a weekend. Trained observers who worked in shifts tracked down each subject every 30 min and as discreetly as possible noted what the patient was doing in terms of a predetermined set of behavioural categories. Reliability in their observations was established so that the lowest percentage agreement between the observers was 81%.

Satisfaction with living conditions was assessed with patient's attitude scales (Wykes, 1982). These scales deal with a patient's satisfaction with eight aspects of his (or her) care: his bedroom, meals, nursing staff, other residents or patients, the regime, his occupation, attitude to discharge, and medical information.

Procedure. Apart from the time budgets, all these assessments were carried out before patients were allocated to their experimental groups. Thereafter they were repeated every six months, with the exception of the PSE and MMS, which were repeated annually. Ratings were made in the community for those patients who were discharged from either group.

The time budgets were carried out at the end of the first and second year. The first time budget was carried out with all patients in their respective group. However, on the second occasion, the original pairing could not be maintained, since patients had been discharged from the control group, and others had returned to hospital from Douglas House. Accordingly, only those patients at Douglas House and the control group still in hospital were compared.

Financial effects

Costs have been analysed and are presented in two ways: first as average daily costs per patient across the two settings; second as the average annual costs of providing care for a patient regardless of where he was during the period of the evaluation. The first method includes only National Health Service costs, while the second includes local-authority costs as well, thus representing the true costs to society of caring for these patients.

In calculating these costs, several assumptions were made:

(a) Capital costs were included.

(b) The total revenue costs of the hostel ward did not include the costs specifically concerned with its evaluation.

(c) Patients in both index and control groups were treated as homogeneous.

(d) It was assumed that Douglas House would run at full capacity; this was thought reasonable both clinically and managerially.

Four categories of costs associated with each form of care were considered.

Capital costs. These refer to the costs of replacing and upgrading the hostel ward compared with the equivalent costs of replacing the total psychiatric unit. The latter is physically separate from the other DGH buildings, making computation of capital costs straightforward. It is assumed that the capital costs would be incurred at some time in the present, although the life of the buildings and hence the benefits from using them would be over 30 years and 50 years respectively. The capital costs were therefore converted to an annual equivalent, using a discount rate of 5%.

Revenue costs. These refer to costs of direct care provided by nursing and non-nursing staff; investigation and treatment costs; and general service costs. All medical records, prescription charts, and nursing records were examined and careful records were made of use of hospital facilities, medical problems, investigations, medication, and evidence of psychiatric relapse throughout the two years. A careful record was also kept of all visits by staff, to measure the service utilisation of each group.

Calculation of all the nursing costs of Douglas House were based on the actual occupancy and actual nursing levels over the two-year period of the evaluation, and repeated assuming full occupancy and a full establishment level. The nursing costs for the hospital were calculated from a 'professional time exercise' mounted over a two-week period and covering 24 h of each day, during which all professional staff in the DGH psychiatric unit systematically recorded, on a standardised form, details of time spent with a variety of patients. This provided information on the amount of time all professional staff were devoting to the control patients in the DGH unit, and takes account of time spent in face-to-face contact with individual patients, time spent discussing the patient on ward rounds, and administrative time. This information was then costed and based on staff salaries. The staff were of all ranks and training and there was 100% co-operation.

General service costs were derived from specialty costing information collected by the health authority. The nature of their accounting system made it possible for the health economist (KL) to have details of the general service costs that were specific to the individual wards of the psychiatric unit in the DGH as well as to Douglas House, which included costs for rates, electricity, telephone, maintenance, food, and other consumables.

Local-authority and Home Office costs. During the period of the evaluation, two patients from the control group lived in local-authority hostels and night shelters after their discharge from the DGH unit, and a third patient spent time in prison. Costs calculated for providing these services were based on figures available for 1983, as published by the Chartered Institute for Public Finance Accountants.

Other social costs. These costs include those falling on the family, as well as any offset of costs due to employment of patients, but exclude transfer payments such as Social Security benefits, which are ignored in the analysis. Although we made efforts to examine the financial burden on the families concerned, it appeared that for these particular patients this was negligible, and none of them gained employment. Such small costs as existed were similar in the two groups, so they were ignored in the present analysis.

Results

Three patients had to be returned to the main hospital from Douglas House. One of these could no longer be nursed in a domestic setting because of progressive ataxia which put him at serious risk of personal injury when climbing stairs to his bedroom and when doing things in the kitchen. Two other residents demonstrated unremitting behavioural problems when initially confronted with the demands of ordinary domestic living and graded behavioural programmes. Both showed acting-out behaviour patterns, with firesetting, exhibitionism, and aggressive outbursts, and it was noteworthy that their maladaptive behaviours quickly remitted once they were back in hospital again.

As it is important to assess the new service irrespective of whether patients were able to benefit from it, the analyses based on the complete group of 11 patients originally assigned to the index group with their 11 controls are reported. However, it is also of interest to study the effects of the new service on those whom we were able to manage there, so we have also examined data based on the 8 patients who did not have to return, together with their 8 matched controls.

There were six men and five women in each group, with an average age of 42 years (s.d. = 14.0). On average the duration of their illness since their first admission to hospital was 12 years, the accumulated time they had spent in hospital was 5 years, and the period of their current admission was 3.5 years. The commonest diagnosis in each group was schizophrenia (14 patients), while the remainder were suffering from epilepsy, organic mental states and manic–depressive illness.

PSE-based Catego classes for these patients were Schizophrenic Psychoses Class S+, index group 8, controls 4; Uncertain Psychotic/Retarded Depression O?/R, index group 2, controls 2; Manic and Mixed Affective Psychosis M+, index group 0, controls 3; Other Psychoses O+, index group 0, controls 2; Obsessional Neurosis B+, index group 1, controls 0.

Non-financial effects

Problem behaviours and impairments. The results for the PIRS scales are shown in Table I for the complete set of 22 patients, using ANOVA with repeated measures. It can be seen that the hostel ward patients are significantly less 'non-assertive', and significantly less 'indifferent' than those remaining on the wards, although other variables did not reach statistical significance. If the analysis is confined to the reduced group of 16 patients, then the 'overall impression' based on the PIRS becomes significant, as shown in Fig. 1. There is a sharp decrease in psychomotor inhibition for the index group immediately on entering the experiment, but thereafter the two groups slowly deteriorated at an approximately equal rate. Other significant differences in favour of the index group were non-assertiveness and indifference.

The 'remediable problem behaviours' scales showed no consistent changes with time nor any significant differences between the groups for 'publicly undesirable behaviours', social skills, hygiene and self-care. For domestic skills there was a significant difference ($F = 7.01$; $P < 0.02$) favouring the index group, which showed a large improvement in the

TABLE I

The Psychological Impairments Rating Scale – PIRS (analyses of variance with repeated measures; between group interaction with time)

	Complete group (11 pairs)		*Reduced group (8 pairs)*	
Measure	*F value (d.f. 4, 76)*	*P*	*F value (d.f. 4, 53)*	*P*
Factor scores:				
Psychomotor inhibition	2.35	0.06	1.60	0.19
Non-assertiveness	3.12	0.02	2.99	0.03
Parathymia	1.06	0.38	0.95	0.44
Indifference	3.01	0.02	2.73	0.04
Overall impression (total score)	2.03	0.10	2.60	0.05
Total items score	1.55	0.20	2.34	0.07

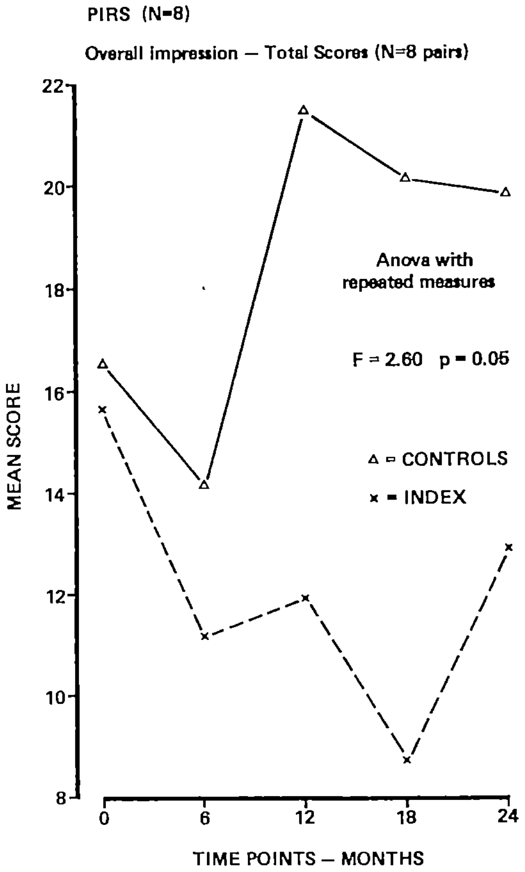

FIG. 1 PIRS-overall impression score: comparison of mean scores of both groups with time.

first six months but no subsequent improvement, whereas the control group showed a slow but steady improvement with time. In each group there was a significant improvement with time in community skills ($F=3.36$; $P<0.02$), but no inter-group differences.

Episodes of acute psychosis. There were no significant differences between index and control groups for any rating measured on the Krawiecka scales, including affective symptoms, and positive symptoms; nor were differences demonstrated between the groups at any time point with the PSE.

For the reduced group it appeared that the control group accumulated more negative symptoms on the Krawiecka scales, but this fell short of statistical significance.

Socially adaptive behaviour. For those remaining in Douglas House, use of social facilities increased for the index group as soon as they arrived, and was sustained thereafter. The differences between the groups was significant and was largely accounted for by an increased use of the public house, cinema, and post office ($F=2.6$; $P<0.05$). There were no significant differences in social deficits or communication skills demonstrated in the television study.

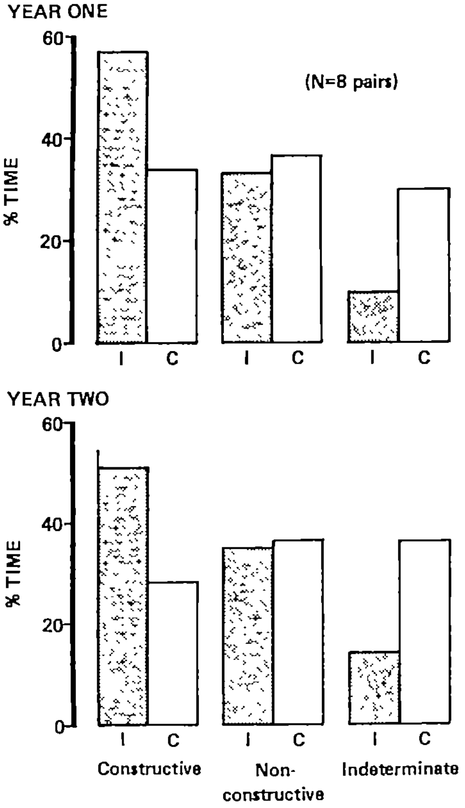

FIG. 2 Time budgets at one year and two years (I = index; C = controls).

Time spent on socially useful behaviours. As some specific behaviours had such low frequencies, they were collapsed into three categories: 'constructive' (e.g. eating, self-care, working, conversation); 'non-constructive' (e.g. doing nothing, lying in bed); and 'indeterminate' (e.g. on leave, whereabouts unknown). In both years when time budgets were carried out, the Douglas House patients were spending significantly more time doing socially constructive activities than the controls. Both groups were similar in terms of the proportion of their time spent doing non-constructive activities (Fig. 2). We thought it unlikely that those unavailable for observation and thus classified as 'indeterminate' were in fact engaged in constructive activities.

Satisfaction. We did not think it meaningful to make direct comparisons of satisfaction between the two groups, since several members of the control group were either in the community or in other forms of institutional care; we therefore confined our attention to the index group. The two residents who were transferred back to the DGH were clearly dissatisfied with Douglas House and the demands of the rehabilitation programme. However, repeated measures of satisfaction assessed by the attitude scale consistently showed that the remaining residents were satisfied with life there. At two years, all said they liked the food, liked the other residents and considered that they had enough to do. The majority (7/8) said they had enough privacy, liked their room, could talk to the staff and found them friendly, felt that they had enough freedom and that any complaints would be dealt with, had been given enough information about their illness, liked the work around the house, felt they had enough to do during the evenings, and did not want to change anything. Five residents had thought of leaving, four said they had plans to do so, and only three wanted to stay indefinitely. When asked to compare life at Douglas House with life in hospital, only one slightly preferred hospital, one had no preference, and the rest preferred Douglas House.

Financial effects

It should be noted that certain aspects of care which provide advantages that cannot be expressed in financial terms – such as the advantages of not needing to be in hospital – also make an important contribution to the 'hard' costs of providing the service. They are therefore presented here rather than in the section on non-financial effects.

Average costs per patient-day for the National Health Service. The costs for providing each service are summarised in Table II. The capital costs for Douglas House were less than those for the psychiatric unit of the DGH, but these costs formed only a small proportion of total costs – 10% and 15% respectively. Nursing costs represented 31% of the hospital costs, but rose to 60% of the costs at Douglas House, due to the smaller number of patients being nursed and the intensity of the nursing. The costs of non-nursing care, and investigations and treatment costs were low both for Douglas House and the DGH; these costs also represented a small proportion of total costs – 9% and 12% respectively. The largest difference between the two groups was accounted for by the costs for general

TABLE II
Summary of relative costs to the National Health Service: Average costs, £/patient-day

	Index group Douglas House	*Control group DGH wards*
Capital costs: £/patient-day		
Actual occupancy	2.96	6.59
(Full occupancy)	(2.67)	
Revenue costs: £/patient-day		
Direct care costs – nursing:		
Actual staffing		
Actual occupancy	17.58	13.12
(Full occupancy)	(15.51)	
Full establishment		
(Actual occupancy)	(22.05)	(14.76)
(Full occupancy)	(19.46)	
Direct care costs – non-nursing:		
Medical costs	0.58	2.80
Non-medical costs	1.06	0.72
Treatment and investigation costs:		
Investigations	0.04	0.08
Drugs	0.83	0.91
General services (hotel costs):		
Actual occupancy	6.33	18.67
(Full occupancy)	(5.59)	
Total NHS costs: £/patient-day	29.38	42.89
Range	26.23–33.92	42.89–44.53

services, such as heating, lighting, laundry, cleaning, administration, maintenance etc. For the DGH unit, these represented 44% of the total costs, as opposed to only 22% for Douglas House. This reflects the fact that patients at Douglas House perform many of these tasks themselves.

Average annual costs of providing care for the two groups. It can be seen from Table III that residents at Douglas House continued to need the services of the hospital. Some needed short admissions during episodes of florid psychosis associated with disturbed behaviour that could not be managed at Douglas House. One needed surgical care for a fractured hip and another required close medical supervision for a suspected heart attack. The control group spend far more time outside hospital than the index group, but of this time out of hospital, 48% was spent in other institutional settings – local-authority hostel, night shelter, or prison. None of the index group were discharged to another institution. Once more, time spent in night shelters and prison must be regarded as having non-financial disadvantages as well as the effects shown in the table.

TABLE III
Comparison of service utilisation by the two groups

	Index group (n=11)	*Control group (n=11)*
Day hospital: attendances	0	573
Industrial therapy: attendances	217	383
Occupational therapy: hours	22	418
Speech therapy: hours	145	0
Medical time: hours/week each group		
face-to-face contact	1.0	6.7
indirect contact	3.5	12.7
Number of nights:		
in Douglas House	5743	—[1]
on wards of DGH	1286	5299
on leave	479	270
in local authority hostel or night shelter	0	783
in prison	0	90
at home, with relatives or in lodgings	533	1599
(Total number of nights)	(7829)	(7829)

1. Not applicable.

The medical staff spent less time in direct or indirect contact with patients in the index group than with the controls. Despite this, the index group were more satisfied than when they were on the ward of the hospital with information given to them about their illness and about their medication as assessed by the attitudes scale.

Some residents from Douglas House had to return to hospital; patients in both groups required medical and surgical care or psychiatric day-care; and certain discharged patients from the control group required local-authority or Home Office care. The costs of providing care to the two groups regardless of where the patients were during the two years of the evaluation showed that care in Douglas House was slightly less expensive: £10 284/patient-year, against £11 504/patient-year for the controls. However, the difference becomes substantial if the analysis is confined to the reduced group of eight pairs of patients: the cost of care in Douglas House now falls to £9240/patient-year, while costs for the controls actually rise to

£12 449/patient-year. This is because the cost of providing care for those returned to the DGH unit is added to the cost of Douglas House for the complete group of 22 patients: for this reason it is of interest to examine the costs for both the full and the reduced group.

Discussion

There are two important reasons why the results of this study should be treated with some caution.

First, the research team had to fulfil two different roles as evaluators and service-providers. They were not blind to which service each group was receiving and it was not possible for them to be independent of the new service being evaluated. Further, it was possible that as the residents became more familiar with them, this may have influenced their behaviour during their assessment interviews. However, the psychiatrists also had clinical responsibilities for the care of some of the control group, and in the evaluation they strove to be scrupulously fair and objective. In anticipation of this problem, it was expected that the major reliance on standardised assessments would minimise any possible bias effects and it was thought that most of the economic data was less vulnerable to these influences.

Second, it is not possible to generalise from these results because of the small number of patients that have been considered at this stage and because the data so far only refer to a two-year period. However, we would observe that we have included everyone produced by a catchment area of a quarter of a million in just over ten years: thus if a larger study is to be done, it will have to involve more than one district health authority. If future studies use similar measuring instruments, it should be possible to pool data between studies. It is important that such further evaluations are carried out rather than firm policies being established on the evidence of a single study.

However, the preliminary results presented support the observations reported on the Maudsley hostel ward (Wykes, 1982), that patients with chronic psychiatric disabilities gain certain advantages from this form of care. With reference to the original hypotheses considered in this evaluation, the results indicate that in some respects residents of Douglas House had fewer psychotic impairments than patients remaining in hospital. This appears to be because the control group seemed to continue to acquire such defects, while the patients at Douglas House remained relatively unchanged. The results also indicate that the residents at Douglas House acquired better domestic skills, used more facilities in the community, were more likely to be engaged in constructive activities and preferred being there rather than in hospital. In several important respects both the control and index patients did equally well, but these results suggest that Douglas House has a function in preventing or at least slowing the development of psychotic impairments, and that it provides a more suitable environment for rehabilitation than an acute admission ward of a hospital.

Although Douglas House proved unsuitable for some patients, the residents who were able to manage in this setting appeared to fall into two main groups. The first comprised patients who progressively improved in psychosocial functioning and who were able to move on to less dependent or independent settings. The second group comprised patients who remain clinically impaired and socially disabled, who did not progress much after the first six months, and who still needed continuous nursing care. In this group, when a new behaviour was no longer rewarded, it tended to disappear. For them the hostel ward was either preventing or perhaps slowing down the rate of further deterioration and appeared to be providing them with a better quality of life.

The results of the economic evaluation indicate that these advantages do not have to be purchased at a price when the costs of this form of care are compared with the costs of caring for these patients in a DGH unit. Even when the entire group of patients are considered, irrespective of where they were during the period of the evaluation, the costs for the index group were less than those for the controls. It would be expected that nursing costs are going to be high when relatively small numbers of patients are being nursed together. However, these costs at Douglas House were offset by the savings made on general service costs, partly because no domestic and catering staff were employed and because the residents were expected to contribute to the housekeeping and catering tasks as part of their daily lives.

The inclusion of capital costs, albeit approximate, suggests that the capital consequences of a policy of hostel ward accommodation are cheaper than those of accommodation in hospital units. It is unlikely that many hostel wards can be built using resources released by closing wards on DGH units, since in many areas these are already providing services from very small numbers of beds, and cannot be further reduced without causing serious adverse effects on existing services. However, moving patients from an existing DGH unit to a hostel ward should make it possible to use the released resources more effectively by offering additional places to patients who would benefit from short-term hospital treatment.

Districts at present served by mental hospitals may wish to use resources produced by ward closures to fund places in hostel wards. The present results cannot be applied to this situation, since the average costs of keeping patients in chronic wards in mental hospitals are very much lower than the costs for equivalent patients in the DGH, and some long-stay patients in mental hospitals develop disabling psychotic behaviour patterns that are less often seen among those nursed in DGH units, and these patients would require much nursing time if cared for in a hostel ward.

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Psychogeriatrics 1974 to 1984

Expanding Problems and Fixed Resources

ALEX B. CHRISTIE and ERIC R. M. WOOD

The impact of a 16% increase between censuses in the population aged 65 and over, on a psychogeriatric department whose bed complement remained static between 1974 and 1984, has been studied. Demand for service, as measured by new referrals, rose by 150%, while admissions fell by 14%. The increase in new referrals was uniform across the diagnostic spectrum, but the fall in admissions was not. Functional admissions fell in all age-groups except that of women of 85 and over, while organic-case admissions other than for cases of dementia virtually collapsed. Overall admissions for dementia rose to the predicted level, but the distribution of the increase was irregular and unexpected. Admissions of males and females aged 65–74 and females of 85 and over fell relatively and absolutely, while those of women aged 75–84 and men of 85 and over were little changed. Only admissions of men aged 75–84 alone increased in real terms.

Much has been written about the need to develop services for the elderly with mental illness. The *British Medical Journal* (1978) drew attention to the difficulties posed by dementia in an ageing population in the UK. Kramer (1980) presented evidence of the massive problems posed by senile dementia of the Alzheimer type (SDAT) worldwide in the last quarter of the twentieth century. Increases in prevalence of 56% in more developed countries and 123% in the Third World are predicted by the year 2000. Various planning documents (e.g. Scottish Health Service Planning Council, 1980), in drawing attention to the need to divert resources into the field of dementia, carry the implicit threat of chaos spreading throughout medical services, with widespread blocking of beds by demented elderly patients for whom no alternative provision exists.

The likely impact of functional illness among the elderly is less easy to predict. Estimates of prevalence vary widely (Hasegawa, 1985), and to complicate matters further, in many centres no distinction is made between elderly and younger patients in the management of functional illness, and consequently resource allocation to the elderly is difficult to define. Nonetheless the studies of Post (1972), Murphy (1983), and Murphy & Grundy (1984) make it clear that depression, the most common functional problem among the elderly, can be very intractable – a fact which, when linked to the increased at-risk population, leads us to predict an increased hospital workload, both in-patient and out-patient, from functional illness.

The present study looks at the period 1974 to 1984, and examines the issues by analysing the experience of one hospital whose psychogeriatric-bed complement did not increase in that time. During the period in question, levels of geriatric beds within the catchment area rose from 188 to 321 beds (+71%), while levels of places in old people's homes remained constant. By the end of 1984, the private sector had not made any impact locally upon the problems of the elderly.

Local factors

The catchment area served by Crichton Royal Hospital contains 143 000 people scattered over some 2500 square miles. Psychiatric resources are concentrated in Dumfries (population 30 000), the only large centre within the catchment area. The elderly are mainly to be found in scattered small communities up to 90 miles from the source of psychogeriatric services.

Despite distance problems, every effort was made to provide a prompt and effective service. The patients were seen initially by a consultant in one of several settings: 61% were seen at home, 25% were seen in the wards of other hospitals, 8% in old people's homes, and the remaining 6% in a variety of settings. Follow-up investigations were undertaken by medical staff, community nurses, or social workers. Emphasis was placed on dealing with the situation locally, despite problems of communication and co-ordination of service over a wide area. Early referral was encouraged, and for both medical and social reasons, admission was a policy of last resort, particularly for those living many miles from the hospital.

In both 1974 and 1984, the bed complement stood at 191 short and long-stay to deal with all psychiatric problems in those over 64. During this time, the proportion of the elderly within the population rose from under 14% to 16%.

Method

All patients admitted to the psychogeriatric wards of Crichton Royal Hospital in the calendar years 1974 and 1984 were studied with respect to age, diagnosis, and outcome at the year's end. The department's responsibility encompassed the full range of psychiatric problems in the population aged 65 and over.

We hope in due course to include details of the final outcome for this group of patients in a wider study and, therefore, emphasise only the features which reflect hospital workload in the shorter term.

In order to present a comprehensive picture of work undertaken, we looked also at new referrals from all sources – clinics, domiciliary visits, and general-hospital departments – employing the same parameters as for admissions. Patients identified as new referrals in a given year were not necessarily among those admitted in that or any other year; for example an established day-patient suffering from SDAT might be a 1982 new referral and a 1984 admission; or a patient with an acute functional psychosis might be admitted as an emergency without prior out-patient contact.

We have chosen to make only passing reference to the work of our day hospital at this time. In practice, it looks after mainly chronic cases and, therefore, serves a distinct and separate function. Furthermore, being available to only half the at-risk population, it does not reflect the work of the department.

TABLE I
Results – by diagnosis

	1974		*1984*	
Diagnosis	*Referrals*	*Admissions*	*Referrals*	*Admissions*
Endogenous depression	38	35	44	30
Hypomania	1	4	3	5
Depressive neurosis	2	6	29	3
Late paraphrenia	2	9	16	4
Other functional	20	12	43	4
Total functional	63	66	135	46
SDAT	43	46	113	68
Multi-infarct dementia	11	28	35	17
Total dementia	54	72	148	85
Toxic confusional states	10	20	19	7
Other organic	3	2	8	2
Total organic	67	94	175	94
Grand total	130	160	310	140

Results

Table I gives details of referrals and admissions. Referrals rose by a factor of 2.5, from 130 in 1974 to 310 in 1984. The number of persons admitted fell slightly, from 162 in 1974 to 140 in 1984. Readmissions during the limited follow-up period were few and played no significant part in the overall picture.

In order to make our results more manageable but at the same time more detailed, we have combined all functional cases into a single group, and formed a second compact group from the dementia – SDAT and multi-infarct dementia (MID). In Table II, the results are separated by age and sex.

Organic cases excluded in this revised classification were few, and more importantly did not contribute to long-stay placements. As a result they had only a short-lived effect on the service overall.

Table II also includes information on the demographic changes arising within the at-risk population. The familiar pattern of greatest increase in the population aged 75 and over is repeated.

Male functional referrals doubled from 19 to 38 between 1974 and 1984; admissions halved from 23 to 12. Changes in a similar direction were even more marked among female functional cases. Whereas in 1974 referrals and admissions both stood at 43, by 1984 referrals had risen to 97 and admissions had dropped to 34.

The picture with dementia groups was quite different. Male referrals rose from 19 to 37, while admissions rose from 25 to 35. There was a proportionately greater rise in female referrals, from 35 to 81, but only a slight increase in admissions, from 49 to 51. A point of interest is that the mean scores of the 1974 and 1984 dementia groups on the Crichton Royal Behavioural Rating Scale were remarkably constant, at 27.27 (s.d. 6.57) in 1974 and 26.87 (s.d. 6.46) in 1984.

The upward trend in referrals was uniform across the various age, sex, and diagnostic groups, with the exception of males aged 85 and over with a functional diagnosis. This was a tiny at-risk group of 300 and, therefore, the absence of referrals over 12 months was unlikely to represent a significant variation in the otherwise uniform picture.

The picture with functional admissions was one of decline, again with one exception. Females of 85 and over showed a rise from 3 to 5 admissions, and while clearly this is not statistically significant, it calls for comment in our discussion.

Among demented patients, our findings were more variable. In 1984, there were rather more admissions of males aged 75–84, a slight increase in admissions of males aged 85 and over and a perceptible fall in those of males aged 65–75. Among female age subgroups, on the other hand, there were falls in admissions in two, the exception being the rise among those aged 75–84.

Demographic changes as outlined in Table II account for only a small proportion of the increased number of referrals, and their impact on admissions is also limited. They serve to reinforce the downward trend in admission of males and females aged 65–74, irrespective of diagnosis. The group of females aged 75–84 with a diagnosis of dementia recorded an increase in admissions commensurate with the percentage

TABLE II
Results (revised classification) by age and sex

Sex	*Age*	*Functional cases*				*Dementia cases*				*Demographic change in at-risk population*
		Referrals		*Admissions*		*Referrals*		*Admissions*		
		1974	*1984*	*1974*	*1984*	*1974*	*1984*	*1974*	*1984*	
Male	65–74	8	24	14	10	5	15	12	9	+14%
	75–84	8	13	9	2	11	29	7	18	+36%
	85+	3	1	2	0	3	9	6	8	0
	Total	19	38	25	12	19	53	25	35	+18%
Female	65–74	26	54	26	18	8	14	11	8	+7%
	75–84	14	33	12	11	22	58	25	33	+25%
	85+	4	10	3	5	5	23	13	9	+26%
	Total	44	97	41	34	35	95	49	50	+14%
	Grand total	63	135	66	46	54	148	74	85	+16%

increase in the at-risk population, while the group of males in the same age and diagnostic category recorded a true increase. By contrast, demographic change only served to emphasise the fall in functional admissions, male and female, in this age group. The increase in functional admissions for those 85 and over exceeded the demographic increase, but with such tiny numbers it would be rash to dwell on such a change.

TABLE III
Observed v. predicted age/sex distribution of 1984 admissions (prediction based on the age/sex distribution of the 1974 dementia and functional admissions adjusted for the overall increase in the over-65 population, and for the increase in population within each age/sex band in Dumfries and Galloway)

Age	*1984 dementia admissions*			
	Male		*Female*	
	Observed	*Predicted*	*Observed*	*Predicted*
65–74	9	13	9	12
75–84	18	8	33	30
85+	8	7	9	16
Total	35	28	51	58

$n = 86$
Comparing observed with predicted admission, $\chi^2 = 18.10$, d.f. $= 5$, $P < 0.01$

Age	*1984 functional admissions*			
	Male		*Female*	
	Observed	*Predicted*	*Observed*	*Predicted*
65–74	10	9	18	17
75–84	2	8	11	10
85+	0	0	5	2
Total	12	17	34	29

$n = 46$
Comparing observed with predicted admissions, and combining 75–84 with 85+ age groups for both sexes, $\chi^2 = 6.00$, d.f. $= 3$, $P \simeq 0.20$.

During the decade in question, important developments occurred in the field of community care. The work of community psychiatric nurses (CPNs) was extended to cover the whole catchment area. This has resulted in an increase in the average monthly return of CPN home visits, from 250 to 450 (+90%). Because of the inadequacy of our records, a comparable figure for medical staff 'return out-patient contacts' is not available. However, the extent to which medical-staff activity outside the wards has expanded may be gauged from the following figures. In the year April 1977 to March 1978, 187 contacts were made; in the calendar year 1984 the figure was 443. There is, therefore, substantial evidence of greatly increased extra-mural activity on the part of both nursing and medical staff while the principal changes described in this paper were unfolding.

Table III compares the actual 1984 admissions with predicted figures based on 1974 results and demographic change. In only the dementia groups are the changes statistically significant ($\chi^2 = 18.10$, d.f. 5, $P < 0.01$).

Discussion

We make no claim that the present study should be seen as representative of other hospitals' experience. Figures of the Common Services Agency of the Scottish Home and Health Department show that the admission rate for the elderly to psychiatric care in Scotland rose by approximately 50% between 1974 and 1984, while Smith *et al* (1986) report a doubling of admissions for dementia in England between 1973 and 1983. What our findings may represent is the experience of a long-established service in the aftermath of its initial development being confronted with a new set of problems in the second phase of its evolution.

After 25 years in operation, the service may be perceived by the community as rather more than the solution of last resort when they are confronted with intractable problems among the confused elderly. Such a proposition is supported by the increased referral rate, which far exceeds the increase in population and, more significantly, is not confined to the demented population. Such a view of this situation implies an expansion in the role of the psychogeriatric service without commensurate increase in resources. In practice, we did not set out to treat any given clinical problem preferentially, being content to deal on a day-to-day basis with matters of greatest urgency. A retrospective view of the situation suggests that dementia has asserted itself as the priority.

Against this background, two matters of fundamental importance call for consideration: firstly there is the fall in admissions and its causation, and secondly the probable outcome for cases who, by 1974 criteria, would have been admitted in 1984 but who did not gain access to the hospital.

Fall in admissions

Possibly the most important of our findings is that we admitted in 1984 the predicted number of patients with cases of dementia, although the sex distribution of this group was unexpected. Given that the number of available beds was unchanged, and that we have evidence from previous studies at Crichton and elsewhere (Blessed & Wilson, 1982; Christie, 1982; Christie & Train, 1984) that the dementing population is tending to survive longer in hospital, it follows that the predicted figure has been achieved at the expense of other patient groups, and to a lesser extent those dementia subgroups whose admission rates have fallen – males and females aged 65–74 and females 85 and over.

Table I illustrates the sources of loss. Among the cases with functional disorders, only hypomania has recorded an increase in the number of admissions, and here the impact is marginal. The group with endogenous depression has fallen slightly. The fall in depressive neuroses is considerable, even from a modest 1974 total. The small group with late paraphrenia has experienced a 50% reduction in admissions. The greatest drop of all has come in the 'other functional' group, largely made up of neurotic disorders flavoured with social problems, which has dropped by more than half. The conclusion to be drawn is that only the more severe illnesses have gained admission with any consistency, but not to a level commensurate with the population increase between 1974 and 1984.

On the organic side, SDAT admissions have increased selectively, while MID admissions have fallen, yielding overall the predicted number of admissions for dementia. Toxic confusional states have fallen back; the miscellaneous 'other organic' group remains small and unchanged. The explanation of the fall in the MID and toxic confusional states (TCS) groups almost certainly lies in the expanded geriatric provision locally. In his London study, Copeland (1981) showed that dementias based on vascular disease were more likely to find their way into medical as distinct from psychiatric care. Increased geriatric provision is likely to have enhanced this tendency. Cases of TCS may have gravitated towards available geriatric places in a similar fashion.

Cases not admitted

In considering the issue of cases not admitted, it is important to recognise that admission policy in 1974 may have been unduly liberal and, as a result, our projection overestimates the 1984 shortfall. We hold this to be unlikely in view of the 'catching up' in admission rates occurring elsewhere between 1974 and 1984. More significantly, we suspect, has been the reluctance of medical staff to send intellectually competent but depressed elderly people into a ward setting where constant contact with demented patients might be considered anti-therapeutic. It is also possible that the media have projected such an exaggerated view of the contemporary mental hospital as a repository for demented old people, that functional cases and their families have tolerated greater misery and distress in order to avoid such a gloomy prospect. This view is to some degree supported by the fact that the only functional group to exhibit an increase in its admission rate is that of females of 85 and over. These people are less likely than their younger fellows to have the home support to sustain them through the crisis, and may also be the most severe and intractable cases, for whom no possible alternative to admission exists.

We inevitably feel concern about the issue of functional illness among the elderly, as represented by our findings. We must either conclude that service to this group of patients has become poorer, or that the combination of family doctor, community nurse, and psychiatrist working with the patient in the community is equally or more effective in providing satisfactory treatment. It would be very comforting to believe that the latter is so, but in the absence of any positive supporting evidence we must accept the previously quoted work of Post (1972) and Murphy (1983) and suggest that a poorer service is the correct explanation.

In the dementia group, we note that the under-75 male and female admission rates have fallen, along with those of the 85-and-over females.

As in the case of functional illness, we have a choice of explanations. Of all our age-groups, the 65–74 is probably the one most likely to receive family support, and to have a spouse willing and able to contribute to management and be receptive to the concept of community care. Taken together, these factors may serve to delay or avert admission, and thereby account for our findings.

Such explanations are unlikely to apply to females of 85 and over. We know the at-risk population is rising, and that the prevalence of SDAT is at its highest in this group (Kay *et al*, 1964), and yet admissions have fallen. Recent pathological studies by Rosser *et al* (1984) point to the presence of less severe and less widely distributed lesions among the very elderly, compared with their younger counterparts. The clinical application of these findings might imply that in the older age-group, the illness is perhaps less dramatic and less frequently characterised by major behavioural problems which channel the patient towards psychiatric services. If this putative clinical analogy is valid, then our very elderly female patients may well gravitate towards the geriatric service more readily than their younger counterparts, emphasis being more readily placed on physical rather than psychiatric aspects of their caring needs.

Conclusions

We acknowledge the possibility that one-year study periods are too brief to enable final judgements to be reached. They have, however, the advantage of being manageable units of time, and for that reason allow us to attach some importance to our findings.

Our principal finding is that, contrary to expectation, functional illness gives way to dementia under the constraints of limited resources. Secondly, age has an important bearing on management of dementia. Patients in the middle band of our age-range are more likely to be admitted than their younger or older counterparts, particularly if the latter are female.

The fate of those whose admission was predicted but nonetheless failed to take place is unclear, and merits further study. We suggest that functional cases may have received a poorer standard of care, that the younger dementia group, male and female, may, at least in the relatively short term, prove manageable within the community, and that the very elderly female group, being less behaviourally disturbed and less physically fit, find their way into geriatric care or tolerant old people's homes.

The most cogent point of all is that setting up a service and allowing it to develop an acceptable style cannot be regarded as an ongoing state of grace. Unless there is continuing development, circumstances will dictate priorities, to the detriment of certain patient groups.

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A Study of Firesetters in the South-West of Ireland

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A survey of 54 firesetters who had been in hospital or prison was conducted. The results confirmed earlier findings on several aspects, such as male predominance, unstable childhood and proclivity to self-injurious behaviour. Revenge emerged as the commonest motive overall and an association with alcohol was found. Only 11% of the fire-raising episodes, whether among hospital or prison groups, were truly trivial. Arsonists in prison and hospital had many features in common and generally were quite psychologically disturbed, which contrasted with those who set fires for profit. A poor outcome was found, with continuing self-harm and a high suicide rate. Our findings suggest that arson recidivism arises in at least 35% of arsonists.

The concept of fire has always attracted attention. Originally it was the gift stolen by Prometheus from the gods. Literature and our everyday language abound in references to fire. Metaphorically, it has connotations of power and passion and we are all aware of the fascination which fire holds for children. Sam Shepard (1985), writing on arson, describes setting a wooded area alight and portrays the subsequent sensuous gratification evoked by watching "the beautiful orange glow eat up the blackness" and smelling "the blue smoke cut through the mountain air".

It is not surprising that firesetting attracted the attention of early psychoanalysts. They regarded it as a regressive phenomenon related to urethral eroticism, with a sadistic element (Freud, 1932). The supposed association with childhood enuresis played a large part in the formation of this hypothesis. Objective evaluation of firesetters has arisen in more recent years, beginning with the comprehensive study of Lewis & Yarnell (1951). Some general features of fireraisers have emerged, including a male predominance, with poor social adjustment and psychosexual problems which could be related to an unstable early environment. However, despite the ubiquity and antiquity of the problem, there is a general sparsity of information, particularly in relation to the long-term outcome of these individuals.

The need for further research on firesetters is highlighted by the fact that it is now believed that in the United States at least 50% of all fires not labelled as arson are started intentionally (Blumberg, 1981).

Self-immolation by fire is reported to be uncommon. Occasionally it is used as a means of dramatisation of political protest. More often it arises in females with severe psychopathology such as a psychotic or depressive illness (Scott, 1978).

Method

Firesetting, for the purpose of this study, was defined as the deliberate setting alight of any property, including one's own clothing, which might or might not endanger life. Several methods were used to identify the sample of firesetters studied. A list of patients who were previously known to have set fire wantonly was collected by personally contacting all the senior medical and nursing staff at the two major long-stay psychiatric hospitals and four acute adult psychiatric units within the Southern Health Board of Ireland. All those approached cooperated, but needless to say some remembered more firesetters than others and the same arsonists were mentioned by several different personnel. We believe that the bias of such a sampling procedure favoured those in the middle range of dangerousness, as the trivial would have been forgotten by the respondents and the extremely dangerous would have been committed either to prison or to the Central Mental Hospital, which is the equivalent of Broadmoor.

This method yielded 37 firesetters. Seven of these were self-immolators, all of whom had carried out their self-immolation while on a hospital ward. Of the remaining 30 arsonists, eight (26.6%) carried out the firesetting act while in hospital, and for 17 (77%) of the remaining 22, admission to a psychiatric hospital was directly related to their fire-raising. As it was not possible to interview personally all of these firesetters, for a variety of reasons (no longer in contact with the psychiatric services or through death), it was decided that the case-notes should be examined in detail. Information was collected about each individual, which included his adult social environment, the circumstances of his upbringing and the recorded type of psychopathology (based on ICD-9) present at the time of the burning. A particular effort was made to clarify the circumstances of the firesetting, including any association with alcohol or drugs, whether done alone or with an

TABLE I
Age and geographical origin

	Prisoners		*Hospital group*				*Total*	
			Self-immolators		*Other hosplal patients*			
	(n = 17)		*(n = 7)*		*(n = 30)*		*(n = 54)*	
	Number	*(%)*	*Number*	*(%)*	*Number*	*(%)*	*Number*	*(%)*
Average age: years	[23.9[1]]		[31.5]		[37]		—	
Age group:								
15–25	11	(65)	2	(29)	8	(27)	21	(29)
26–35	4	(24)	4	(57)	7	(23)	15	(28)
36–45	1	(6)	—		10	(33)	11	(20)
46+	1	(6)	1	(14)	5	(17)	7	(13)
Urban	7	(41)	6	(86)	18	(60)	31	(58)
Rural	10	(59)	1	(14)	12	(40)	23	(44)

[1] Excluding 65-year-old schizophrenic

accomplice, what the likely motivation was and what were the likely consequences. If information was lacking in any of these areas, it was sought from the medical and nursing staff who had dealt with the individual.

Seventeen prisoners were examined individually for the study at Cork Jail, which serves the counties of Cork, Kerry, Waterford and Tipperary. The authors are confident that these were all the fireraisers who served time in Cork Jail during 1985, either as remand or convicted prisoners. Each admitted the burning to the examining doctor (MJK). A detailed semi-structured clinical interview was used, covering the same areas as for the hospital sample.

An effort was made to obtain a third sample comprising those who criminally burn for personal gain. These are people who set fire to their own property with a view to benefiting from the insurance or from a malicious damages claim. For the purposes of constructing such a sample, after several preliminary meetings, a two-hour discussion was arranged with three loss adjusters, who prefer to remain anonymous (a loss adjuster is a professional person employed by an insurance company to put a value on the extent of damage to property and if possible to ascertain the cause).

It was not possible to collect information from the loss adjusters on individual cases of possible non-accidental burning (because of their fears of breaching confidentiality) which could be compared in a quantitative way with the prison and hospital samples. From their point of view, non-accidental burning falls into two groups. The larger is where the property is maliciously burnt by people other than the owner. (Several of the prison population in the present study were known professionally to the loss adjusters.) The smaller group is where the property is burnt by the owners themselves.

Results

The prisoners were younger than the hospital patients and more likely to be of rural origin (Table I). The difference in mean ages between the two samples is statistically significant (t-test, $P<0.01$). One prisoner, aged 65 years, was suffering from schizophrenia and is excluded from the average age quoted for the prisoners. The difference in urban/rural origin fails to reach statistical significance.

All the prisoners were male by virtue of the sampling procedure. Within the hospital group, 86% of the self-immolators were female, but the situation was reversed for the other hospital patients, 77% being male. The prison and hospital samples did not differ in other demographic characteristics. Both showed a preponderance of single marital status (70%), lower social class (70%) and a high degree of unemployment (78%).

On examination of the childhoods of these individuals it was found that five (10%) had been reared in orphanages or some place other than the parental home. Of the 49 who were reared at home, there was mental illness or psychopathy present in the parents of 37%. Altogether an unstable early environment can be inferred for a total of 46%.

The diagnoses of psychosis and personality disorder were commonest, constituting two-thirds of the sample (Table II). For those ascribed a diagnosis of personality disorder, there was independent evidence in the history to support the diagnosis other than the act of arson itself. The paucity of those ascribed a diagnosis of depression is surprising, particularly for the self-immolators, 71% of whom were psychotic.

On examination of the tendency to engage in self-injurious behaviour, firesetters emerge as a disturbed group of individuals. A history of prior self-mutilation was present in 9 (53%) prisoners and 12 (32%) patients. Prior parasuicide had arisen in 5 (29%) prisoners and 24 (65%) patients. If cognisance is taken of the tendency to self-harm, it can be inferred that the prison sample is quite psychologically disturbed. Eight (47%) prisoners had previously been in a psychiatric hospital and four (13%) of the hospital group had previously been in prison. Overall there is considerable overlap between the hospital and prison groups. However, the prisoners were more likely to have engaged in other antisocial activities: 11 (64.7%) prisoners had a history of antisocial activities compared with 10 (27%) of the hospital group, the difference between the two groups being statistically significant ($\chi^2=3.84$; d.f. $=1$; $P<0.05$). When

TABLE II
Diagnoses ascribed at any time

Diagnoses	*Prisoners* (n = 17)		*Hospital group*				*Total* (n = 54)	
			Self-immolators (n = 7)		*Other hospital patients* (n = 30)			
	Number	*(%)*	*Number*	*(%)*	*Number*	*(%)*	*Number*	*(%)*
Psychosis	3	(18)	5	(72)	13	(43)	21	(39)
Depression	—		—		4	(13)	4	(7)
Mental handicap	3	(18)	—		1	(3)	4	(7)
Personality disorder only	4	(24)	2	(28)	10	(33)	16	(30)
Alcohol dependence only	2	(12)	—		2	(7)	4	(7)
No psychiatric illness	5	(29)	—		—		5	(9)

the relationship between diagnosis and symptoms was examined, those ascribed the diagnosis of personality disorder emerged as the most severely disturbed, many having engaged in prior self-harm and antisocial behaviour.

Regarding the fireraising act itself, an accomplice was present in only three cases (9%). It transpired that nine of the prisoners and ten patients had taken substantial quantities of alcohol at the time, and alcohol may have played a causative role in the act, as it appears that underlying aggressive instincts were unveiled which might otherwise have remained dormant. Only two subjects were under the influence of drugs and both of these were prisoners.

Revenge was the commonest motive in both populations (Table III). As expected, firesetting instigated by suicidal and delusional motives was more prevalent within the hospital sample, but delusional motivation was also represented in the prison sample. The motive was recorded as delusional if the fireraising arose secondary to a false belief which had developed during a psychotic illness; for example, one man set fire to cars under the delusion that the firesetting would avenge the suffering of deprived children of the world.

Where an individual attempted to influence others or enforce change to secure personal favours by the act, the motive was listed as manipulative; for example, one prisoner set fire to his cell in an effort to precipitate his transfer to another prison. The category 'motiveless' was used for those who, despite their admission of guilt, could not provide any reason for carrying out the act.

Regarding the relationship between diagnosis and motivation, revenge was the primary motive for about half of all diagnostic groups, except for the psychotic, of whom only two of the 21 cited revenge as their motive. Almost half of the latter were motivated by delusions and eight by suicidal attempts. Alcohol was present in 50% of those motivated by revenge, compared with 35% for the total, but the difference does not reach statistical significance. The targets of the revenge were diverse and included relatives, neighbours and figures of authority. Those motivated by tension-reduction tended to engage in other tension-reducing activities such as self-mutilation and parasuicide. Four of the five so motivated were recurrent firesetters.

Regarding the potential consequences of the fireraising activities, in only 11% of the cases, whether among the prison or hospital group, were they truly trivial. The prisoners' fireraising was more likely to result in serious damage to property, while only the hospital group endangered themselves or others. Generally, the prisoners were more likely to externalise their aggression, while the patients tended to direct it inwardly. This difference was statistically significant ($\chi^2 = 5.02$; d.f. = 1; $P < 0.025$).

The average time interval between the firesetting episodes and the study was 5.65 years for the hospital group. At the time of the study, of the 30 hospital arsonists, 15 (50%)

TABLE III
Motivation

Motives	*Prisoners* (n = 17)		*Hospital group*				*Total* (n = 54)	
			Self-immolators (n = 7)		*Other hospital patients* (n = 30)			
	Number	*(%)*	*Number*	*(%)*	*Number*	*(%)*	*Number*	*(%)*
Revenge	8	(47)	—		12	(40)	20	(37)
Manipulative	2	(12)	—		—		2	(4)
Tension reduction	2	(12)	—		3	(10)	5	(9)
Sexual gratification	—		—		—		—	
Delusional	2	(12)	2	(29)	5	(17)	9	(17)
Suicidal	—		5	(71)	7	(23)	12	(22)
Gain	—		—		—		—	
Motiveless	3	(18)	—		3	(10)	6	(11)

were living in the community, 12 (40%) were in long term care; 1 (3%) was in prison; 1 (3%) had died of natural causes and 2 (7%) had died by suicide. This latter finding contrasts with a suicide rate of 13/100 000 (0.013%) described by Clarke-Finnegan & Fahy (1983) in a population-based study in the West of Ireland. Overall, a poor prognosis with a high suicide rate is the picture which emerges for hospital arsonists.

At the time of the study, from the entire sample, 19 (35%) had been involved in one or more episodes of further firesetting. Of these, seven had a recurrence within the first six months of the original act, and six were recurrent firesetters (i.e. engaged in repeated episodes). The remaining six had a recurrence after periods of between six months and ten years, indicating that arson recidivism can arise even after a lapse of many years.

Of the six recurrent firesetters, all were male, of lower social class, and effectively single, and had unstable childhoods. All had a psychiatric diagnosis: four were personality disordered, one was mentally handicapped and one schizophrenic. All had shown some form of behaviour disturbance: four had engaged in self-mutilation, three had attempted suicide and five had committed antisocial acts. One of them eventually committed suicide.

Material gain did not feature as a motive in this study, but it merits consideration, as the loss adjusters estimate that in Ireland, of the claims against insurance companies for property loss due to fire, there is suspicion that the fire is engineered by the owner in 5% of the cases and there is convincing evidence in about 1%. Typically, those motivated by material gain burn their own property, which is usually overinsured in a pre-planned calculated way, often employing accelerators (e.g. petrol). They are of average or above-average intelligence and mentally normal, and generally come from stable families with assets. Their personalities are resourceful, they have the ability to generate property, and they subsequently deny committing the act. This contrasts with hospital and prison arsonists, whose firesetting arises impulsively against a background of mental instability, personality problems, limited intelligence and deprived childhoods.

Discussion

Literature of the nineteenth century indicated that arson was usually a crime committed by females. Currently it is generally accepted that males predominate and comprise approximately 85% of all arsonists (Lewis & Yarnell, 1951). However, others have suggested that the supposed rarity of female arsonists could be apparent rather than real, due to the tendency to avoid prosecuting them for the crime (Tennent *et al*, 1971). Our results show a preponderance of males among the arsonists. However, among those who used fire for the purpose of self-immolation, there was a reversal of the male-to-female ratio. These appear to be a distinct subgroup, with severe psychopathology in the form of a psychotic illness being present in the majority.

With regard to the other demographics, our results are consistent with earlier studies along several aspects such as a large age range, low social status, high unemployment rate and a paucity of long-term psychosexual relationships. The age distribution was much less scattered for the prison sample, leading to a lower mean age, which is not unexpected, as in general the prison population is comprised of young males. In fact, our results indicate that age may be a factor which biases society's methods of dealing with firesetters. Considering the sparsity of psychosexual relationships, it is likely that the failure of these individuals to achieve or sustain relationships is part of a general inadequacy, which Inciardi (1970) has tersely described by comparing these individuals to hoboes because of the lack of emotional and social ties, together with their susceptibility to problem drinking.

Almost half the individuals in this study may have been rendered vulnerable to psychological disturbance through parental absence or parental mental illness or psychopathy during their childhoods. An unstable early environment has frequently been found among arsonists (Lewis & Yarnell, 1951; Tennent *et al*, 1971; Yesevage *et al*, 1983). Macht & Mack (1968), reporting on four adolescents, hypothesised that their firesetting represented an attempt to re-establish links with their fathers, from whom they had been separated. However, it is more likely that traumatic emotional experiences during childhood and adolescence predispose people to psychological disturbance, of which firesetting may be one manifestation.

No single diagnosis has been found to characterise firesetters. Likewise, our results showed diagnostic heterogeneity. The rather large figure of 38.8% who were psychotic is obviously a reflection of the selection procedure, as in general it is held that psychotics comprise only 10–15% of all firesetters (Blumberg, 1981). Various figures have been cited for the proportion of mentally ill firesetters who are mentally handicapped (Yesevage *et al*, 1983). Although only 7.4% of our cases were actually mentally handicapped, many more were functioning in the dull normal and low average intelligence ranges. Levin (1976) suggested that arsonists frequently have psychopathic personalities. Almost a third of the individuals in this study had personality disorder as a primary diagnosis, many more having it as a secondary diagnosis.

Despite the diagnostic variability, a consistent finding was the proclivity of individuals of all diagnostic types to engage in self-destructive behaviour. McKerracher & Dacre (1966), in a study of arsonists in a special security hospital, found symptoms of self-mutilation and attempted suicide in 13% and 23% respectively. It appears that, for some, firesetting is an activity resorted to at times of despair or tension, and in this respect it is akin to self-mutilation and parasuicide.

Simpson (1975) noted that self-mutilators often have difficulty expressing their needs and emotions and generally have poor verbalising abilities. Geller & Bertsch (1985) observed the tendency of firesetters to engage in non-lethal self-injurious behaviour, and hypothesised that firesetting might be used as a communicative vehicle by those with poor verbalising skills. Mentally handicapped individuals may also use it for this purpose, and this is substantiated by the preponderance of these individuals in the motiveless category.

Ascertainment of the underlying motivation is important as it may be valuable in prevention. Revenge has been found to be the commonest motive among arsonists of all diagnostic types. Virkkunen (1974) estimated that 50% of schizophrenics were motivated by hate, often directed against society in general, and he concluded that their motives were understandable on normal psychological terms. However, in our study the motives of the psychotic were more likely to be related to their illness rather than being rational, and this is reminiscent of the findings of Taylor (1985), that 80% of the offences of the psychotic were either directly or indirectly attributable to their illness. Our results highlight the disinhibiting effect of alcohol, particularly for those whose firesetting was motivated by revenge.

Among the recurrent firesetters, the motive of tension-reduction was commonest, and two of these had some of the features of pyromania as outlined in DSM-III. These include recurrent failure to resist impulses to set fires, mounting tension prior to setting the fire and subsequent release, lack of other motivation such as material gain, and the absence of another disorder. True pyromania fulfilling all these criteria is now considered rare, even though Lewis & Yarnell (1951) estimated that up to 40% of male arsonists were pyromaniacs.

It has been suggested that fire has sexual symbolic meaning for some arsonists and therefore firesetting may be a means of obtaining sexual gratification. However, this did not emerge as a motive in this study, even among the recurrent firesetters, for whom firesetting is supposed to be a means of obtaining sexual gratification. This suggests that previous literature has placed far too much emphasis on the importance of sexual gratification as a motive among firesetters.

In contrast to the degree of psychopathology displayed by many arsonists, those motivated by material gain are generally psychologically 'normal, and their arson represents a calculated, rational act. It follows that prevention of this type of arson can only be achieved by elimination of the financial gain involved.

The poor outcome is a reflection of the general inadequacy and the degree of psychological disturbance of these individuals. Our results suggest that self-destructive behaviour tends to continue and may eventually culminate in suicide. The suicide rate of 66 per 1000 for the hospital sample (excluding the self-immolators) is markedly elevated compared with a suicide rate of 1 per 1000 psychiatric admissions found by Farberow *et al* (1971) and a rate of 1.3-2.5 per 1000 in six psychiatric hospitals (Gale *et al*, 1980). An inability to deal with aggressive instincts can be inferred, aggression sometimes being directed inwards and at other times outwards.

Bradford (1982) has suggested a linkage between homicide, suicide, and arson. Certainly the results of this study would espouse an association between suicide and arson. Furthermore, the firesetting of three individuals contained elements of danger to the lives of others, one of which represented a serious homicidal attempt. Further studies are needed to explore this possible linkage, but our results lend support to it.

Lewis & Yarnell (1951) found recurrence of arson in 30% over a 15-year period. In another study, arson recidivism was reported to be rare: in a 20-year cohort study of 67 arsonists, only three were reconvicted for arson (Soothill & Pope, 1973). However, the low rate of arson recidivism among these individuals might be apparent rather than real, due to failure of detection of further arson by the law-enforcing authorities. Among the 35% who were recidivists in our study, the fact that recurrence was most likely within the first six months implies that adequate assessment, management, and follow-up for at least six months may be potential means of prevention of arson recidivism.

Overall, the prisoners appear to be a more dangerous group, when the consequences of their fireraising and history of prior antisocial activities are considered. However, there is considerable overlap between the two populations, many prisoners showing evidence of psychological disturbance and a third of the hospital sample having a history of antisocial activity. Society's method of dealing with these appears to be haphazard and arbitary, with irrelevant variables such as age and origin playing determinant roles in the disposal of these individuals. It is generally recognised that there is a high prevalence of psychiatric disorder among prisoners. In a recent survey of those on remand in Brixton, Prison, Taylor & Gunn (1984) found major symptoms of psychiatric illness in 9% and withdrawal symptoms in a further 8.6%. Interestingly, they found that 65% of arsonists were psychiatrically disturbed. Our study demonstrates the overlap between the mental health and the criminal justice systems and also highlights the arbitrariness of society's method of dealing with these individuals.

Overview of the results of this study leads us to make certain recommendations regarding the assessment and management of firesetters. Even though no reliable prediction can be made on the diagnosis of a mentally ill firesetter, he is likely to be of below average intelligence and to display a general inadequacy in many aspects of his life. Other areas might also be usefully explored, including the ability to communicate and express emotions, the methods of dealing with tension and frustration, and the presence of problem drinking and any unveiling of aggressive instincts under its influence. Ascertainment of the underlying motivation, even though it may be difficult and elusive, should be attempted as far as possible.

Whatever setting these individuals are assigned to, there should be adequate provision of medical care and rehabilitation. Bowden (1978) has shown that nearly two-thirds of mentally abnormal criminals who get the chance of treatment show some improvement. It is likely that many have been subjected to much deprivation, beginning in their childhoods and perhaps continuing thereafter, predisposing them to a general inadequacy in many aspects of their lives. Further education of those with poor communicative skills and low intelligence scores would be beneficial.

In view of the large number who offend as a direct or indirect consequences of their illness, it is important that adequate treatment is provided, followed by suitable rehabilitation programmes. Regular follow-up examinations for at least six months, with admission at times of stress and tension, is advisable and is also likely to be a valuable preventative measure.

In conclusion, firesetting is a highly complex behaviour: it is not simply a manifestation of a basic sociopathic inclination but for many is indicative of severe underlying psychopathology. Firesetting appears to have many different meanings and determinants for various individuals, and one hypothesis alone is unlikely to explain why some resort to it. However, it is clear that many of these individuals suffer much inner disharmony, and attempts should be made to alleviate this where possible. The importance of adequate assessment and management is borne out by the potentially serious consequences of many incendiary acts and also by the high rates of arson recidivism.

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In Vivo Assay for Neuroleptic Receptor Binding in the Striatum
Positron Tomography in Humans*

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Using PET, we investigated the potency in six patients of therapeutic doses of neuroleptic drugs for preventing specific binding of trace doses of intravenously administered ^{76}Br-labelled bromospiperone to corpus striatum *in vivo*. Measured receptor occupancy showed a clear-cut dose-dependent saturation curve with increasing daily oral dose of neuroleptics, indicating the validity and reliability of the method when used as an *in vivo* radioreceptor assay. Following drug withdrawal in eight patients, recovery to normal or supranormal receptor availability occurred in a matter of days. The results demonstrate an approach that may help resolve controversies about, and design better strategies for, neuroleptic treatment schedules.

Neuroleptic drugs are effective in controlling acute episodes of schizophrenia. However, the relationships between dosage and clinical response remain unclear (Davis *et al*, 1983; Curry, 1985), and the indications of maintenance therapy are a matter of debate (Hershon *et al*, 1972; Heinrichs & Carpenter, 1985; Gaebel & Pietzcker, 1985). These uncertainties may be due to insufficient information regarding the access of neuroleptics to, and rate of elimination from, target structures in brain.

The reliability of neuroleptic plasma levels has been questioned because they are not consistently correlated with antipsychotic effects, although radioreceptor assay, which takes into account the active metabolites, has occasionally provided better correlations (Creese & Snyder, 1977; Dahl, 1982; Meltzer *et al*, 1983; Rivera-Calimlim & Hershey, 1984). Neuroleptic drugs levels in brain tissue do correlate with behavioural effects in rats (Campbell *et al*, 1980), but this method is seldom applicable to humans.

Blockade of the central dopamine receptors is held to be the essential mechanism in the pharmacological action of the neuroleptics (Crow *et al*, 1976). Hence, to measure *in vivo* the actual occupation of brain dopamine receptors should provide an indirect estimate of the effective neuroleptic tissue levels, by analogy with the radioreceptor assay principle.

Positron emission tomography (PET) has recently emerged as a unique tool to investigate the striatal dopamine receptors in humans *in vivo* using radiolabelled high-affinity neuroleptics (Wagner *et al*, 1983; Baron *et al*, 1983; Leenders *et al*, 1984; Wong *et al*, 1984; Arnett *et al*, 1985; Maziere *et al*, 1985; Farde *et al*, 1985, 1986). Bromospiperone is a particularly suitable radioligand for this purpose, as shown by extensive validation studies in animals and humans (Kulmala *et al*, 1981; Crawley *et al*, 1983; Maziere *et al*, 1984, 1985; Baron *et al*, 1986). This approach should provide a semi-quantitative estimate of the neuroleptic binding sites available for interaction *in vivo* with the administered radioligand, and be ideally suited to evaluate the receptor occupancy rate both during and following oral treatment with neuroleptics.

Method

Patients

Ten patients (three women, seven men) were studied (Table I) after informed consent. Their age range was 21–82 years (mean 51.2). All had been on chronic medication with oral neuroleptics for more than 2 months. The clinical indications for neuroleptic treatment (Table I) were deliberately heterogeneous in order to ensure an as-wide-as-possible neuroleptic dosage range. Some patients were also receiving benzodiazepines, anticholinergic drugs or antidepressants at the time of the study, but none was on lithium. Four patients were studied twice, first on treatment, and then after withdrawal. In all, there were six studies of patients on treatment, and eight of patients off neuroleptics for periods of 1–12 days.

In order to allow comparison between the different studies, each neuroleptic medication was expressed as a weight-adjusted equivalent daily dose of chlorpromazine (CPZ). The calculation was based on established dose equivalence (Peroutka & Snyder, 1980). When this was

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TABLE I
Clinical data and results

Patient age and sex	*Clinical diagnosis*	*Neuroleptic treatment: mg/kg per day*	$(S/C)_{th}$[1]	*Studies on treatment*			*Studies after withdrawal*		
				CPZ eq. dose[2] *µmol/kg per day*	$(S/C)_m$[3]	P[4]	*Days stopped*	$(S/C)_m$[3]	P[4]
1. 62, M	Alzheimer's disease	Haloperidol (0.010)	1.86	1.4	1.81	93.7	—	—	—
2. 82, M	Senile dementia	Haloperidol (0.017)	1.69	2.4	1.64	92.2	—	—	—
3. 68, F	Chronic hallucinatory psychosis	Haloperidol (0.205)	1.81	—	—	—	1	1.90	111.6
4. 21, M	Schizophrenic disorder	Haloperidol (0.254) Levomepromazine (1.271)	2.21	94.3	1.33	27.3	3	1.87	71.9
5. 50, M	Syphilitic dementia	Haloperidol (0.042) Levomepromazine (0.174)	1.96	13.9	1.40	41.1	3	1.76	79.1
6. 43, F	Schizophrenia	Thioproperazine (1.429)	2.02	285.4	1.27	26.8	3	2.31	127.8
7. 72, F	Post-stroke agitation	Haloperidol (0.027)	1.78	—	—	—	7	1.59	76.3
8. 21, M	Schizophrenic disorder	Haloperidol (0.031)	2.21	4.4	1.65	53.6	7	2.17	96.9
9. 33, M	Alcoholism	Propericiazine (0.526)	2.11	—	—	—	10	2.15	103.6
10. 60, M	Vertigo	Thiethylperazine (0.375)	1.88	—	—	—	12	1.83	94.4

1. $(S/C)_{th}$ is the age-adjusted striatum/cerebellum theoretical ratio.
2. Neuroleptic daily dose expressed as chlorpromazine (CPZ) equivalent.
3. $(S/C)_m$ is the S/C ratio actually measured.
4. P is the percentage of unoccupied binding sites calculated from equation (4).

unavailable for a given neuroleptic, we calculated it from its inhibition constant (K_I) for ^{3}H-haloperidol binding *in vitro* (Leysen, 1984), since a very good correlation between the average clinical daily dose and the K_I for ^{3}H-haloperidol binding has been established (Creese *et al*, 1976; Seeman *et al*, 1976).

PET studies

The method used has been described elsewhere (Maziere *et al*, 1984, 1985; Baron *et al*, 1986). About 1.3 mCi of bromospiperone (BSP) labelled with ^{76}Br ($T_{1/2} = 16.2$ h) was injected i.v. as a bolus with a specific activity of 330±100 mCi/µmol. The amount of bromospiperone injected was 2.75±1.26 µg. From a whole-body kinetic biodistribution study performed in a baboon, it was shown that about 25% of the injected dose was concentrated in the liver. For a ^{76}Br–BSP study in man, the radiation dose absorbed by the liver has been estimated as 1.9 rad, which is lower than that of standard nuclear medicine procedures such as bone marrow or liver scans (Robertson, 1982; Maziere *et al*, 1985). Permission to carry out ^{76}Br–BSP studies was granted by the ethical committee of the Atomic Energy Commission.

PET studies were performed on the time-of-flight positron camera at the Laboratoire d'Electronique et de Technologie de l'Informatique, Grenoble. Spatial resolution in the plane of section is about 12 mm, slice thickness about 13 mm and undetected space between slices about 3 mm using the medium-resolution mode. Seven slices were acquired simultaneously, levels being chosen from a standard PET atlas (Inoue *et al*, 1985) so that the cerebellum was studied at the lower cut and the striatum at the third cut; 1 cm and 4 cm, respectively, above and parallel to the orbitomeatal plane. A ^{68}Ge–^{68}Ga transmission scan was performed for accurate attenuation correction. A 30-min scan was acquired, starting 4.5 h after injection.

From these images, the striatum and cerebellum radioactive concentrations were obtained by means of a standardised, previously validated method using regions of interest (ROIs) (Maziere *et al*, 1985; Baron *et al*, 1986). In the four patients on large doses of neuroleptics,

TABLE II

^{76}Br–BSP relative radioactive concentrations (mean ± s.d.) showing no significant differences among subgroups for any variable

76 Br–BSP concentration	*Controls*	*On neuroleptics*	*Off neuroleptics*	*P*
Cerebellum,[1] $t = 5$ min	1.36 ± 0.52 ($n = 11$)	1.23 ± 0.55 ($n = 6$)	1.19 ± 0.21 ($n = 8$)	NS
Cerebellum,[1] $t = 4.5$ h	1.41 ± 0.41 ($n = 17$)	1.42 ± 0.27 ($n = 6$)	1.42 ± 0.20 ($n = 8$)	NS
Blood,[2] $t = 5.0$ h	0.47 ± 0.19 ($n = 17$)	0.50 ± 0.19 ($n = 6$)	0.50 ± 0.12 ($n = 8$)	NS

1. As percentage injected dose per litre of brain.
2. As percentage injected dose per kilogram of blood.

the striata could not be clearly depicted on the 4.5 h PET images because of receptor occupancy by neuroleptic treatment as shown and discussed below. Since these patients were also studied after drug withdrawal, the ROIs defined on the latter PET images were copied automatically on to the former by means of dedicated software. This was possible because of special care taken for reproducible positioning in these repeated PET studies by use of crossed laser beams projected on standardised bony landmarks.

Determination of the percentage of unoccupied binding sites

Since the cerebellum is virtually devoid of specific binding for neuroleptics (Luabeya *et al*, 1984; Martres *et al*, 1985), the difference between the ^{76}Br–BSP concentrations in the striatum and in the cerebellum represents the specifically bound ligand in the striatum (Kuhar *et al*, 1978). The fraction f of dopamine sites occupied by the neuroleptic medication can therefore be described by the following equation:

$$f = [(S_{\text{th}} - C) - (S_{\text{m}} - C)] / [S_{\text{th}} - C] \qquad (1)$$

where C is the radioactive concentration in the cerebellum, S_{th} the theoretical (expected) radioactive concentration in the striatum and S_{m} that actually measured in the study of the patient. By dividing each term by C, equation (1) becomes

$$f = [(S/C)_{\text{th}} - (S/C)_{\text{m}}] / [(S/C)_{\text{th}} - 1] \qquad (2)$$

where $(S/C)_{\text{th}} - (S/C)_{\text{m}}$ are the expected and the measured striatum-to-cerebellum radioactive concentration ratios, respectively.

The use of equations (1) and (2) relies on the assumption that the parameter C, which is measured at 4.5 h and represents both free ligand and non-specific binding, is unaffected by neuroleptic treatment.

The percentage of neuroleptic sites left unoccupied by the neuroleptic medication can be expressed as

$$\text{percentage of unoccupied sites} = 100\,(1 - f) \qquad (3)$$

which can be rewritten as

$$\text{percentage of unoccupied sites} = 100\,\{[(S/C)_{\text{m}} - 1] / [(S/C)_{\text{th}} - 1]\} \qquad (4)$$

Data analysis

From the radioactive concentration values measured in the striatum and the cerebellum, the $(S/C)_{\text{m}}$ ratio was calculated for each subject.

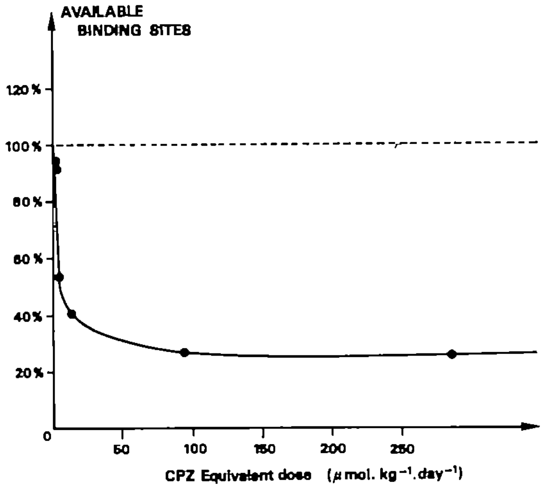

FIG. 1 Available binding sites (percentage of unoccupied neuroleptic sites) in the six ^{76}Br–BSP PET studies performed during neuroleptic treatment, plotted against the weight-adjusted neuroleptic daily dose expressed as chlorpromazine (CPZ) equivalent (CE). The data were best fitted by the bi-exponential line $P = 98 \exp(-0.3\ \text{CE}) + 34 \exp(-0.001\ \text{CE})$.

To obtain the $(S/C)_{\text{th}}$ value, data from 17 control subjects studied by the same procedure were used. However, it was necessary to adjust for age according to the highly significant negative linear regression relating S/C and age found in controls (Baron *et al*, 1986). Finally, using $(S/C)_{\text{m}}$ and $(S/C)_{\text{th}}$, the percentage of unoccupied sites was determined according to equation (4).

Results

Blood and cerebellar values

There was no significant difference between patients and controls in early cerebellar tracer uptake, late blood tracer concentration, or 4.5 h cerebellar uptake (parameter C, see methods) (Table II).

Studies on neuroleptic treatment

For patients on neuroleptic treatment, we found a clear-cut dose-dependent decrease in the $(S/C)_{\text{m}}$ ratio, which ranged from 1.81 at lowest dosage to 1.27 at highest dosage (Table I).

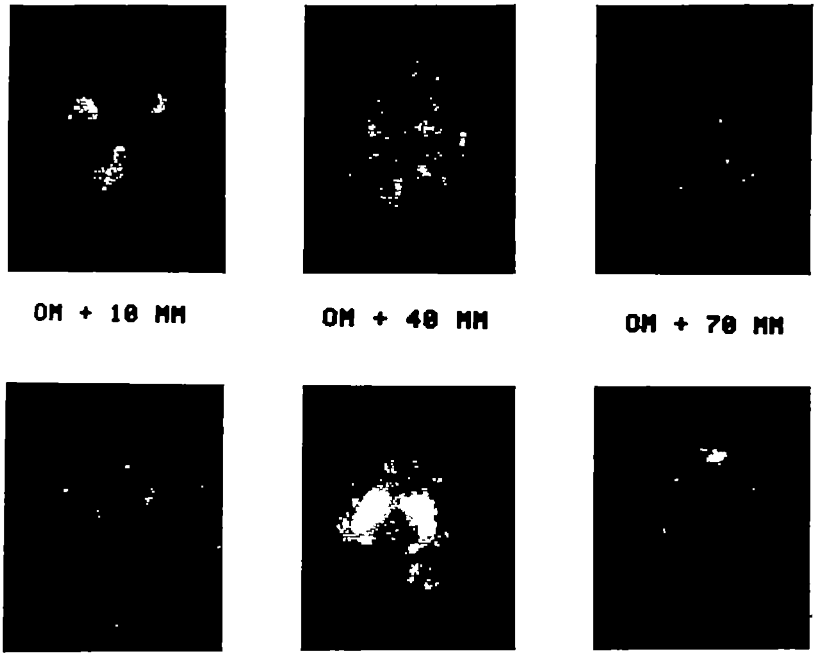

FIG. 2 ^{76}Br–BSP PET images (obtained at $t = 4.5$ h) at cerebellum level (orbitomeatal plane + 10 mm), basal ganglia level (OM + 40 mm), and corona radiata level (OM + 70 mm) in patient 6 both during high-dosage neuroleptic treatment (upper row) and one week later following 3 days of withdrawal (bottom row) (see Table I for details). These images strikingly depict the featureless pattern typically found during high-dosage treatment, indicating almost full receptor occupation by neuroleptics, and the rapid recovery of high radioactive uptake in the striatum, demonstrating in this patient a particularly fast neuroleptic washout from brain.

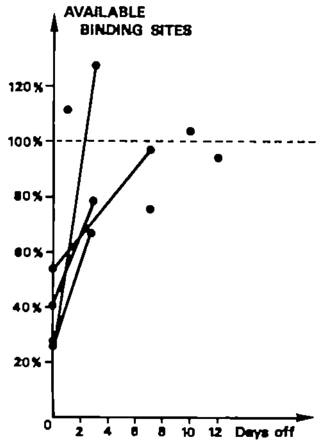

FIG. 3 Measures of available binding sites obtained in the eight studies performed after neuroleptic withdrawal, plotted against the number of days off. The lines join the values measured in four patients both during treatment and following withdrawal.

The percentage of unoccupied sites showed a striking dose-dependence (Fig. 1), and ranged from 93.7% to 26.8% for lowest and highest doses, respectively (Table I). The CPZ equivalent daily oral dose corresponding to 50% of unoccupied sites was about 6 μmol/kg (about 2 mg/kg).

Studies after neuroleptic withdrawal

Within days of withdrawal, there was a rapid trend for the $(S/C)_m$ ratio to return towards normal values (Table I). This was strikingly depicted in the four patients who were studied on both occasions, and particularly in the two patients on high neuroleptic doses, in whom the 4.5-h PET ^{76}Br–BSP image at basal ganglia level changed from a featureless pattern while on neuroleptics to the pattern of high striatal uptake typical of normal subjects 3 days only after withdrawal (Fig. 2).

When the results were expressed as the percentage of unoccupied sites (Fig. 3), it was observed that the majority of patients (6/8) fell into a somewhat curvilinear recovery line apparently crossing the 100% level by 7–12 days after withdrawal. However, the extent of recovery seemed to depend on both the withdrawal interval and the percentage of unoccupied sites during treatment; for the three repeated studies that followed this pattern, the mean percentage of recovery per day was 11.3% (range 6.2–14.9%). The last two patients (patients 3 and 6) were somewhat atypical in that both not only reached but even fell above the 100% level very early (112% and 128% one and three days after neuroleptic withdrawal, respectively), including patient 6, whose percentage of unoccupied sites during treatment was very low (27%).

Discussion

Our results are the first to show a dose-dependent occupation of human striatal binding sites by orally given neuroleptics in chronic schedule; there is no comparable data in the literature, although Meltzer *et al* (1977) found a positive linear correlation between prolactin plasma levels and oral dose of CPZ in humans up to 600 mg/day. Using ^{76}Br–BSP or other radioligands, several previous PET studies demonstrated total occupation of striatal neuroleptic binding sites by various binding sites by various neuroleptics in humans (Baron *et al*, 1983; Maziere *et al*, 1985; Farde *et al*, 1986), but none studied the full range of neuroleptic dosage and, in turn, of receptor occupancy.

The fact that despite very high doses of neuroleptics the striatum-to-cerebellum ratio did not fall to unity (Table I) may seem surprising, but it is consistent with previous results obtained in baboons loaded with unlabelled spiperone and in two human subjects administered haloperidol i.m. (10 mg) before PET study (Maziere *et al*, 1984, 1985). Errors in the estimation of occupancy rates because of competition between neuroleptics and labelled bromospiperone are unlikely, for the amount of radioligand administered was so small (5.8 ± 2.7 nmol) as theoretically to occupy less than 1% of the striatal receptors (Maziere *et al*, 1985). We believe that there was indeed full receptor occupation, but that the *S/C* ratio did not reach 1.0 because of higher non-specific uptake of bromospiperone in the striatum than in the cerebellum (Laduron *et al*, 1978; Barone *et al*, 1985), possibly a reflection of regional differences in, for example, ligand internalisation, neuronal–glial relative density, gray/white matter ratio or spirodecanone sites (Laduron, 1984; Dannies *et al*, 1984; Chugani *et al*, 1985).

Our data indicate that it should be possible to estimate the rate of striatal dopamine-receptor blockade from knowledge of the daily oral dose of neuroleptics. Although the antipsychotic effect of the neuroleptics may be more related to dopamine blockade in the mesolimbic system (Crow *et al*, 1976), there is little evidence to suggest that mesolimbic and striatal receptors respond differently to chronic neuroleptic treatment (Jenner & Marsden, 1983; Roth, 1983); unfortunately, present PET methods are not well suited to investigate this issue. From our work, however, one may infer the dose necessary to achieve full saturation of receptors, above which there may be no further therapeutic benefit, but increased risk of side-effects.

The studies performed after neuroleptic withdrawal showed return to 100% unoccupied sites within a few days (Figs 2 and 3), establishing that washout of neuroleptics from their striatal binding sites is a rapid process. Up till now, it was widely assumed that this washout was much slower, based on clinical, behavioural, and pharmacokinetic data (Hershon *et al*, 1972; Marsden *et al*, 1975; Byck, 1975; Korpi *et al*, 1984; Campbell *et al*, 1985). Our results, however, fully agree with reported neuroleptic clearance rates from plasma (Byck, 1975; Forsman & Ohman, 1977; Itoh *et al*, 1984) and brain tissue (Ohman *et al*, 1977), and with *in vivo* studies in rats (Saelens *et al*, 1980; Ferrero *et al*, 1983; Owen *et al*, 1983). Our findings may help in the design of improved treatment strategies. In addition, they strongly suggest that long-lasting remissions of psychotic patients following neuroleptic withdrawal are not due to persistent dopamine-receptor occupation. Other explanations must be sought, such as protracted neurochemical or cellular changes from neuroleptics (Benes *et al*, 1985), non-dopamine-blocking antipsychotic action of neuroleptics (Nishikawa *et al*, 1985), psychosocial therapy (Hogarty & Goldberg, 1973), or the spontaneous course of the mental illness with time (Davis *et al*, 1983).

In patients 3 and 6, the recovery rate was even faster, with levels above 100% being reached one and three days after neuroleptic withdrawal, respectively. These data presumably indicate dopamine-receptor supersensitivity (Jenner & Marsden, 1983). However, the intersubject variability in underlying mental disorder and in neuroleptic drug regimen may have affected these recovery rates, and will have to be controlled in the future.

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Midsagittal Magnetic Resonance Imaging of Autism

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Since recent reports suggest structural brain abnormalities in autistic patients, we analysed magnetic resonance imaging (MRI) scans of autistic children. Planimetric measurements were done on midsagittal MRI scans, produced with a 0.5 T superconducting magnet. Scans of 13 'high-level' autistic subjects were compared with 35 control MRI scans, read as anatomically normal by a neuroradiologist. Corpus callosal, fourth ventricular, cerebellar, cerebral, and cranial areas were measured. The fourth ventricle was found to be significantly larger in the autistic group. No other areas in the midsagittal scans differed statistically between groups. Results suggest that structures defining the fourth ventricle are anatomically altered in autistic patients.

Past investigations of brain anatomy and morphology in autistic children have yielded variable results. A pneumoencephalography study found dilated left anterior ventricular horns in autistic patients (Hauser *et al*, 1975). Computerised tomography (CT) scan studies present variable results. Several studies found significant parenchymal or ventricular abnormalities in autistic subjects (Damasio *et al*, 1980; Caparulo *et al*, 1981; Campbell *et al*, 1982; Gillberg & Svendsen, 1983); however, there were several equivocal or even negative CT studies as well (Rosenbloom *et al*, 1984; Prior *et al*, 1984; Harcherik *et al*, 1985; Creasey *et al*, 1986).

Likewise, autopsy studies offer conflicting evidence of brain abnormalities in autism (Williams *et al*, 1980; Bauman & Kemper, 1985). A Harvard group reported neuropathological gross and microscopic abnormalities of limbic structures, fourth ventricle, and cerebellum in an autistic 'brain' (Bauman & Kemper, 1985). The same group also reported abnormalities of fourth ventricle and cerebellum in autistic patients studied by CT scan (Bauman *et al*, 1985). Recently a group from UCLA reported cerebellar changes found in a number of post-mortem examinations of autistic patients (Ritvo *et al*, 1986).

Magnetic resonance imaging (MRI) offers several advantages over X-ray CT, especially in investigating the posterior fossa. There is no skull artifact in MRI as found in CT; further, MRI offers multiplanar imaging, whereas CT imaging currently is limited to transverse scans. Considering the reports indicating anatomical abnormalities in autistic brains, we felt it would be of interest to investigate the gross brain morphology of autistic children, by MRI. Thus, we wish to report our initial analysis of the midsagittal MRI scans of autistic children.

Method

Subjects

MRI scans were obtained on 13 autistic patients, seen at the University of Iowa Hospitals, who were diagnosed by the American Psychiatric Association DSM–III criteria. Age, sex, and IQ are given in Table I. IQs were mostly assessed by WISC–R (however, in one case by the Leiter and another by the Merrill–Palmer). Eleven patients were right-handed, one was left-handed, and one was mixed-handed. One patient had generalised tonic clonic seizures and was taking anticonvulsants. Two autistic patients had *café au lait* spots on physical examination, and thus had neurofibromatosis diagnosed by a paediatric neurologist. None of the patients, however, had major structural abnormalities on MRI scans.

The control group was ascertained from the records at the MRI centre. Records were examined of patients aged 4 to 19, who were referred to MRI at the same time as the autistic patients were, and whose MRI scans were read anatomically as 'normal' by the consulting neuroradiologist. Hospital records, and sometimes treating physicians, were consulted to determine significant behavioural problems or developmental delays; control patients with mention of these problems were excluded from this study. There were 35 scans of patients in childhood and adolescence that met criteria.

The control patients were referred for MRI scanning for a variety of reasons; these included a past history of histiocytosis X, headaches, Duane's syndrome, congenital optic atrophy, and others. One control child had neurofibromatosis, diagnosed by *café au lait* spots. Two control children had psychomotor seizures and were taking anticonvulsive medications. Age and sex of the controls are given in Table I. IQs and handedness of the children who served as controls were unknown; however, as mentioned, children with significant learning and developmental abnormalities were excluded from the study.

The control group used in the study is a 'medical' control group. Although it appears that this group is devoid of patients with autism, it may be argued that a 'non-medical' normal control group should be ascertained. However, for a pilot study the 'medical' control group seems adequate,

TABLE I

Demographic data of the autistic and control groups

	Autistic group	*Control group*
Number	13	35
Age range	5–22	4–19
Mean age[1] (±s.d.)	11.3±4.7*	12.0±5.2*
IQ range	60–135	—
Mean IQ (±s.d.)	84.9±26.7	—
Males	10 (77%)	21 (60%)

1. Age data in the groups were tested independently for skewness. The null hypothesis of no skewness was not rejected.
*NS (*t*-test).

especially considering the ethical issues of exposing normal children to a procedure that may require sedation. Moreover, since the medical control group was referred to the same hospital as the autistic group, referral bias would be controlled.

Procedures

The MRI scans were done on a 0.5 tesla superconducting magnet, Picker Vista MR. Scans were recorded on transparent film with a 14 cm to 4 cm scale reduction. Most of the autistic children were sedated with chloral hydrate, 50–100 mg/kg. No problems were encountered during these scanning procedures.

The midsagittal scans were projected from a photographic condenser, life size, on to a digitising tablet (Houston Instruments, Hipad) interfaced to a microcomputer (IBM PC/AT) for digitisation and area measurements. Planimetric measurements were calculated by computeraided design software (Autocad, by Autodesk). Scans were digitised three times by two investigators. The digitising was done blind to diagnostic group. Areas measured by planimetry included corpus callosum, fourth ventricle, cerebellum, cerebrum, and cranium.

Data were analysed by Pearson correlations, multivariate Hotteling's t^2, and univariate *t*-tests in appropriate applications. These statistical tests were done by the Statistical Package for the Social Sciences on a mainframe computer at the University of Iowa's Computing Center, and by SAS on mainframe at the University of Kansas.

Intra-rater correlations were calculated for the five areas. All three raters were above 0.93, with one exception; one rater had a intra-rater correlation of 0.84 of the callosal area.

Inter-rater correlations also indicated that the measures were consistent and reproducible: correlation for the corpus callosum area was 0.94 (average of the three Pearson's *r*); fourth ventricle, 0.96; cerebellum, 0.96; cerebrum, 0.97; cranium, 0.99.

Results

As seen in Table I, the two groups were not skewed in age distribution, and thus comparisons involving brain morphology between groups are valid. Also, there was no differences in results between the sexes by statistical analysis; thus, results (Table II) combine both sexes.

Using only brain areas, multivariate analysis of variance by Hotteling's t^2 was significant ($F=3.24$; $P<0.0106$). Univariate *t* tests were performed independently for each brain area measure (Table II). The results indicate that the fourth ventricle was significantly larger in the autistic group than in the control group. Cranium, cerebrum, and corpus callosum were not significantly different. The cerebellum was smaller in the autistic group, but the difference was not statistically significant.

Ratios were also analysed. Univariate *t*-tests revealed statistically significant differences for the fourth ventricle to cerebellum ratio (0.015±0.006 in the autistic group and 0.009±0.003 in the control group; $P<0.001$) and the fourth ventricle to cerebrum ratio (0.014±0.039 in the autistic group and 0.063±0.025 in the control group; $P<0.0002$). This indicates proportionally larger fourth ventricles in the autistic patients.

Discussion

Results of this study indicate that the MRI brain morphology of the autistic group was different from that of the control group. Specifically, on planimetrically analysed midsagittal MRI scans, the fourth ventricle of autistic children is larger than the same area of control children. The ratio of fourth ventricle to cerebrum also indicates that, proportionately, the fourth ventricle is larger in autistic children, and that the finding is not an artifact of larger brain size. Although there was a trend toward smaller cerebella in the autistic children, no other midsagittal areas measured differed statistically between autistic and control children.

Since this is a small sample size, the study needs replication not only with midsagittal views, but also with transverse and coronal images. However, if replicated, this finding, considered along with the autopsy and CT material, suggests strongly that the fourth ventricle is enlarged in autistic subjects.

The significance of an enlarged fourth ventricle finding is unclear. The fourth ventricle is related to the pons, cerebellum, and medulla. The midsagittal MRI scan images the ventricular length. This allows all portions of the fourth ventricle - inferior, middle, superior - to be seen. The superior portion of the fourth ventricle is bounded by the superior medullary velum and superior vermis in the roof, and the locus ceruleus and medial longitudinal fasciculus (MLF) of the upper pons in the floor. The middle fourth ventricle roof includes the cerebellar vermis and the central cerebellar nuclei; the floor of the middle part includes the MLF, and abducens (VI) and vestibulocochlear (VIII) nuclei. Finally, the inferior

TABLE II
Midsagittal areas (cm^2; mean ± s.d.) in autistic v. controls

	Austistics (n = 13)	*Controls (n = 35)*
Corpus callosum	5.89 ± 1.04	6.24 ± 1.37
Fourth ventricle*	1.51 ± 0.52	0.99 ± 0.33
Cerebellum	15.00 ± 2.46	16.39 ± 2.76
Cerebrum	105.48 ± 9.79	107.42 ± 12.00
Cranium	165.10 ± 14.77	170.23 ± 18.58

*Significant by *t*-test ($P < 0.002$).

medullary velum and the vermal nodulus define the roof of the inferior fourth ventricle, while the hypoglossal (XII) and vagal (X) nuclei, and MLF define the floor.

Of import are structures that have been implicated in autism, that define the fourth ventricle and, if altered, might alter the morphology of the ventricle. The central cerebellar nuclei have been reported to be aberrant in a neuropathological study of one 'autistic' brain (Bauman & Kemper, 1985). In another series of 'autistic' brains, there were widespread abnormalities in several cerebella (Ritvo *et al*, 1986). There are no anatomical studies of the brain stem to our knowledge. However, neurophysiological experiments suggest brain stem nuclei – especially areas in the pons (such as nucleus VIII) – are involved in autism (Ornitz, 1985). Thus, there is evidence that brain areas surrounding the fourth ventricle are involved with the clinical manifestations of autism; alterations in these discrete areas might then alter the space that they define, resulting in a relatively larger fourth ventricle.

Both brainstem and cerebellum have connections with, and affect function of, the limbic system, an area implicated in infantile autism. This suggests that widespread brain abnormalities, in neuronal circuits, are involved in autism. Although speculative, such abnormalities could be caused by aberrant development or early brain injury resulting in brain-tissue loss. Such brain-tissue loss would then result in the enlarged ventricular space.

This study indicates that in midsagittal MRI images, the fourth ventricle appears to be enlarged in autistic patients when compared with medical controls. This has several implications: replication is needed, with midsagittal and with other scanning orientations; and structures defining the fourth ventricle – the brainstem, the pons, and the cerebellum – deserve further morphological investigation in autism.

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Substance-use Disorders in DSM–III–R

Evidence for the Dependence Syndrome Across Different Psychoactive Substances

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Using the newly revised DSM–III–R criteria for substance-abuse diagnoses, we examined dependence syndrome elements among 83 psychiatric patients. The sample included 14 with no history of substance abuse. The remainder abused alcohol (52), sedatives (31), hallucinogens (12), stimulants (33), cannabis (44), cocaine (52), or opiates (47). Many patients (52) had abused more than one type of drug. Ten items assessing the proposed dependence symptoms for each type of drug were factor-analysed. The dependence syndrome items formed a single factor for opiates, cocaine, and alcohol, but not for other drugs. When the items were combined into cumulative scales, they had excellent internal consistency. Furthermore, they formed good approximations of unidimensional Guttman scales on which higher scores indicated greater syndrome severity. The items associated with higher scores differed across drugs, with opiates having the most striking differences from the other substances. Medical-psychosocial consequences were relatively independent of the dependence syndrome, although alcohol and cocaine dependence had some association with other problem areas. These findings support the utility of a common dependence syndrome concept for drugs of abuse as well as alcohol, and provide empirical support for the current revision of the DSM–III diagnostic criteria.

The alcohol-dependence syndrome concept, proposed in 1976 by Edwards & Gross (1976), has stimulated considerable interest in the alcoholism field as evidenced by the development of six different scales measuring syndrome elements and a number of empirical studies providing support for the construct (Orford *et al*, 1976; Litman *et al*, 1979; Stockwell *et al*, 1979; Chick, 1980; Polich *et al*, 1981; Skinner & Allen, 1982; Heather *et al*, 1983; Hesselbrock *et al*, 1983; Foy *et al*, 1984; Edwards, 1986). Within the context of the ongoing work of the World Health Organization (WHO) on nomenclature and classification of alcohol- and drug-related problems, the syndrome construct, hypothesised initially only for alcoholism, has been broadened to apply to other psychoactive substances and is part of the proposed diagnostic criteria for psychoactive substance use disorders in the 1986 draft of the International Classification of Diseases (ICD–10) (Edwards *et al*, 1981; Kendell *et al*, 1986). As articulated in a WHO memorandum (Edwards *et al*, 1981) the drug-dependence syndrome includes two testable postulates about other types of drug dependence: unidimensionality of the syndrome elements and independence of the syndrome from consequences. Unidimensionality means that the syndrome is manifested by the clustering of certain elements to form a single dimension, and since the syndrome is not all-or-none but dimensional, with increasing severity a substance abuser manifests more of its elements. Statistical unidimensionality results from relatively uniform levels of intercorrelation among the items or symptoms within the syndrome and can be assessed by factor analysis and by various measures of internal consistency for the syndrome scale. Independence of the syndrome means that the dependence syndrome constitutes one axis of drug problems, and other problems related to substance abuse, such as legal, occupational, and family consequences of drug use, form a separate, relatively independent axis. Thus, a patient with many or most symptoms of the dependence syndrome would not necessarily have severe legal or family problems, for example, and conversely, a patient with few syndrome elements might have many other severe problems.

In revising the American Psychiatric Association's *Diagnostic and Statistical Manual*, third edition, and in the proposed ICD–10, several major changes have been made in the criteria for substance-use disorders which make this diagnostic system more reflective of the hypothesised dependence syndrome (Kendell

TABLE I

Relationship of DMS-III-R substance-dependence criteria to WHO drug-dependence syndrome elements

	DSM-III-R criteria	*Dependence syndrome elements*
1.	Repeated effort or persistent desire to cut down or control substance use.	Readdiction liability
2.	Often intoxicated or impaired by substance use when expected to fulfil social or occupational obligations or when substance use is a hazard (e.g. goes to work high, drives when drunk).	Salience
3.	Tolerance: need for increased amounts of substance in order to achieve intoxication or desired effect, or diminished effect with continued use of same amount.	Tolerance
4.	Withdrawal: substance-specific syndrome following cessation or reduction of intake of substance.	Withdrawal
5.	Frequent preoccupation with seeking or taking the substance.	Compulsion
6.	Has given up some important social, occupational, or recreational activity in order to seek or take the substance.	Salience
7.	Often uses a psychoactive substance to relieve or avoid withdrawal symptoms (e.g. takes a drink or diazepam to relieve morning shakes).	Withdrawal avoidance
8.	Often takes the substance in larger doses or over a longer period than he or she intended.	Compulsion
9.	Continuation of substance use despite a physical or mental disorder or a significant social or legal problem that the individual knows is exacerbated by the use of the substance.	Salience
—	Reinstatement of excessive substance use after a period of abstinence is much more rapid than initial development. (Dropped from final DSM-III-R.)	Readdiction liability
—	Not represented in DSM-III-R.	Narrowing of repertoire

et al, 1986; Rounsaville *et al*, 1986). The revised DSM-III criteria are described in Table I along with the corresponding elements of the WHO drug-dependence syndrome. The elements hypothesised as clustering together in the dependence syndrome are the following: (a) narrowing of substance-use repertoire such that substance use becomes stereotyped around a regular schedule of almost continuous or daily consumption; (b) salience of substance-taking behaviour such that, despite negative consequences, substance use is given higher priority than other activities which had previously been important; (c) increased tolerance; (d) withdrawal symptoms; (e) substance use to avoid withdrawal; (f) subjectively experienced compulsion to use substance; and (g) readdiction liability (Edwards & Gross 1976; Edwards *et al*, 1981). Because several items have been used to tap some of these dependence elements, the DSM-III criteria include ten, rather than seven, items.

Purpose of the present study

In the current study, the Substance Dependence section of the Structured Clinical Interview for DSM-III (SCID) (Spitzer & Williams, 1985; Rounsaville *et al*, 1986) was used to measure dependence syndrome elements. Two settings were included: (a) an out-patient substance-abuse treatment unit, chosen for the high prevalence of individuals likely to receive a substance-abuse diagnosis, and (b) an in-patient psychiatric unit, in which substance-abuse is seldom the primary focus of treatment, chosen in order to test the system's ability to discriminate between those with and without a substance-use disorder. In the current report, data are analysed to assess the validity of the drug-dependence syndrome as it applies to use of both alcohol and other substances of abuse. To the extent that the dependence syndrome is shown to be valid for a range of substances, this would provide empirical support both for the dependence syndrome construct and for its inclusion in the DSM-III-R and ICD-10.

Two postulates of the dependence syndrome were evaluated: (a) the prediction that syndrome elements cluster into a unidimensional scale, and (b) the relative independence of this syndrome from legal, occupational, medical, family, and psychological problems associated with substance use. To evaluate the clustering of dependence syndrome elements into a unidimensional scale, the ten items pertaining to each substance were assessed for internal consistency using item-scale correlations and by the formation of Guttman scales for each type of drug. The ten items for each drug were also factor-analysed. The syndrome scales were correlated with measures of consequences from the Addiction Severity Index to assess their relative independence.

Method

Setting and sample

Subjects were interviewed at two settings: (a) 41 patients from an in-patient unit of a community mental-health centre, which treats patients with a wide range of diagnoses for 28 days, and (b) 42 patients applying for treatment at an ambulatory substance-abuse treatment unit. In both settings, patients were interviewed as a sample of convenience, when the interviewers were available. For the in-patient unit, the sample was predominantly male (54%), white (51%), with a high school education or more (80%) and with an average age of 34 (s.d. 10 years). For the out-patient setting, the sample was predominantly male (74%), white (62%), with a high-school education or more (81%) and with an average age of 29 (s.d. 7 years). Thus, both samples were demographically comparable.

The patients were also assigned DSM-III diagnoses using the Diagnostic Interview Schedule (DIS), as described in a companion paper (Rounsaville *et al*, 1987). Percentages of in-patients meeting DSM-III criteria (American Psychiatric Association, 1980) for substance abuse or dependence were as follows: alcohol 44%; sedatives 20%; cannabis 24%; stimulants 15%; opioids 34%; cocaine 22%; and hallucinogens 2%. The rates of out-patients meeting abuse or dependence on substances according to DSM-III criteria were as follows: alcohol 55%; sedatives 14%; cannabis 14%; stimulants 14%; opioids 64%; cocaine 74%; and hallucinogens 2%. As anticipated, the rates of substance-use disorders were considerably higher in the out-patient sample who identified themselves as substance abusers by seeking specialised treatment.

Although 14 subjects had no history of drug abuse and 17 reported abuse of only one type of drug, many subjects reported abusing more than one drug. The mean number of drugs abused was 2.3. The three most common combinations were cocaine and alcohol (25 patients), cocaine and opiates (24 patients), and opiates and alcohol (20 patients). Eleven patients reported abuse of all three – cocaine, opiates and alcohol – and two of these reported abuse of all seven types of drug. Other drug combinations were less common, except for cannabis with cocaine (19 patients) and cannabis with alcohol (17 patients). To evaluate the clustering of dependence-syndrome elements and to evaluate their relationship to substance-related consequences, the data from the two samples were first evaluated separately and found to be nearly identical. Hence, for economy of presentation, only the results from the combined samples will be presented here.

Instruments

Two instruments were employed in the present study: (a) the Substance Dependence Disorders section of the Structured Clinical Interview for DSM-III (SCID) (Spitzer & Williams, 1985), and (b) the Addiction Severity Index (McLellan *et al*, 1980; Kosten *et al*, 1983). The SCID is designed to be used by clinicians and provides an interview guide for determining whether subjects meet DSM-III-R criteria for a range of psychiatric disorders. Two graduate-level clinicians conducted all interviews for the present study. For each criterion, which is rated as absent, subthreshold, or prototypical, the interviewer is provided with one or more questions which are read out. If the subject's response is positive or unclear, the interviewer is instructed to continue inquiry using questions of his/her choice in order to elicit examples and confirm the subject's initial response. The SCID is designed to elicit symptoms of current and/or past episodes of psychiatric disorders. For the Substance Dependence section used for the current study, subjects were asked to describe substance use during the most severe episode in their lifetime, regardless of the presence or absence of a current episode.

The version of the SCID used in this study contained an additional DSM-III-R criterion which was subsequently dropped from the final DSM-III-R. This criterion defined the syndrome characteristic of 'readdiction liability' by the statement, "Reinstatement of excessive substance use after a period of abstinence is much more rapid than initial development." The criterion was dropped from DSM-III-R because it presupposed a pattern of excessive substance use and was considered to be a hypothetical characteristic of the entire syndrome rather than an element of it. Because the primary purpose of the current report is to evaluate the drug-dependence syndrome in this sample, we included this additional criterion despite its having been eliminated from DSM-III-R.

The Addiction Severity Index (ASI) was also completed for all in-patients (one had incomplete data) and a pilot sample of 15 of the 43 out-patients. The ASI is a clinician-administered instrument that covers six major problem areas: legal, employment, medical, family, psychological, and substance abuse (McLellan *et al*, 1980; Kosten *et al*, 1983). Composite scales involving several items in each area have been developed to reflect current functioning. The five non-substance-abuse scales were related to the dependence-syndrome elements to examine the relative independence of these consequences from the dependence syndrome (Edwards *et al*, 1981). The 28 out-patients who did not complete the ASI were similar to the remaining patients in demographics and in the frequencies of substance-abuse diagnoses.

Data analyses

The unidimensionality of the dependence syndrome elements was examined using three related techniques: item–scale correlations, Guttman scaling and factor analysis. The item–scale correlations and the Guttman scaling used the ten dependence items from the SCID interview to develop scales to measure the dependence syndrome. The Guttman scaling was attempted with all ten dependence items shown in Table I, in order to examine whether some syndrome elements were common and likely to occur in isolation (low Guttman scores), while other elements were primarily reported by patients with the full-blown syndrome (high Guttman scores). In using Guttman analyses the usual reproducibility coefficient for an acceptable 'perfect' Guttman scale is 0.9. If the coefficients are substantially below this level, the cumulative scores and sequence of high-scoring (frequent) and low-scoring (rare) items will be unreliable (Guttman, 1944). A good fit of the items to a Guttman scale suggests that the cumulative scores formed by adding all the items together, when scored as 0 for absent and 1 for present, were reasonable estimates for the severity of the dependence syndrome. Comparing mean scale scores for abusers, as well as the specific dependence items associated with high and low Guttman scores, across different substances allowed us to examine how the dependence syndrome differed among various drugs. Factor analyses were also performed on the ten dependence items for each drug. When more than one factor had an eigenvalue above 1 by the initial principal components extraction, a varimax rotation was undertaken for all factors with eigenvalues greater than 1. After these dependence syndrome scales were examined for unidimensionality, the dependence scales and the five composite scales from the ASI were correlated to check for independence of the dependence syndrome from these other problem areas.

Results

Internal consistency of dependence syndrome across drugs

The dependence syndrome items were scored as 0 for not present and 1 for subthreshold or prototypical and then added together for each drug type to form seven dependence syndrome scales with scores ranging from 0 to 10. The prototypical and subthreshold ratings were combined to simplify scaling and because subthreshold ratings were rarely made in our sample. The mean scores on these scales for the subjects reporting any of these items, and the number of subjects represented in each mean are shown in Table II, along with two measures of internal consistency - item-scale correlations and Cronbach's alpha. The values for Cronbach's alpha (Cronbach & Furby, 1970) indicated excellent internal consistency for all seven scales, and the item-scale correlations were quite good overall. All item-scale correlations were above 0.5 except for three items in the sedative scale (preoccupation, continue despite problems, and impaired in daily activity), one hallucinogen item (rapid restart of use), and three cannabis items (relief use, withdrawal, and tolerance). The mean scores show that opiate addicts reported the largest number of dependence syndrome elements and had the tightest clustering of these items, while sedative and cannabis abusers reported the fewest syndrome elements. We examined this difference in more detail by studying the distribution of number of items reported for each drug among those patients who reported any dependence syndrome items for each of the seven drugs.

As shown in Table III, item distributions of sedatives, hallucinogens, and cannabis are negatively skewed, with few patients (3–8%) reporting large numbers of syndrome items (8–10 items). In contrast, the distribution of cocaine and opiates are positively skewed, with many patients reporting large numbers of items (48% and 72%, respectively). Furthermore, 27% of the cocaine abusers and 49% of the opiate addicts reported all ten of the dependence syndrome

TABLE II

Item–scale correlations, Cronbach's alpha, and mean scale scores on dependence syndrome items for each drug of abuse (n = 83)

	Drug of abuse						
	Alcohol	*Sedatives*	*Hallucinogens*	*Stimulants*	*Cannabis*	*Cocaine*	*Opiates*
Dependence syndrome items:							
Relief use	0.60	0.55	0.62	0.75	0.39	0.67	0.96
Withdrawal	0.68	0.56	0.58	0.76	0.41	0.71	0.91
Preoccupation	0.74	0.40	0.69	0.67	0.60	0.88	0.89
Rapid restart	0.63	0.58	0.42	0.67	0.60	0.81	0.90
Continue despite problems	0.70	0.43	0.72	0.67	0.59	0.78	0.79
Give up non-drug activities	0.69	0.61	0.68	0.53	0.54	0.79	0.86
Impaired in daily activity	0.69	0.48	0.67	0.66	0.53	0.78	0.86
Tolerance	0.60	0.68	0.68	0.64	0.48	0.77	0.90
Inability to cut down use	0.67	0.58	0.63	0.77	0.53	0.84	0.92
Use more than intended	0.71	0.69	0.78	0.63	0.51	0.77	0.88
Cronbach's alpha[1]	0.91	0.85	0.90	0.91	0.83	0.95	0.98
Mean scores[2] for users only	5.6	3.3	3.7	4.3	3.3	6.9	8.0
Number abusing each drug type	52	31	12	33	44	52	47

1. Cronbach's alpha >0.8 shows excellent internal consistency.
2. Scores ranged from 0 to 10, items scored as 0 = no, 1 = yes. Mean scores only for subjects reporting use of that drug

TABLE III
Number of dependence syndrome items reported for each drug of abuse (n = 83)

	Number of items										
Drug of abuse	*0*	*1*	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*	*10*
Alcohol	31	4	4	7	7	4	3	5	9	5	4
Sedatives	52	8	8	3	2	3	4	2	0	1	0
Hallucinogens	70	3	3	2	1	0	0	1	0	0	1
Stimulants	50	5	7	3	7	2	2	0	2	3	2
Cannabis	39	12	9	6	5	5	3	2	1	0	1
Cocaine	31	3	2	6	1	6	3	6	4	7	4
Opiates	36	2	4	1	1	1	1	3	4	7	23

items. By comparison, 35% of the alcohol users reported large numbers of items. Stimulant users had an intermediate position, with 21% of them reporting large numbers of items. Thus, opiate and cocaine abusers who apply for treatment have a substantial clustering of dependence-syndrome elements, similar to alcohol abusers, but typical sedative, cannabis, and hallucinogen abusers appear to have fewer syndrome elements.

Unidimensionality of dependence syndrome

The above analyses for internal consistency strongly suggested that the ten dependence-syndrome items formed cumulative, unidimensional scales, particularly for opiates, cocaine, and alcohol. As a test of unidimensionality, we attempted to form Guttman scales for each type of drug from the ten items. The results of these analyses are shown in Table IV. As indicated by the reproducibility coefficients, most of the drugs demonstrated good approximations of 'perfectly' unidimensional and cumulative scales. The coefficients were 0.9 or above for all but two drugs – alcohol and cannabis – and these two coefficients were close to 0.9 (0.85 and 0.87, respectively). This indicated that within each type of drug, higher scores were consistent with a more severe dependence syndrome, but the items associated with a more severe syndrome differed across the various drugs. The most striking differences were between opiates and the other drugs. For example, opiate abusers reporting "withdrawal symptoms" almost always reported every other item of the dependence syndrome (score of 10), but abusers of other types of substance often reported this item with few or no other dependence-syndrome items (score of 1–3). At the other extreme, opiate abusers often reported "using more opiates than they intended" along with few or no other dependence-syndrome items (score of 3), but abusers of other types of substance usually reported this item in conjunction with most other items in the syndrome (score of 8–10).

To test further for unidimensionality, we factor-analysed the ten items within each type of drug and found that all ten items loaded on to single factors for opiates, cocaine, and alcohol, as shown in Table V. These single factors accounted for a substantial part of the variance: 83% for opiates, 68% for cocaine, and 56% for alcohol. Obtaining these single factors suggested unidimensionality, that is, endorsing more items was consistent with the unitary concept of more severe drug dependence. Stimulants formed two factors that accounted for 67% of the variance. The factors were labelled "compulsion" and "problematic use", based on the items loading above 0.5. Sedatives and cannabis each had three factors with eigenvalues above 1. Based on the items loading on each factor, two common factors were labelled "can't stop" and "compulsion/salience". The other factor was different for the two drugs and was labelled "problematic use" for sedatives and "withdrawal consequences" for cannabis. The three factors accounted for 69% (sedatives) and 68% (cannabis) of the variance. Because five of the hallucinogen dependence syndrome items were reported by less than four respondents,

TABLE IV
Guttman scale scores[1] using dependence-syndrome items for each drug of abuse (n = 83)

	Drug of abuse						
	Alcohol	*Sedatives*	*Hallucinogens*	*Stimulants*	*Cannabis*	*Cocaine*	*Opiates*
Dependence syndrome items:							
Relief use	1	1	1	1	2	2	4
Withdrawal	2	3	2	6	1	1	10
Preoccupation	3	2	4	2	7	5	9
Rapid restart	4	4	3	5	5	4	2
Continue despite problems	5	5	6	3	3	3	1
Give up non-drug activities	6	6	7	4	4	7	7
Impaired in daily activity	7	10	10	7	10	9	6
Tolerance	8	9	7	9	6	8	8
Inability to cut down use	9	7	5	8	9	6	5
Use more than intended	10	8	9	10	8	10	3
Reproducibility coefficient[2]	0.85	0.91	0.97	0.91	0.87	0.90	0.94

1. Numbers are Guttman scores for patients reporting item; less frequent items have higher scores (see text).
2. Reproducibility coefficient >0.9 indicates good approximation of unidimensional scale.

TABLE V

Factor structure of dependence syndrome items for each drug of abuse[1] *(n = 83)*

	Factor labels by drug of abuse											
	Alcohol	*Cocaine*	*Opiates*	*Stimulants*		*Hallucinogens*	*Sedatives*			*Cannabis*		
Dependence syndrome items	*Dependence syndrome*	*Dependence syndrome*	*Dependence syndrome*	*Compulsion*	*Problematic use*	*Dependence syndrome*	*Can't stop*	*Problematic use*	*Compulsion*	*Compulsion*	*Can't stop*	*Withdrawal*
Relief use	0.67	0.97	0.72	0.57	0.59	*	–	0.59	–	–	–	0.84
Withdrawal	0.74	0.93	0.76	0.74	–	*	0.80	–	–	–	–	0.86
Preoccupation	0.80	0.91	0.91	0.76	–	*	–	–	0.90	0.76	–	–
Rapid restart	0.70	0.92	0.85	–	0.58	*	0.74	–	–	–	0.83	–
Continue despite problems	0.77	0.83	0.82	–	0.83	0.81	–	0.77	–	–	–	0.65
Give up non-drug activities	0.76	0.88	0.84	–	0.83	0.83	–	0.79	–	0.67	–	–
Impaired in daily activity	0.76	0.89	0.83	–	0.65	0.80	–	0.66	–	0.91	–	–
Tolerance	0.76	0.92	0.81	0.81	–	0.80	–	–	0.73	–	0.69	–
Inability to cut down use	0.74	0.94	0.88	0.52	0.66	*	0.86	–	–	–	0.84	–
Use more than intended	0.77	0.90	0.81	0.81	–	0.89	–	–	0.65	0.55	–	–

1. Dashes (–) indicate loadings below 0.4.
2. Rare items (*) deleted.

TABLE VI

Correlations between dependence syndrome scales[1] *and Addiction Severity Index problem severity scores*[2] *for each drug of abuse* ($n=55$)

ASI problem severity scores	*Alcohol*	*Sedatives*	*Hallucinogens*	*Stimulants*	*Cannabis*	*Cocaine*	*Opiates*
	Drug of abuse						
Medical	0.07	0.12	−0.23	−0.08	−0.19	−0.01	0.09
Work	0.14	0.08	−0.29*	−0.10	−0.11	−0.32*	0.12
Legal	−0.12	0.23	0.07	0.21	0.26	0.46***	0.03
Family	0.29*	0.10	−0.05	−0.01	−0.15	0.03	0.13
Psychological	0.32*	0.10	−0.25	−0.06	−0.13	−0.13	−0.12

1. Dependence scale is additive score from the ten dependence syndrome items for each type of drug, scores range from 0 to 10, with higher scores indicating more dependence syndrome items reported; See Tables II and III.
2. Problem scales from ASI are composites of self-report items on which higher scores indicate more severe problems; scores range as follows: legal (−0.01 to 0.18), work (−0.5 to 0), medical (0 to 0.33), family (−0.06 to 0.16), psychological (0.33 to 1.0).

*$P<0.05$, ***$P<0.0005$.

these infrequent items were removed. Including all ten items resulted in three factors, of which one included all five infrequent items. When these items (indicated by * in Table V) were deleted, the other five items loaded together as a single factor that accounted for 69% of the variance.

Independence of dependence syndrome from drug-use consequences

The dependence syndrome was quantified for each drug type using a simple additive scale including all ten items, as described above, and these seven dependence syndrome scales were correlated with five composite problem severity scales from the ASI: legal, work, medical, family, and psychological. Higher scores on these ASI scales indicated more severe problems in each area. The mean scores (± standard deviations) on these five ASI scales were: legal 0.06(±0.05), work −0.12(±0.18), medical 0.09(±0.10), family 0.02(±0.07), psychological 0.74(±0.17). As shown in Table VI, five of the 28 correlations between the seven dependence scales and the ASI scales were significant, and this is more than expected by chance alone. The alcohol-dependence scale correlated with family problems ($r=0.29$, $P<0.04$) and psychological problems ($r=0.32$, $P<0.02$); the cocaine dependence scale correlated with legal problems ($r=0.46$, $P<0.0004$) and work problems ($r=-0.32$, $P<0.02$); and the hallucinogen dependence scale correlated with work problems ($r=-0.29$, $P<0.04$). Overall, however, the ASI scales were relatively independent of the dependence syndrome scales, and even for significant associations the shared variance was small.

Discussion

Is the dependence syndrome valid for all drugs?

This study is a first systematic psychometric examination of the dependence syndrome among abusers of non-alcoholic substances. Based on the newly revised DSM-III-R substance-abuse criteria (Rounsaville *et al*, 1986), we found that the ten items in the draft form of the SCID formed internally consistent scales for all seven drugs under study. For alcohol, opiate, and cocaine abusers the dependence syndrome scale met several criteria for unidimensionality, including all items loading on to a single factor, and forming good approximations to 'perfect' Guttman scales. For the other drugs – stimulants, sedatives, hallucinogens, and cannabis – a clear unidimensional structure was not confirmed by every type of analysis, but even for these drugs, simple cumulative scales seemed to provide satisfactory measures of a dependence syndrome that was independent of medical–psychosocial problems. However, these problems were somewhat related to the dependence syndrome for alcohol and cocaine. Thus, our findings supported the utility of the dependence syndrome for other drugs besides alcohol by specifically demonstrating that the syndrome elements clustered into unidimensional scales for most drugs and that this syndrome was relatively independent of medical–psychosocial problems.

The different types of statistical analysis used to test for unidimensionality of the dependence syndrome were not consistent for all seven drugs of abuse. The simplest tests using raw correlations among items suggested that several items did not substantially contribute to a simple dependence-syndrome scale for every drug. These low contributors differed among the seven drugs, but for most drugs each item was an important contributor. Based on these statistical analyses, no particular item seemed to merit deletion in order to improve internal consistency over all seven drug types. Even when the low contributors were included in a total cumulative scale, we found excellent scale properties according to coefficient alpha, and the mean scale scores were consistent with the item–scale correlations. Scales with high intercorrelations (e.g. opiates) also had high mean scores for the abusers of this drug, because those subjects who reported one item were also likely to report most other items. Scales with lower intercorrelations (e.g. sedatives) had lower mean scores, because abusers of these drugs tended to more frequently report only a few items. Thus, the inconsistencies with unidimensionality seemed minor using simple statistical approaches.

These correlational results encouraged the examination of a stronger model for unidimensionality, Guttman scaling. The good fit of these items to Guttman scales suggested that the cumulative scores were reasonable approximations of severity for the dependence syndrome, allowing us to systematically compare relative severity of dependence across drugs. Among the users of each type of drug, opiates were associated with the most severe dependence and cannabis with the least dependence. This is consistent with general clinical experience for substance abusers and gives further support to this new system for classifying substance abuse. Moreover, polydrug use was common in our samples, and patients who used several drugs during their lives consistently rated opiates and cocaine as more severe problems than cannabis or sedatives. In general, all seven scales appeared to be good unidimensional measures of dependence, in which higher scores indicated more severe dependence.

Comparing frequent to uncommon items within these scales suggested which elements of the syndrome were likely to occur early (frequent items) or late (uncommon items) in the substance-dependence careers of the various types of abuser. Among alcoholics and most other abusers, withdrawal, and "preoccupation with use" were frequent and probably early elements, but these occurred less frequently and were probably later elements among the opiate addicts. This sequence difference among opiate addicts may simply be an artifact of the large percentage of opiate addicts who reported every dependence item, but it is also consistent with the 'chipping' described by most opiate addicts at the start of their careers. Chipping addicts do not get withdrawal symptoms and are not yet preoccupied with getting their next 'fix' of heroin (Chein *et al*, 1964). In contrast, alcohol use is usually not considered a problem until some type of at least mild withdrawal symptoms begin occurring, and the same may be true for cannabis (Litman *et al*, 1979; Marlatt & Gordon, 1980). For cocaine abusers, a 'crash' is commonly described, even following limited recreational use (Gawin & Kleber, 1986), and this crash may be reported as withdrawal symptoms. At the other extreme, many opiate addicts reported "using more than intended" early in their careers. The relatively frequent occurrence of accidental overdoses with opiates compared with accidental overdoses of the other drugs may be an extreme clinical manifestation of this difference, but otherwise there seems no obvious reason why opiate abusers might not share this early characteristic with other illicit drug abusers such as cocaine abusers. Moreover, opiate abusers appeared to be atypical in their development of the dependence syndrome, although they had the strongest support for a coherent syndrome.

The factor analyses of the ten items for each drug raised the most significant questions about the unidimensionality of the dependence syndrome for stimulants, sedatives, and cannabis. For hallucinogens, the separation into three factors seemed partially due to low-frequency items that reflected idiosyncratic responses to hallucinogens, such as withdrawal symptoms. After these items were dropped, the remainder loaded on to a single factor. Although a compulsion factor was formed for stimulants, sedatives, and cannabis, its content was not uniform across the three drugs and these analyses did not support a unidimensional dependence syndrome. The biaxial concept of Edwards *et al* (1981) that dependence may be relatively independent of social consequences was given some support by the "problematic-use" factors for stimulants and sedatives, because the items in these factors reflect social consequences of abuse. Moreover, the single factors that were found for alcohol, cocaine, and opiates demonstrated more coherence among the dependence-syndrome elements than other studies have found for alcohol abuse (Edwards, 1986). In summary, the status of the dependence syndrome for stimulants, sedatives, and cannabis needs further work with larger samples, but based on the other internal reliability analyses we decided to use simple additive scales for all seven drugs as a dependence syndrome axis to examine the postulate that the dependence syndrome was relatively independent of 'drug-related problems'.

The correlations of the dependence syndrome with consequences clearly indicated that many problem areas were relatively independent of the dependence syndrome. The strongest association was between the cocaine dependence syndrome scale and legal problems, and it may reflect the need for increased amounts of illegal activities as the abuser becomes more 'dependent' on the drug. In support of this argument, the only licit substance – alcohol – demonstrated a negative correlation with legal problems, while for all the other drugs the correlations were positive. Other significant correlations accounted for less than 10% of the variance and were inconsistent across drugs, suggesting the need for confirmation before being overinterpreted.

Study limitations

This preliminary study has several limitations. First, the sample size is small and limited to one treatment centre, although it included both in-patients and out-patients as well as subjects who were not substance-abusers. These non-abusers assured that the correlational analyses using the dependence syndrome scales were not biased by limited variability, although the large number of non-abusers for some drugs such as hallucinogens would overstate the tendency for a single syndrome to fall out from the factor analyses. Future studies will need larger samples, perhaps from different cultural settings. Second, these subjects had all sought treatment, and this self-selection bias may need to be addressed through community studies. Third, the ASI was designed to assess both lifetime and current (last 30 days) problems, but the SCID was administered to reflect the period of most intense substance abuse. For the vast majority of these subjects, the current episode of abuse was their most severe, but for hallucinogens, in particular, many of the subjects were not currently experiencing their most severe abuse. Relatively few subjects also reported hallucinogen abuse, and so any interpretations of these findings must be quite tentative. Future studies will need to be designed to have better correspondence between time frames for the dependence-syndrome assessments and the psychosocial measures. Fourth, some subjects had abused more than one drug. The most frequent combinations were opiates with cocaine, and alcohol with cocaine. Using 'pure' drug-abusers would facilitate interpretations across the different drugs, but this is practically impossible in most current treatment programmes, in which abuse of more than one drug is common (Simpson & Sells, 1974; Hubbard *et al*, 1983).

Conclusion

The present study provides encouragement for undertaking more intensive work with other substances besides alcohol using the newly revised DSM-III-R.

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Brief Reports

Conversive Hallucinations

Conversive hallucinations are rare in the psychiatric literature. The authors present a case which demonstrates the psychogenesis and phenomenology of conversive hallucinations in a young female patient.

Hallucinations are usually considered to be psychotic in nature. Conversive hallucinations are very rare. Nemiah (1980) stated that they are rare and usually visual in nature, and are of complex scenes of fragments of action that appear repeatedly and with stereotyped repetition after reproducing the scenes of a real past event of emotional significance to the patient. Farley *et al* (1968) defined conversive symptoms as "neurological symptoms (excluding pain) which are clinically inexplicable". They have reported that the hallucinations occurred as conversive symptoms in 12 of 100 unselected post partum cases. Hirsch & Hollender (1969) described conversive hallucinations in hysterical states. Levinson (1966) reported on an hysterical patient who had auditory hallucinations. McKegney (1967) described two cases of auditory hallucinations as a conversive symptom. Fitzgerald & Wells (1977) described a complex of visual and auditory hallucinations in a young woman as a conversive reaction. Modai *et al* (1980) reported on conversive hallucinations, visual and auditory in nature. Andrade & Srinath (1986) reported on true auditory hallucinations as a conversive symptom in a 10-year-old girl.

Case report

Mrs H – a 28-year-old woman, born in Israel, of Iraqi origin, married and mother of three children – was admitted to Geha Psychiatric Hospital, Israel, because of increased psychomotor activity and visual and auditory hallucinations. She was the third of eight children. The domestic atmosphere was very difficult and tense. She worked in various jobs from the age of 10 years, when her studies stopped. She never felt well at home, and at the age of 15 years she left home and married a 21-year-old man, a delinquent and drug addict without permanent occupation. She gave birth to three daughters. The family lived on welfare funds and Mrs H worked from time to time as a cleaning woman. Her husband was put in jail for long periods for drug-pushing. Mrs H was left alone to cope with the hard reality. While he was at home, her husband exhibited violent behaviour towards his wife and daughters. Despite the constantly deteriorating relationship, she refused to divorce him.

A week before her admission, her husband was discharged from jail and demanded that she give him money for drugs. Mrs H reacted to this demand with increased anxiety, lay in bed, refused to eat or talk, and became restless every time someone tried to contact her. At this stage she was admitted to our psychiatric ward.

On admission, her restlessness, apprehensiveness, anxious affect, and visual and auditory hallucinations were prominent. Her consciousness was clear and her temporal and spatial orientation were intact. She did not exhibit any delusions. The auditory hallucinations were peoples' voices in her ear that prohibited her to eat and talk and threatened to murder her if she talked to anyone. The visual hallucinations were composed of tiny monsters having a human body without a definite colour, very figuratively, that appeared while her eyes were closed at a distance of 5–6 m. Their appearance and disappearance was sudden and they frightened her very much. The hallucinations were tension and spatial-dependent: they appeared only at her home in the presence of her husband. She had insight that the hallucinations were not real. Her physical examination was intact. Her laboratory tests were within normal range. Her electroencephalograph and computerised tomography of the brain were normal.

The patient was diagnosed as having a histrionic personality disorder with conversive hallucinations. This diagnosis was based on her overly dramatic and intensely expressed behaviour, as manifested by exaggerated expression of emotions, incessant drawing of attention to herself and overreaction to minor events, egocentricity, demanding, and dependence and constant seeking of reassurance. She was treated with diazepam 5 mg t.i.d. and psychotherapy.

Twelve hours after her admission the tension, anxiety, and hallucinations disappeared and she resumed her normal level of functioning. After several days she went home for a short time, and the hallucinations recurred at night with the same characteristics as described above. She was brought back to the hospital and as she entered the ward she calmed down and the hallucinations disappeared. During her stay in hospital, together with the individual psychotherapy she and her husband were treated with couple psychotherapy. She was discharged after six weeks in a good remission. At six-month follow-up the hallucinations had not reappeared.

Discussion

Mrs H suffered from two types of hallucinations, namely visual and auditory, while awake. Her visual hallucinations were very primitive and naive. Visual hallucinations of this type, not real, that appear in the inner subjective space out of the dark fields of the eyes with a sense of distance, and suddenly appear and disappear, might be termed pseudo-hallucinations according to Kandinsky, as cited by Jaspers (1963).

These hallucinations had frightened Mrs H, who was in a state of permanent anxiety because of her husband's threatening and her incompetence to cope with his violent behaviour. The hallucinations were an expression of her ambivalent projected aggression towards her husband, a feeling and attitude that were very prominent in her from childhood, when the object of this ambivalence was her father. Her aggressive drives towards her husband and her desire to get rid of him were suppressed by very deep guilt feelings and a fear of being punished for these drives and prohibited wishes. In this way she was able to escape from guilt feelings and at the same time to express her aggression towards him. Using Engel's criteria (1970), this patient was expressing clinically a conversive phenomenon. Engel described four targets of the conversive symptom:

(a) allowing the expression of prohibited wishes (in our case it was a wish to escape from punishment because of her deep guilt feelings);
(b) punishment through guilt feelings (the patient punished herself by becoming ill and suffering from frightening hallucinations);
(c) avoidance of a threatening situation (Mrs H's detention in hospital let her run away, at least temporarily, from coping with her husband's behaviour and attitude toward her);
(d) accepting the sick role (and indeed she received a new role – that of a psychiatric patient).

We can see that our patient had expressed through the conversive hallucinations all four of Engel's criteria. The symptoms originated as a consequence of psychological stress and their appearance offered the patient an escape from a life-threatening situation. Freud (1948) wrote: "flight from a disturbing life situation could take the form of wish-fulfilling delusions and hallucinations". Our patient fits to this statement by her symptoms as described above.

Hirsch & Hollender (1969) stated that a person in a state of extreme anxiety can lose his ability to react promptly to a situation and at this point symptoms like hallucinations can appear.

The only solution which was left for our patient was the production of a conversive symptom, and through it to change the role of a wife and mother for that of the invalid.

Usually the conversive symptom serves a primary gain and a secondary gain. Mrs H's primary gain was preventing the severe anxiety which resulted from unresolved aggressive and sexual conflicts. The secondary gain was running away from a life-threatening situation at home, being detained in hospital and treated as a psychiatric patient, and developing a dependency on the medical and nursing personnel and through it gaining protection and security.

In discussing this case we have to discuss the concepts of hysterical psychosis, culturally sanctioned psychoses, and acute paranoid reaction (boufée delirante). Merskey (1979) wrote that hysterical psychosis is a debatable concept. 'Hysterical insanity' was used by Kraepelin (1904) for severe prolonged hysterical symptoms without delusions or hallucinations being present. 'Hysterical psychosis' was used by Breuer & Freud (1893–1895) in a more usual way, but Bleuler (1950) and Reichard (1956) criticised the term; it was, however, revived by Hollender & Hirsch (1964) for psychotic symptoms of sudden onset and brief duration, arising in patients with hysterical personalities after a period of increasing stress. The symptoms included delusions, hallucinations, depersonalisation, and grossly unusual behaviour, and usually lasted 1–3 weeks.

Mallet & Gold (1964) described 13 patients, all women, that at some point had been diagnosed as schizophrenic because of unsuccessful treatment, emotional emptiness and bizarre depersonalisation, phenomena, paranoid ideation, and dramatic visual and auditory hallucinations. All the patients displayed hysterical personality traits. In every case, admission to hospital followed an acute episode of violent or negativistic and symbolic behaviour or a suicidal attempt. (Five of the patients ultimately committed suicide.)

Hirsch & Hollender (1969) suggested that the illness might present in three ways:

(a) as socio-culturally sanctioned behaviour
(b) as simulation of psychotic behaviour
(c) as a true psychosis with temporary ego disruption and impairment of repression.

Siomopoulos (1971) considers that the delusional ideas in hysterical psychosis lack the characteristics of incorrigibility, which is an essential feature of the delusion proper. The patient described by us differs from the hysterical psychotic patients described above in that she lacked delusions, depersonalisation, and grossly disturbed behaviour. The hallucinations were spatial-dependent and she had insight that they were not real. All these characteristics excluded the diagnosis of hysterical psychosis.

The differential diagnosis of our case should include culture-bound or exotic syndromes. The first are what Hirsch & Hollender (1969) noted where a cultural practice sanctions extreme behaviour. Other examples include latah, ainu, amok, piblokto, wihtigo, voodo and phii pob, all of which are described by Merskey (1979). Allodi (1982) stated that many of the exotic conditions are regarded now as the equivalent of hysterical, obsessional, and anxiety syndromes in which there may be a partial or transient withdrawal from reality; they have been ascribed to the idiosyncratic ego organisation of people living in rural or pre-technological societies and to the conditions prevalent in those cultures. These types of behaviour are determined by the prevailing belief system of the given culture. In contrast to this essential feature, our patient's behaviour as described above was determined far more by individual dynamics than by the prevailing belief system of our culture, or social considerations.

The last entity to be differentiated is the acute paranoid reaction studied by Allodi (1982), named also bouffée delirante in the French literature and psychogenic psychosis in the Scandinavian literature. This diagnosis refers to a subcategory of reactive or other non-organic psychoses which reportedly occurs frequently among immigrants, refugees, and other groups subjected to major social stress. Our patient does not fit this diagnosis because she lacked delusions, had good insight into her hallucinations, did not lose reality-testing, did not have rapid switches from elation to depression, and was not a new immigrant or refugee.

We would like to close this discussion with a quotation from Merskey (1979): "culturally sanctioned behavior, simulation of psychosis, reinforcement of repression, and gross regressive behavior occur in a variable mixture in different syndromes. It is easier to understand these illnesses than to group them systematically."

The authors suggest that psychogenesis and phenomenology of this type in a non-psychotic and non-dissociative patient represent conversive hallucinations and are similar to the descriptions of Fitzgerald and Modai. In the background of these hallucinations were repressed aggressive and libidinal drives that could not be expressed in the patient's daily life with her husband. Only via the conversive hallucinations she was able to express them deliberately. The appearance of hallucinations in the clinical symptomatology does not imply a psychotic state, and the clinician should bear in mind that they can reflect a conversive phenomenon.

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Lithium Toxicity and Mefenamic Acid

A Possible Interaction and the Role of Prostaglandin Inhibition

An elderly patient with bipolar manic-depressive psychosis, stabilised satisfactorily on lithium carbonate, developed acute symptoms of lithium toxicity shortly after commencing mefenamic acid. Vulnerability factors for the interaction may have included age, history of cardiac failure, and impaired glomerular function. It is hypothesised that the interaction may have been mediated by inhibition of prostaglandin activity.

Non-steroidal anti-inflammatory drugs (NSAIDs), such as indomethacin, diclofenac, ibuprofen, and piroxicam, have been suggested to increase serum lithium levels (e.g. Reimann *et al*, 1983). The mechanism of the effect is unclear. This report is the first of a possible interaction between lithium and the NSAID mefenamic acid (Ponstan), which resulted in a clinical syndrome of lithium toxicity. Moreover, it considers the putative role of prostaglandin inhibition in the interaction.

Mefenamic acid is effective in the treatment of pain. Its mode of action appears to be through inhibition of prostaglandin synthesis or possibly through prostaglandin receptor antagonism. Reported adverse side-effects have included non-oliguric renal failure. None has involved lithium carbonate, a drug which is excreted almost exclusively by the kidney.

Case report

Interaction between mefenamic acid and lithium occurred in a 62-year-old female who had a 12-year history of bipolar manic–depressive psychosis. She had been maintained well for seven years on lithium carbonate when she was admitted to hospital following a myocardial infarction. At this time asymptomatic hypercalcaemia was discovered. Lithium carbonate was discontinued, as it may induce hyperparathyroidism.

Thereafter the patient experienced three further manic-depressive mood swings, and as a consequence lithium prophylaxis was reinstated. Assessment of renal function revealed a creatinine clearance of 35 ml/min, indicating an impaired glomerular filtration rate. Frusemide 40 mg *alt. die* was required to control congestive cardiac failure. The combination of a natriuretic diuretic and the compromised glomerular filtration rate may explain the low dose of lithium carbonate (200–250 mg per day) required to maintain serum lithium levels within the normal therapeutic range. The patient, despite the combined risk, was safely maintained at stable serum lithium levels without any untoward effects.

Six months after recommencing lithium carbonate, however, she presented acutely with symptoms indicative of lithium toxicity: ataxia, mild dysarthria, disorientation, and decreased attention span. Clinical assessment and investigation revealed no other abnormalities. The only recent change in medication was the introduction four days previously of mefenamic acid in a dose of 250–500 mg *q.i.d.* for the relief of pain arising from cervical osteoarthritis. The patient reported that she had previously taken mefenamic acid without side-effects during the time when lithium prophylaxis had been discontinued. A serum lithium estimation 6–8 h after the onset of lithium toxicity was 1.16 mmol/litre, which is close to the upper limit of normal. Although symptoms of lithium toxicity can occur within the normal therapeutic range, especially in the elderly, it seems probable that the serum lithium level would have exceeded the upper limit of normal (1.5 mmol/litre) if venipuncture had been closer to the time of lithium ingestion, approximately 10 h previously.

Discussion

The role of prostaglandins in the maintenance of normal renal function is uncertain (Gullner *et al*, 1980). However, their role is more clearly established in cases where renal function is already compromised, such as with renal disease or cardiac failure (Sellars & Wilkinson, 1983). In such cases, inhibition of prostaglandin synthesis or receptors is likely to compromise renal function further, thereby reducing renal elimination of drugs like lithium. Furthermore, NSAIDs are recognised to have adverse effects on the kidney (Calin, 1983). Predisposing factors to NSAID induction of renal failure include age and diuretic therapy (Henrich, 1983); in particular, NSAIDs may antagonise the effects of frusemide (Yeung Laiwam & Mactier, 1981).

The facts that the patient had previously taken mefenamic acid without any problems and that, whereas she had been safely stabilised on lithium for some time, the onset of toxicity only occurred shortly after commencement of mefenamic acid suggest that this was an interaction between lithium and the NSAID. This is supported indirectly by reports of similar effects with other NSAIDs. Rechallenge, however, was thought unjustifiable because of the potential hazards to the patient; thus the possibility that toxicity was unrelated to mefenamic acid has not been completely excluded.

The question arises as to the mechanism involved. In this case the patient was probably vulnerable to the development of lithium toxicity by virtue of age, impaired glomerular filtration rate, and associated natriuretic diuretic therapy. Because of her physical condition, the prostaglandin system was likely to have been important in maintaining optimal renal function. When a prostaglandin inhibitor, mefenamic acid, was introduced, it probably acted as the trigger for the development of raised serum lithium levels through the further impairment of renal lithium excretion.

This possible interaction suggests that precautions, such as more frequent serum lithium estimations and a reduction in lithium dosage, should be taken whenever lithium and mefenamic acid are combined, especially in those patients where prostaglandin inhibition is likely to have important clinical consequences.

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The Effect of Maternal Psychotic Illness on a Child

We report the case of an 11-year-old girl who had been living with only her mother, a woman with chronic schizophrenia. The girl had been caught shoplifting with her mother, and had a long history of non-attendance at school. The case emphasises that adult psychiatrists should always consider the impact of chronic parental psychiatric disorder on children, particularly in one-parent families.

This case was of interest to one of the authors (AJM) as he had been involved in the mother's presentation during her first, and so far only, admission to a psychiatric hospital.

Case report

An 11-year-old girl, G, the only child in a single-parent family, came to our attention with a four-year history of increasing non-attendance at school. Three years previously, her mother, then aged 38, was admitted to a psychiatric hospital with a diagnosis of chronic schizophrenia. During that admission, the mother described many psychotic symptoms which she had experienced intermittently for about eight years. These included auditory and visual hallucinations, a well-developed grandiose delusional system, and ideas of reference. For instance, she described the emission of a great heat and light coming from a cross on her bedroom wall, that had frightened her so much that she had to take it down. She had seen pictures of a man with a beard and wreaths around his head on the wall and thought this was Jesus Christ. She also had heard evil voices from her stomach calling her names and she was convinced these were from a "higher medium". She thought she was an important person in "The Movement", with special powers. She insisted she was trying to resist being taken to "the astral plains to see God". She talked in a vague and mysterious way about being "tuned in to Ronald Reagan and Margaret Thatcher", but could not describe this connection more clearly. She hinted that things were going on that were too dangerous to talk about, but she was convinced that the interviewer knew about these happenings. She described pains in her foot "like a nail being put through it", without any supporting evidence. She experienced "sex vibrations" inside herself that she interpreted as a sign that Jesus Christ was making love to her. Finally, she was convinced that people were plotting against her and wanted to harm her.

Treatment with phenothiazines reduced her agitation, without affecting her delusional system. The possible impact of the mother's chronic illness on G was not recognised by the hospital psychiatrists, even though the mother had said that "these things must be happening because G experiences them too". The latter statement was regarded as part of the mother's continuing psychotic illness rather than as evidence of the daughter's involvement. G was not interviewed at this time, nor was the family seen at home. Two weeks after admission, the mother took her own discharge and was soon lost to follow-up investigation.

During the next three years, there was further evidence of the detrimental effect of the mother's illness upon G: G was caught shoplifting with her mother; school attendance was irregular and erratic; referral was necessary to the Children's Panel (the equivalent in Scotland of the juvenile court system in England and Wales). G was received into care and placed with foster-parents. The panel subsequently requested a psychiatric opinion, prompted by a strange quality in the mother and daughter's relationship, as well as the mother's lack of concern about G's schooling. Neither the school, the education department, the social work department, nor the Children's Panel were aware of the mother's chronic mental illness, this possibly explaining the delay in referral to child psychiatry.

At the initial joint interview, the mutually dependent and inappropriately close relationship between mother and child became very apparent. They had slept together, and spent most of the day together, as the mother did not work and G had not attended school on any regular basis for some time. G was very protective towards her mother, interrupting when her mother began to talk about her strange experiences, effectively preventing her mother from saying too much. G also said that she was so concerned about her mother that she was reluctant to leave her mother alone in the house.

The mother described psychotic phenomena similar to those described previously, adding that her daughter had similar experiences. G agreed, saying that she heard voices uttering obscenities and swearing and that she had seen a crucifix on the wall: G was reluctant to elaborate further, simply saying "the same as my mum". G did not seem concerned about these events, which only occurred when she was with her mother, implying that G was agreeing with her mother in order to avoid upsetting her. Consequently, we did not think she was independently psychotic.

When interviewed alone, G was a pleasant, co-operative girl, with good rapport. There was no evidence of any hallucinations or delusional experiences. She was, however, unwilling to discuss the unusual experiences described by her mother, saying that they were in the past and were best forgotten.

Independent information provided by the foster-parents confirmed that G had not shown any bizarre behaviour or reported any unusual experiences. Additional information from the school revealed that her IQ was 85, and her reading age 7 years 9 months, at a chronological age of 11 years 5 months. The school were especially worried about G's emotional immaturity, describing her as "childlike", with poor peer relationships. Her teachers thought that she would be at great risk at an ordinary secondary school, particularly because she was physically mature and superficially appeared older than her years.

The subsequent psychiatric report to the Children's Panel recommended residential school placement for G, with regular liaison between home and the school from the social work department. G has now been away at school for two terms and is progressing well.

Discussion

The association between parental mental illness and disturbance in children has been recognised for over half a century. Rutter (1966) showed that the parent–child association of psychiatric disorder was strongest when the parental symptoms impinged upon or were involved with the child in some way. The children were most at risk when they were the victims of aggressive acts of hostile behaviour, were the target of parental delusions, were neglected for pathological reasons or were involved in parental symptoms. Other studies of clinical and general population samples have confirmed this association, with the greatest risk when the illness is chronic or recurrent (Beardslee, 1986; Cytryn *et al*, 1986; Quinton & Rutter, 1984; Weissman *et al*, 1984).

In this case, the mother's chronic psychotic illness was responsible for the development of an over-close and over-dependent relationship with her daughter, with serious detrimental effects on the child's education, independence, and peer relationships. Although the child's bizarre statements were not psychotic, the case shows how a child may become so involved in the parental psychopathology that she complies with parental requests for confirmation of psychotic experiences and also refuses to attend school in order to protect and look after her psychiatrically disturbed parent.

Many of G's problems might have been averted if she had been seen during her mother's admission, when the adverse effects of the mother's illness on G would have been apparent. Adult psychiatrists should always consider the whole family, especially in one-parent families, when a parent has a major psychiatric illness. The consequences of this omission in G's case was a further three years of poor school attendance, with impaired social development. A family assessment could also have alerted the social work department and the Children's Panel to the child's needs at an earlier stage. Finally, we would recommend strenuous efforts to maintain follow-up investigations with such cases, with the involvement of the community psychiatric nursing services and the general practitioner, to ensure that the risks for children in such situations are anticipated and, one hopes, ameliorated.

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Early-onset Huntington's Chorea

Diagnostic Clues

A patient with early-onset Huntington's chorea is described. The diagnosis had been overlooked during previous contacts with psychiatric services because there was no family history, and features typical of the adult disease were absent. Distinctive findings on CT scan and electroencephalography were observed.

The early onset of Huntington's chorea can lead to marked diagnostic difficulty, particularly in the absence of a positive family history. The so-called 'juvenile' variant is characterised less often by chorea than by extrapyramidal rigidity, dysarthria, and occasionally, cerebellar signs. Intellectual deterioration with failure at school and difficulty with concentration are usually the presenting features, but may for some considerable time be attributed to poor initial endowment. Personality change, accompanied by anxiety, is often prominent. Though depression is held to be the most common affective disturbance in Huntington's chorea, its true incidence is difficult to determine. The incidence of schizophreniform psychosis preceding or accompanying the abnormal movements varies from 5% (Brothers, 1964) to 12% (Oliver, 1970), and the delusions are usually paranoid in nature. There is no obvious relationship between this presentation and other clinical features (such as chorea or dementia), but it seems that schizophreniform psychosis is more commonly seen in younger patients with Huntington's chorea and that it may be an associated feature of the disease (Garron, 1973). Seizures occur in up to a half of patients affected in childhood, in contrast to their rarity in the adult disease.

The prevalence of early-onset cases among patients with Huntington's chorea has been variously estimated at 1% (Stevens, 1976) and at 10% (Oliver, 1970). Hayden & Beighton (1982) found that 4.7% of 2699 cases in the literature had begun before the age of 20. This may yet represent an underestimate. Progress of the disease was noted by Hayden (1981) to be more rapid than in adults. An intriguing feature is the finding that children with Huntington's chorea have an affected father approximately four times as frequently as an affected mother (Osborn *et al*, 1982), though the possibility of sampling errors in contributing to this cannot be excluded (Vegter-Van Der Vlis *et al*, 1976).

We report here a young man who presented to a hospital for the mentally handicapped after a history of various social and psychiatric problems from about the age of 11. The diagnosis of Huntington's chorea was made only at the age of 27, and shortly thereafter he died. Important clues to the diagnosis lay with distinctive findings on the CT scan and the electroencephalogram. He was illegitimate and the fate of his father was unknown. There was no positive family history.

Case report

In 1983 a single 25-year-old man was admitted to a hospital for the mentally handicapped. He was judged at that time to have low intellect and to suffer from a personality disorder and depression. Physical signs on admission included ataxia, tremor, muscular rigidity, and dysarthria, but neurological opinion was that these were attributable to the neuroleptics he had been given. There was no family history of psychiatric disorder.

Labour had been prolonged, but otherwise birth and early development were normal. His mother described him as a bright and inquisitive child who had performed well at primary school. At the age of 11, a verbal reasoning test had shown an IQ equivalent of 103.

From then on, however, he showed decreased willingness to work, vagueness, and increasing indiscipline. Over the years he became physically aggressive, with truanting, stealing and 'failure to mature'. He was noted to be generally anxious. Upon leaving school he obtained numerous labouring jobs, felt to be inconsistent with his early educational promise. The first of many appearances for theft at juvenile courts took place at the age of 16.

At 18 he was assessed psychiatrically as having a personality disorder with depression. At 20 his IQ was measured at 86, with a mental age of 13 years 4 months. At about this time his speech became difficult to understand because of slurring, and he became progressively more withdrawn. During several hospital admissions thereafter, he was variously regarded as psychotic and mentally handicapped, and was treated with a variety of neuroleptics, without clear evidence of benefit. At 22 he was deemed fit to plead to charges of indecent exposure and of a sexual offence against a pet dog.

In hospital from 1983 onwards he remained withdrawn, anxious, and extremely slow both verbally and physically. He ate poorly, complained of abdominal pains, and lost a great deal of weight. Psychometric assessment was very difficult, but in 1984 he scored an IQ equivalent of 67 on the coloured progressive matrices and obtained a level of only 4 years 7 months on the Vineland Social Maturity Scale. All medication was discontinued in 1984.

On detailed review in 1985 he showed marked slowness and anergia and walked with a stooped, wide-based ataxic gait. He replied monosyllabically to questions, with a marked dysarthria, but proved to be oriented and to know his age. Reading and writing skills appeared to be intact. Tone was markedly increased in the arms but not in the legs, and all tendon reflexes were exaggerated. The plantar responses were probably flexor. The fingers of the left hand showed abrupt jerking movements, mainly when handled but occasionally spontaneously, and there were jerking tremors around the mouth. He found difficulty in rising from the seated position, although there was no evidence of muscular wasting. The cranial nerves and optic fundi were normal. Sensory examination was difficult to perform, but revealed no clear deficits. The extremities were markedly blue and cold.

The diagnosis was felt to lie between Huntington's chorea and Wilson's disease or some more obscure neurological degenerative condition. An EEG revealed a very low-amplitude tracing, almost totally devoid of alpha rhythm, with bursts of low-amplitude theta waves bilaterally from time to time. A CT scan was also abnormal, with moderate diffuse atrophy and flattening and concavity of the lateral borders of the anterior horns of the lateral ventricles due to shrinkage of the heads of the caudate nuclei (Fig. 1(a)). Review of a CT scan carried out 18 months earlier, and then considered to be normal apart from some prominence of sulci, showed, with hindsight, similar features. An EEG carried out in 1984 had also been reported as abnormally flat and featureless. On the basis of these investigations a diagnosis of Huntington's chorea was made.

Over the next few weeks the patient declined abruptly, with a marked increase in extrapyramidal rigidity, then died suddenly, with post-mortem evidence of pneumonia. Examination of the brain confirmed Huntington's chorea. The brain was smaller than normal, weighing 1131 g, with mildly widened sulci. The caudate nuclei and putamina were small. The medial borders of the heads of the caudate nuclei were straight, not convex, with corresponding enlargement of the frontal horns of the lateral ventricles (Fig. 1(b)). These appearances corresponded to what was seen on the CT scans. Microscopy showed a loss of nerve cells – particularly the small nerve cells – in the striatum, with a

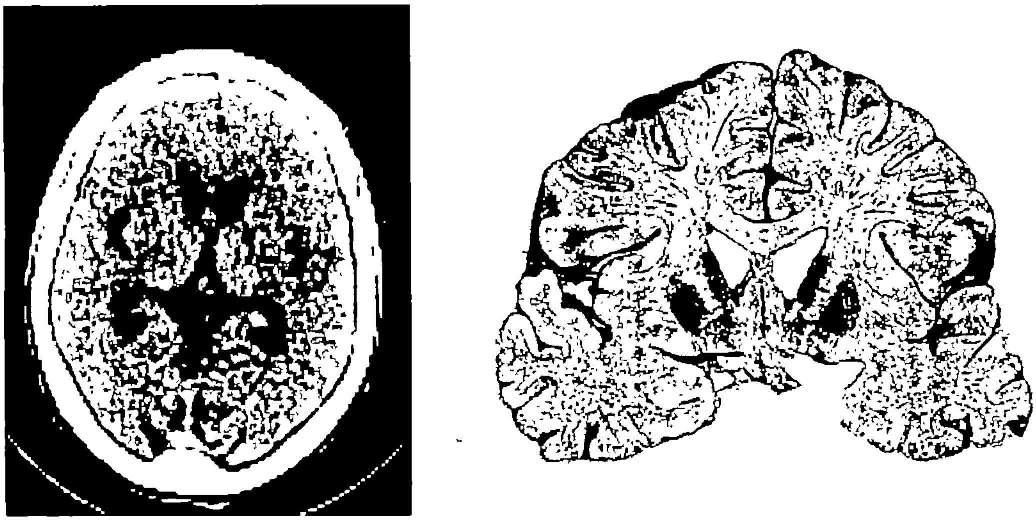

FIG. 1 (a) CT scan; (b) autopsy appearances, illustrating shrinkage of heads of caudate nuclei.

prominence of astrocytes. Some of the bundles of axons, the so-called pencils of Wilson, in the affected striatum, were pale in sections stained for myelin and for axons, rich in small dark nuclei and astrocyte processes. There were no other obvious microscopic abnormalities in the cerebral hemispheres or in the cerebellum and brain stem.

Discussion

The patient we report highlights the difficulty in the diagnosis of Huntington's chorea when a family history is not forthcoming and when the typical choreiform movements are absent. The 15-year-long presentation, with decline in performance from schooldays onwards, antisocial conduct, and quasi-psychotic developments, obscured the true situation, and the administration of neuroleptics led to misinterpretation of the significance of the extrapyramidal features he displayed. The frequency of this form of motor abnormality in juvenile-onset Huntington's chorea is still probably insufficiently appreciated.

The importance of distinguishing dementia from amentia is similarly illustrated here. The prominence of behavioural disturbance, and the patient's disorganised social background, led to an undervaluing of the history of his early progress at school. A label of mental handicap was too readily applied, and no doubt delayed the search for responsible brain pathology.

The value of the often distinctive features on the electroencephalogram and CT scan in pointing towards Huntington's chorea deserves close attention, and would appear to warrant wider application in patients who show intellectual and/or social deterioration of obscure cause.

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Two Cases of Erotomania (de Clérambault's Syndrome) in Bipolar Affective Disorder

Two female patients with longstanding bipolar affective disorder demonstrated de Clérambault's paradigm of erotomania during the euthymic phase of their illness. Although a diagnosis of schizophrenia or paranoid disorder is often given, a review of the literature shows that many cases have features of a major affective disorder, probably bipolar affective disorder and its spectrum.

The belief that love could be a form of, or the cause of, madness has a long history and many entities have fallen under the rubric of erotomania. De Clérambault advanced the concept of pure erotomania, one of the "*psychoses passionnelles*", as a distinct disorder in the 1920s. The "fundamental postulate" required the sudden, precise onset of a woman having "the conviction of being in amorous communication with a person of much higher rank, who has been the first to fall in love and was the first to make advances" (Enoch & Trethowan, 1979); the delusion persists chronically, with the elaboration of secondary themes.

Controversy still centres on the place of this condition in psychiatric nosology, due in part to the poor descriptions of premorbid history, personality, and phenomenology. The DSM-III restricts the delusions of the paranoid disorders to persecution and jealousy, making it difficult to assign a place to erotomania.

Erotic delusions, including a wide range of sexually related beliefs (sexual pursuit by an enamoured party, jealousy, impending marriage, pregnancy, rape, and venereal disease), are common, occurring in one third of women with psychosis (Rudden *et al*, 1978).

Ellis & Mellsop (1985) discussed the content of 58 cases of de Clérambault's syndrome with regard to conformity to the presence of the fundamental postulate and its derivatives, finding it variable. They concluded that the syndrome did not merit a separate nosologic category, and that most cases could be classified as having schizophrenia or related disorders. However, a mental disorder cannot be characterised by its content or cross-sectional picture alone, and attention must be paid to its form, the disturbance of mood, course, outcome, and response to treatment.

We present two cases of erotomania in women with bipolar affective disorder arising or persisting during periods of euthymia.

Case histories

Case 1

Ms A is a 48-year-old right-handed single female whose father was alcoholic. Of her three siblings, one brother is alcoholic, with paranoid traits; a female first cousin has bipolar affective disorder.

Although her family was not particularly devout, the patient joined a Carmelite convent at age 17, which she was forced to leave two years later because of bizarre behaviour and sexual preoccupations.

First psychiatric contact was for depression upon leaving the order. The patient worked, mostly as a private-duty nurse, until 1979, when she went on disability pension. Her first admission to hospital occurred at age 26, with the diagnosis of schizophrenia, and she had six subsequent admissions, all of which averaged 3–4 months and were treated with neuroleptics. However, the patient showed clear episodes of mania and depression interspersed with periods of euthymia.

The first admission was precipitated by an erotic delusion concerning a physician. Similar delusions involved an orthodontist for seven years and a doctor in sociology over ten years. For the past seven years her delusions involved a hospital staff member. She sent letters, gifts, and pictures to him, while attributing letters soliciting intercourse to him; the ward staff were seen as trying to thwart the expression of their love for each other. These beliefs were maintained during periods of euthymia, although they intensified considerably and led to action during hypomania. .

The mainstay of treatment had been neuroleptics until 1976, when lithium carbonate was started, although chlorpromazine 50–100 mg was used for sedation. The affective episodes were partially controlled by lithium but she was only infrequently compliant; serum level never exceeded 0.75 mmol/litre.

A trial of carbamazepine 200 mg t.i.d. achieved a serum level of 25 μmol/litre (therapeutic range 17–50 μmol/litre). When it was increased by 200 mg, the patient's affective state normalised and she renounced her delusions completely. However, she grew less adherent to the treatment regime and further blood levels could not be obtained, with an admission to hospital ensuing soon thereafter.

Case 2

Ms B is a 64-year-old single woman who has had a number of admissions due to depression and elation. Her first psychiatric illness was at 26 when she had a mild, quickly remitting depressive episode. After a suicide attempt at age 45, she was admitted to hospital and treated with ECT, with a relapse the following year. At age 59 she had a one-week episode of hypomania and thought that she was the reincarnation of Christ. Following this episode she was maintained on small amounts of perphenazine, which she has refused to reduce despite lack of psychotic symptoms.

During her latest admission, she had a severe retarded suicidal depression. Clomipramine produced a rapid switch in mood, with elation and racing thoughts. She agreed to a trial of ECT, which with perphenazine left her less depressed but still delusional, with some fluctuation between states. She achieved a euthymic state with no evidence of delusions on the combination of lithium and perphenazine, re-establishing her social and work life quickly.

Six months later and the day after her regular appointment, she called her psychiatrist to say that she was disappointed because she had waited at home expecting his visit. She knew her feelings of love were reciprocated but appreciated that he could not openly profess his feelings because of the professional relationship. The delivery of many letters and cards expressing her affection soon followed. Ms B interpreted his continued refusal to visit as proof of his love for her and thought she could determine his thoughts. She has continued to send letters at regular intervals and to allude to their relationship.

She has been very compliant with her medication and has remained euthymic, with no evidence of delusional thought in other areas of her life. She had no previous episodes of delusional erotomania.

Discussion

Several psychological explanations for erotomania have been proposed. Self-love, denied or projected on to another person, or as a displacement from unconscious homoerotic trends similar to those in pathological jealousy; struggles with unsatisfied affection; and the wish for rebellion against social roles have been proposed as causative. The syndrome has also been viewed as serving an adaptive function, providing an externalised source of nurturance, protection, and control during periods of strain to mask depression and loneliness (Enoch & Trethowan, 1979).

Hollender & Callahan (1975) divided erotic delusions into two subtypes: a primary or pure form, with sudden onset, fixing upon a single object and developing into a chronic encapsulated system (paranoia); and a secondary one, with gradual onset, superimposed on other previous symptoms (schizophrenia). Although there is little formal consideration, the literature supports a predominant role for severe affective disorder rather than schizophrenia or paranoid disorder in producing this delusional symptom; only Guirguis (1981) and Remington & Book (1984) clearly report their cases as having bipolar affective disorders. Hypersexual behaviour or promiscuity is not uncommon. Erotic delusions, appearing prior to the index episode or as recurrences, are frequent, as well as waxing and waning of the presenting delusion.

In terms of disturbances which may be included under affective disorders, recognised and often unrecognised bipolar affective disorder, "grandiosity", secondary mania, borderline personality disorder, "hysterical psychosis", and numerous cases of depression can be found.

A more precise recording of phenomenology may permit valuable clinical distinctions to be made. After separating changes in libido, particularly hypersexuality often found with hypomania and mania, several categories of erotic delusions (erotomania) can be described. These are:

(a) persecution (e.g., erotic pursuit, influence, and control)
(b) ideas concerning the conduct of oneself and/or others (e.g., jealousy, homosexual reference)
(c) delusional relationship (e.g., de Clérambault's, "incubus", "phantom lover")
(d) somatic delusions (e.g., rape, pregnancy, venereal disease, metamorphosis).

Erotomania has been believed to be relatively resistant to psychotherapy and to pharmacotherapy, with chronicity the rule (Enoch & Trethowan, 1979). However, a review of the few reports that record treatments does not bear out this gloomy opinion. Cocchi *et al* (1982) reported the "anti-erotic" effect of chlordesmethyldiazepam, a GABA-ergic agent, on five patients, of whom one was depressed and three (including Case 4) probably had bipolar disorder. Responses to lithium (Remington & Book, 1984), ECT, and antidepressants and neuroleptics (Guirguis, 1981), as well as previous response to ECT, provide further evidence in favour of a role for severe affective disorder.

Conclusion

Erotic delusions are common in a number of forms of affective disorder, particularly in the bipolar spectrum. As well, patients with paranoia, especially those with grandiosity or affective features, may present with erotomania; these may represent mixed states, unrecognised affective disorder, or mood-congruent delusions which persist into the euthymic state. The mood changes, or waxing and waning of the intensity of the beliefs may be falsely ascribed to the vicissitudes of the content of the delusion, psychodynamic changes, or psychosocial stressors.

Only with appropriate trials of medication, considering the possible origins of these delusions in affective disorder, can the importance of this category of delusional content be understood.

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A Case of Self-Inflicted Leucotomy

A suicide attempt with a gun resulted in left frontal lobe damage in a 19-year-old obsessive man. While intelligence did not suffer and no frontal lobe syndrome emerged, the patient's obsessive rituals were significantly reduced.

The advance of medicine is frequently furthered by natural experiments, as this report of unintended psychosurgery, in a patient with severe obsessive compulsive disorder (OCD), vividly illustrates.

Case report

On 11 November 1983, Tom, 19 years old, was found with blood running down his face. He had shot himself through the mouth with a .22 calibre rifle. Shortly thereafter, he was admitted to neurosurgery.

A CT scan revealed blood in both maxillary sinuses. The bullet had penetrated through the ethmoid sinus and the cribriform plate, and penetrated to the base of the left frontal lobe. The bullet was lodged in the premotor cortex, and fragments could be seen along the left-side pathway through the head of the caudate nucleus and splenum of the corpus callosum. The patient manifested a mild right-sided hemiplegia, involving the leg more than the arm.

The onset of his OCD followed shortly after he became aware of his homosexuality at age 10 (father accepted it but mother was very upset). Obsessive ideas of contamination, and minor checking and hand-washing rituals did not interfere with his life, including his studies, until age 15. He was then admitted to hospital for OCD, and treated with a combination of exposure and response prevention and supportive psychotherapy, but without benefit. At age 16 he first presented to one of the authors (LS), suffering from compulsive doubt, checking, hand-washing, and shower-taking. He was also depressed, feeling hopeless, lacking interest and pleasure in almost everything, feeling weak and tired, oversleeping, and blaming himself for not being able to continue his studies or go to work. Formerly an 'A' student, his grades had deteriorated and he had left school. When psycho-, behaviour, and pharmacotherapy (clomipramine to 200 mg daily) effected no change in OCD or mood, he left treatment.

During the next two years he made two suicide attempts, thinking that if he sacrificed his life, he would expiate God, his father would drink less, and his mother would be more loving and kind. He then returned to treatment. His depression was more severe, and in addition to the earlier OCD symptoms, he had to repeat ideas in order to gain perfection, and had to recall 50 different chants.

He was placed on phenelzine 15 mg t.i.d. Three weeks later, he became frightened at his inability to ejaculate. His mother threatened him with commital to a mental institution, and when he complained to her about how wretched he felt, she replied: "Go and shoot yourself." Five minutes later he did so.

Three weeks after neurosurgery, he underwent neuropsychological testing and an initial assessment of OCD. The same obsessions as before the suicide attempt were present, but at a much lower frequency and intensity. His bathroom rituals had decreased from four hours pre-injury to half an hour.

During the next two months he remained in hospital on the psychiatric ward, where he received behaviour (exposure and response prevention) and drug therapy (phenelzine to 75 mg daily). His compulsive hand-washing, toilet ritual and checking were gradually reduced to a daily average of 15 minutes. Because he remained depressed, he was placed on carbamazepine 400 mg b.i.d. After two weeks, he rated himself as "mostly cheerful". He was discharged, and followed up at monthly intervals. He continued on carbamazepine for one year but did not receive further behaviour therapy.

At two years post-discharge, his mood remained consistently calm and cheerful. He completed his high school education. His obsessions were minimal – he remained intolerant of disorder and demanded that the kitchen and bathroom be kept spotless.

Neurological examination was normal except for anosmia. A CT scan revealed that part of the left frontal lobe was infarcted. There was no sign of social disinhibition, tactlessness, or lack of drive – no indication of frontal-lobe syndrome.

On neuropsychological measures, he showed impairment of right-handed fine motor co-ordination, and right-hand performance in the Finger Tapping test (Reitan & Davison, 1974); impairment was shown also on the Controlled Oral Word Association Test (Benton, 1968), which is particularly sensitive to left frontal damage (Crockett *et al*, 1986). Performance on the Wisconsin Card Sorting Test was unimpaired. Patients with right frontal lobe lesions make more errors on this task (Drewe, 1974).

There was marked improvement in intellectual functioning. Scores for IQ on the Wechsler Adult Intelligence Scale before (1980) and after (February 1984) the suicide attempt and at two-year follow-up (February 1986) were

verbal: 115, 110, 121
performance: 117, 120, 140
full scale: 116, 114, 133.

There was also marked improvement on psychometric tests of obsessions (self-rating), fears and phobias (Fear Survey Schedule), anxiety (Institute for Personality and Ability Testing Anxiety Scale), and introversion and neuroticism (Maudsley Personality Inventory). At the three testing points (before, after and follow-up) his Leyton Obsessive Inventory scores were

symptom: 32, 21, 12 ($P<0.01$)
resistance: 38, 32, 13 ($P<0.05$)
interference: 51, 24, 11 ($P<0.01$)

and Beck Depression Inventory scores were -32, 6 and 5 ($P<0.05$).

Discussion

It seems likely that the observed changes were not just coincidental to the self-inflicted psychosurgery. The rate of spontaneous remission or, at least, improvement in OCD patients is estimated at 40% (Rachman & Hodgson, 1980) to 70% (Pollitt, 1957). The most favourable estimates include cases of recent onset (e.g. three-month duration for adult onset), and show a slower time course. Black (1974), however, summarising four major reports, found a static course in 54–61%, and progressive worsening in 33%. In the case described here, early onset and a chronic, progressively worsening course, the dramatic and almost immediate effects of unilateral leucotomy, and the sustained improvement over two years rule out spontaneous remission.

There is a striking similarity between the anatomic and psychological changes of our patient and those reported after psychosurgery of the fronto-orbital cortex and anterior cingulum. Kelly (1973) cites dramatic changes in mixed depressive and obsessional states, the depressive symptoms being relieved by the frontal lesion, and the obsessive symptoms by the cingulectomy, with intelligence, learning, drive level, and control unaffected.

The neuropsychological and power spectral findings of Flor-Henry *et al* (1979) suggest predominantly left frontal function in OCD, and this is consistent in the present case with the site of the lesion.

The issue of concomitant factors should be raised. Several factors may have influenced the OCD in addition to the anatomical change. The drama of the suicide attempt, with its attendant notion of 'hitting bottom'; the subsequent stay in hospital, necessitating removal from a noxious home environment; perhaps the mother's changed behaviour, being somewhat more regretful than earlier, may all have been instrumental in bringing about the observed improvement. However, previous suicide attempts, periods in hospital, and removal from the home had not improved his condition. In a fashion similar to that achieved by psychosurgery, the self-inflicted left frontal damage altered the course of OCD. Hence we conclude that in this case of self-inflicted neurosurgery, the change in brain anatomy led to the psychological change – a change that seems to be permanent.

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Rudolph Hess's Mental Illness

SIR: The recent suicide of Rudolph Hess, aged 91, in Spandau Prison recalls the spirited controversy about his correct psychiatric diagnosis (Dicks *et al*, 1947; Slater, 1972). When Hess parachuted into Scotland on 10 May, 1941, he was Deputy Führer and a close friend of Hitler. Hess claimed he made the jump in order to make contact with the Duke of Hamilton, who would take him to King George; together they would arrange to stop the war. The immediate impetus for Hess's flight to Scotland, according to his own account, was a dream by a friend whom he believed was a prophet. German radio attributed Hess's flight to "an illness of many years' standing. . .mental derangement" and added that "the National Socialist Party regrets that this idealist fell a victim to his hallucinations."

Almost immediately Hess was noted by his captors to be depressed and to be expressing beliefs about a plot to poison him. Within one month of his landing he was examined by psychiatrists – Brigadier-General J. R. Rees and H. V. Dicks. The latter spoke fluent German. Rees diagnosed Hess as suffering from "a psychopathic personality of the schizophrenic type", while Dicks' immediate impression was "typical schizophrenic"; Dicks later diagnosed "paranoid psychosis". In subsequent months Hess was noted to have auditory hallucinations, much evidence of delusional thinking, and odd behaviour "such as lying with his fingers in his ears smiling to himself". He also made two suicide attempts, fracturing a femur in a leap shortly after his arrival in 1941 and stabbing himself in the chest in 1945. Hess's clinical picture was complicated, however, by intermittent amnesia "which varied with circumstances and with the observer, and at times suggested malingering rather than hysteria".

On 10 October, 1945, Hess was taken to Nuremberg to stand trial for war crimes; the question was whether he was mentally competent to stand trial. A Rorschach protocol indicated "the possibility of a psychotic episode". However, initial psychiatric examination by Major Kelley concluded that Hess had "a severe psychoneurosis of the hysterical type". Hess was therefore examined by American, British, Russian, and French physicians. The Americans (D. Ewan Cameron, Nolan D. C. Lewis, and Paul L. Schroeder) concluded that Hess "is suffering from hysteria characterised in part by loss of memory" and "is not insane at the present time in the strict sense of the word". The British (Lord Moran, J. P. Rees, and George Riddoch) diagnosed Hess as "a psychopathic personality" with "a marked hysterical tendency" and declared that "He is not insane in the strict sense". The Russians (Professors Krasnushkin, Sepp, and Kurshakov) labeled Hess's illness "a psychogenic paranoid reaction" with "hysterical amnesia", and said "there are no changes typical of the progressive schizophrenic diseases". The French (Professor Jean Delay) participated in both the American and Russian examinations and tried to conduct a narcoanalysis of Hess, but he refused. Hess was allowed to stand trial, was found quilty and was sentenced to life imprisonment.

Slater (1972) published a little-known but sharply-worded critique of the psychiatric conclusions, and especially of the part played by J. P. Rees. Slater claimed that, "the conclusion is irresistible that Rudolph Hess had a schizophrenic psychosis. . . The psychotic process came in a series of acute attacks. . . It is very difficult to explain the incapacity or unwillingness on the part of the psychiatrist [*sic*] to reach this diagnosis." Slater continued, "If the diagnosis of schizophrenia is correct, then there would seem to have been serious deficiencies in the clinical judgment or the open-mindedness of a number of distinguished world psychiatrists . . .The diagnostic blindness, the failure to reach an obvious conclusion becomes a problem in its own right, demanding explanation."

Rees, according to Slater, was the key to the error. Rees's understanding of "psychopathological phenomena on exclusively psychodynamic lines" and his contention "that diagnosis is a bagatelle" led to "the results of grave misconceptions of the task of the psychiatrists of what constitutes psychiatric knowledge, and of the function of psychiatry as a medical discipline *vis-à-vis* the individual patient". Rees, as the only psychiatrist on the British examining team and as the only person present with extensive involvement with the case, was in a position to influence the others. This, in Slater's opinion, "led to a trial which seems now to have been a miscarriage of justice".

In his 1972 article Slater called for the release of the subsequent psychiatric records on Hess when he died, to settle the diagnostic issues. From 1946 until his recent death, over 41 years of such records have accumulated. What effect, for example, did Hess's psychiatric status have on the decision not to release him from Spandau? If Hess was psychotic, was he treated during those 41 years? What effect did press reports that Hess might be malingering have on the decision to try him at Nuremberg? Since Hess was a Deputy Führer whose flight to the West was deeply embarrassing to Hitler, did political considerations enter into the diagnostic decision to not label him as insane? If Hess was psychotic, what was his mental status as Deputy Führer when he and Hitler were formulating the policies which led to the war? It is also interesting to contrast the case of Hess, apparently psychotic but found fit to stand trial, with the simultaneous case in the United States of Ezra Pound, who was not psychotic but claimed to be unfit to stand trial on 19 counts of treason (Torrey, 1983).

In the interest of history, the psychiatric records of Rudolph Hess should be made available for professional examination.

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Expressed Emotion and First Episodes of Schizophrenia

Sir: The debate about the validity of the Northwick Park Study in the papers by Mintz *et al* and Macmillan *et al* (*Journal*, September 1987, **151**, 314–323) highlights a number of important issues about the EE concept. The level of discrimination between high and low EE in predicting relapse among first-episode schizophrenics, whether significant or not, is certainly lower than in previous studies of EE in undifferentiated samples. In fact, Leff & Brown (*Journal*, April 1977, **130**, 417) have previously revealed that the nine-month relapse rates for high EE first admissions is 38% (compared with 13% for low EE) and 69% (compared with 17%) for readmissions. If high/low EE are stable characteristics, why should such temporal changes in predictive power be observed? The possibility that EE may in fact be an unstable characteristic is revealed in the recent large-scale study of Hogarty *et al* (1986): 25% of the high EE sample not exposed to family treatment 'spontaneously' reduced to low EE, compared with 39% who reduced under family treatment. In both groups no relapse was observed, suggesting perhaps that the spontaneous reduction in high EE was genuine and not an artefact of measurement. It seems that there is an important developmental dimension in the genesis of stable EE which is concerned with the cumulative interaction of inherent schizophrenic vulnerabilities and the family system (Birchwood & Smith, 1987).

Macmillan *et al* discuss the applicability of EE: only one-third of their sample come from a family home, and of these only a minority were high EE. Our experience is roughly similar, although we have observed that many first admissions return to the family home subsequently; McCreadie & Robinson (*Journal*, May 1987, **150**, 640–644) found that 50% of their undifferentiated sample lived with a relative. It is not at all clear to us that low EE families cope in a 'model' way; in some cases we have observed that families manage the burden of living with schizophrenia in such a way as to exacerbate other clinical or social features, such as social withdrawal (Birchwood & Smith, 1987). The prelevance of high EE does not therefore adequately reflect the therapeutic or service implications of family interventions.

Finally, Macmillan *et al* draw attention to the comments of Hogarty *et al* (1986) that family interventions delay rather than prevent relapse and that the success of these interventions might be inflated by drug defaulters among the controls. It is important to recall that many have argued that neuroleptics themselves serve to delay relapse (Englehart *et al*, 1967) and that, to quote Macmillan *et al* (*Journal*, February 1986, **148**, 128–133), "relapse rates were lower on... medication than placebo, *but however assessed, outcome at this early stage was poor for many patients*" (our italics).

Clearly, we have no cause for complacency, and both environmental and biological research needs to be actively encouraged and pursued.

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A Comparative Trial of Amitriptyline and Fluoxetine

Sir: The comparative trial of amitriptyline and fluoxetine in depressed out-patients by Young *et al* (*Journal*, September 1987, **151**, 337–340) found no significant difference in outcome except for a slight difference, discounted by the authors, on the Beck Scale for Depression (BSD). Improvement in BSD scores commenced in the first week, and six-week improvement is described as moderate. I may not be alone in being baffled by the conclusions drawn from these results: "Efficacy was similar in the two groups"; "Overall both drugs proved to be effective antidepressants over the six-week trial..."; "The apparent efficacy of fluoxetine..."; "... both drugs proved equally efficacious"; "... the absolute usefulness of both drugs can only be assumed".

With the possible exception of the last of these statements, none of them (*pace* the pharmaceutical industry) is warranted by the evidence presented. Perhaps the most convincing result was that amitriptyline gave patients dry mouths and made them fat; fluoxetine made them sick. This seems a good example of the "me-too" variety of drug trial. A placebo group was omitted on ethical grounds. Similar grounds might have been invoked for the omission of the conclusions.

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Tuberous Sclerosis and Psychosis

Sir: Dennis & Hunt (*Journal*, September 1987, **150**, 413–414) have recently drawn attention to two case reports in which tuberous sclerosis in associated with psychotic symptomatology (Lawlor & Maurer, *Journal*, March 1987, **150**, 396–397; Clarke *et al*, *Journal*, May 1987, **150**, 702–703). In addition, they refer to their own study of ninety children with tuberous sclerosis, over half of whom demonstrated psychotic behaviours (predominantly autistic or hyperkinetic) (Hunt & Dennis, 1987). They propose that profound language delay, severe impairment of social interactions, hyperkinesis and sleep disturbance constitute a behavioural phenotype. They believe that this occurs so frequently as to be included within the diagnostic criteria for tuberous sclerosis.

Within the field of mental handicap, there are few specific organic disorders which are associated with specific psychiatric disorders. Exceptions might include organic mental states in Down's syndrome, self-mutilation in Lesch-Nyhan and Cornelia de Lange syndromes, autistic-like symptomatology in Rett's syndrome, and perhaps a few others.

The problem of associating tuberous sclerosis with autistic symptoms is of interest, but difficult, as it raises our uncertainty over the aetiology of autism itself, although this is presumably the result of some organic lesion. The association really resolves itself around two questions: is tuberous sclerosis *directly* associated with autistic symptoms? Or does it frequently give rise to degrees of mental handicap, which are themselves frequently associated with autistic symptoms?

The authors need to show that, having controlled for degrees of mental handicap, patients with tuberous sclerosis are more likely to develop autistic symptoms than other groups of brain-damaged individuals. Unless this is done, the suggestion that autistic symptoms are specifically applicable to individuals with tuberous sclerosis, any more than to patients with, say, hydrocephalus, may be entirely spurious.

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Comparative Trial of a New Antidepressant

SIR: Few would argue with the point raised by Dunn (*Journal*, August 1987, **151**, 269) and Thompson (*Journal*, November 1987, **151**, 702–703) that a comparison of a new antidepressant with a placebo to establish that some efficacy exists is desirable. Clearly, ethical issues are a problem, and there is an extensive literature highlighting these (Klerman, 1986; Rickels, 1986). Practical issues are also a factor, and in a current clinical trial involving placebo control an eminent colleague withdrew his involvement because he felt that every patient should receive an active compound.

Studies previously carried out comparing fluoxetine, a tricyclic antidepressant, and a placebo (Stark & Hardison, 1985; Colin & Wilcox, 1985) confirmed that fluoxetine was more effective than placebo.

My study (Levine *et al, Journal*, May 1987, **150**, 653–655) was carried out to establish further evidence of efficacy and to compare the occurrence of side-effects with those of imipramine. The numbers involved allowed for any significant difference to be discriminated. A minimum score on the HRSD of 17 permitted less severe cases to be included. The suggestion by Thompson that 75 mg of a tricyclic antidepressant is "well accepted to be inadequate" is surely an idiosyncratic view, and is at variance with established practice over almost 30 years. Perhaps the as yet unpublished paper quoted in his letter will inform us otherwise.

The need for new antidepressants clearly exists when present compounds are only marginally more effective than placebo at any dose level, and future research will hopefully not be influenced by the "overcrowded market".

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Depressed Mood After Stroke

SIR: For Wade *et al* (*Journal*, August 1987, **151**, 200–205) to compare their finding that 20% of their stroke patients were definitely depressed and 10% probably depressed with Bergmann's (1982) reported rate of 4.4% for depressive illness and neurosis in 360 elderly women is of doubtful relevance. Reported rates of depression in community studies of the elderly vary enormously, and one could equally well argue on the basis of comparison with the work of Zung (1967) or Stenback (1979) that stroke protects against depression, as both these studies can be interpreted as demonstrating depression in well over 40% of elderly subjects. It is possible to quote studies which purport to show rates of depression in old age which vary from 48% right down to 1%. None of them help to interpret the findings of Wade *et al*, whose study is flawed by the lack of an age-matched control group, and the use of an instrument which was not developed in order to measure depression in stroke victims with an mean age of 70 but to measure the severity of the depressive syndrome in a group of patients already diagnosed as depressed whose mean age was in the mid-forties (Snaith *et al*, 1971).

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ZUNG, W. (1967) Depression in the normal aged. *Psychosomatics*, **8**, 287–292.

SIR: We thank Ames for stressing that the Wakefield Self-Assessment Depression Inventory was not developed for use with elderly people, and for pointing out that we did not have an age-matched control population. We discussed both points in the original paper, although we acknowledge that we did not refer to the population studies he mentioned.

The study was under several contraints. In particular, we were not aware of any simple measure of depression which could be used in a large number of elderly people. Indeed, we still do not know of any such index. There was not time within the constraints of this study to identify and assess a similar group of control patients. This study is purely descriptive, and generates many hypotheses needing further investigation. We sincerely hope that the two major weak links identified by Ames will be investigated in future.

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Long-Term Psychiatric Patients in the Community

Sir: The debate concerning the wisdom of continuing psychiatric hospital closures and the advantages of community care ultimately rests on the evidence, which at present is scanty. Bell *et al* (*Journal,* August 1987, **151,** 275) have investigated this issue. The interpretation of their data is really a question of one's outlook, but whether one chooses to describe a cup as half empty or half full, it certainly will not be running over.

Out of 25 patients who had been selected for discharge over an 11-year period, and who had been in hospital for at least two years, we are advised that "half" had lost contact with psychiatric services, one was in prison and four were dead. It is true that the dead had all been "at an advanced age", but one might question the wisdom of relocation in these circumstances. Of the remainder, we are advised that five had been readmitted to hospital, one indefinitely, and that when interviewed, in a semi-structured way, "a roughly equal" proportion of the remaining population who were still in touch with psychiatric services were leading "somewhat discontented lives". The authors place a generally optimistic interpretation on this data.

We compared two well-supported out-patient groups with long-stay hospitalised patients and found that the in-patient groups had a better quality of life on a number of measures. This was despite the fact that in one of the hospitalised groups the patients were significantly older than the comparison out-patient group.

Two groups of in-patients, at St Mary's Hospital, Stannington, Northumberland ($n=40$) and Friern Hospital, in North London ($n=23$), were interviewed, together with out-patients from both hospitals ($n=40$, St Mary's; $n=16$, Friern). All the out-patients were actively supported by community nurses. The interviews were conducted according to a prepared schedule that had been piloted, modified, and discussed with the nurses prior to use. The nurses, including community psychiatric nurses, held a variety of views on the intended transition to community care.

The Friern in-patients were significantly older than the out-patients ($U=66.5$, $P<0.001$) (in-patients, range=37–73 years, mean=56.5, s.d.=11.2; out-patients, range=20–64 years, mean=40.9, s.d.=13.77) but were carefully matched for age and sex in the St Mary's groups (in-patients, range=23–77 years, mean=51.3, s.d.=15.14; out-patients, range=24–73, mean=50.4, s.d.=13.86).

In all, we enquired into 20 indicators of quality of life. Hospitalised patients were significantly advantaged (Mann-Whitney, two-tailed probabilities) in terms of attending social gatherings ($U=550$, St Mary's; $U=107.5$, Friern; $P<0.05$, both groups), going to places of entertainment ($U=242$, $P<0.001$, St Mary's), the number of meals per week ($U=24$, $P<0.001$, Friern), the number of hot meals ($U=154$, $P<0.001$, St Mary's; $U=26.5$, $P<0.001$, Friern), and the number of baths per week ($U=481.5$, $P<0.005$, St Mary's). As might be expected, the in-patients were also advantaged in the amount of time they spent in rehabilitation programmes ($U=260$, $P<0.001$, St Mary's), and in passive amusements ($U=585$, $P<0.05$, St Mary's; $U=103$, $P<0.05$, Friern, watching TV; $U=479$, $P<0.005$, listening to the radio at St Mary's). However, if contacts with nursing, medical, and paramedical staff are disregarded, the Friern in-patients enjoyed less company ($U=275$, $P<0.01$), which might reflect their advanced age, and they attentively listened less to the radio ($U=287.5$, $P<0.005$). In other respects the groups were comparable.

Our study, which we hope to report more fully, causes us to wonder whether the intended closure of the two hospitals will be entirely advantageous to all our patients. We are incompletely reassured by the findings of Bell *et al* in a selected population.

We thank the nursing staff at St Mary's and Friern Hospitals for their help in data collection.

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Pseudo-AIDS, AIDS Panic or AIDS Phobia?

SIR: Miller *et al* (*Journal,* May 1985, **146,** 550–551) reported on two men with excessive concern about AIDS: one had a diagnosis of anxiety neurosis, and the other had a diagnosis of depressive illness. The authors argued that this was a new psychiatric condition arising from the AIDS epidemic and coined the term 'pseudo-AIDS'.

Since the publication of this article, similar conditions have been described and called in turn 'AIDS-induced psychogenic state' (O'Brien & Hassanyeh, *Journal,* July 1985, **146,** 91); 'AIDS phobia' (Jacob *et al, Journal,* March 1987, **150,** 412); or 'AIDS panic' (Windgassen & Soni, *Journal,* July 1987, **151,** 126–127).

All of these definitions seem to place AIDS in a causative role in creating such conditions. We join O'Brien (*Journal,* July 1987, **151,** 127) in stating that what is important in patients presenting with excessive concern about AIDS, but without the disease, is not AIDS itself, but the underlying psychiatric state.

The common feature of the psychiatric illness, with AIDS concern as a symptom, is hypochondriasis. In hypochondriasis, the illness of which the sufferer has fearful apprehension is usually inherently frightening and often stigmatised by society, classical examples being syphilis and cancer. In addition, the sufferer is often someone who has already been brought into psychological contact with the disease, e.g. by a relative, friend, or even health workers. The fear of AIDS meets all of these criteria (Thompson & Riccio, 1987). Indeed, Miller (1987) has stated that such patients are similar to those with worries about other venereal diseases.

Fear of AIDS may also be a part of the content of any psychopathology, such as obsessional disorders (Goldmeier, 1987), affective disorders (Lippert, 1986), schizophrenia (Rapport, 1985), or monosymptomatic hypochondriacal delusional states (Lippert, 1986). In such cases of severe psychiatric disorders, the fact that AIDS is the content or object of the psychopathology is of little significance in the diagnosis or treatment of the disorder, which should be carried out in the usual way (Thompson & Riccio, 1987).

Finally, it can be argued that the term 'AIDS phobia' is misleading. The modern definition of phobia requires the presence not only of irrational fear of a harmless stimulus, but also avoidance of that stimulus. Thus, to be genuinely AIDS phobic, the patient would be required to irrationally avoid AIDS – almost a paradox, except when there is avoidance of AIDS even as a thought or a topic of conversation. The term 'AIDS phobia' is therefore a misnomer, since the patients to whom the term is most applied, i.e. the 'worried well', show little or no tendency to irrationally avoid AIDS.

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Neuroleptic Malignant Syndrome (NMS): A Misnomer?

SIR: Kellam's review (*Journal,* June 1987, **150,** 752–759) is apt. Perhaps it is time for the nomenclature of so-called NMS to be revised. Besides its possible occurrence before the advent of neuroleptics, it is now well reported that non-neuroleptics are also implicated, e.g. tetrabenazine (Burke *et al,* 1981), levo-dopa (Henderson & Wooten, 1981), and dothiepine (Grant, 1984). One case of NMS was attributed to metoclopramide combined with cimetidine (Destee *et al,* 1981), another was seen in an overdose of benzodiazepines, phenelzine, dothiepin, and alcohol (Ritchie, 1983), and yet another was attributed to amphetamines (Chayasirisobhan *et al,* 1983). The current restricted nomenclature, using the word neuroleptic, is not an accurate reflection of current facts and predisposes those not familiar with the literature to a sense of false security.

As most cases appear to occur in the context of treatment procedures, "Iatrogenic malignant syndrome" might be more appropriate, but we would guess that this would prove unpopular, particularly in the context of explaining the condition to patients, their relatives, or possibly their legal advisers.

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Sir: Kellam (*Journal*, June 1987, **150**, 752–759) has included Stauder's lethal catatonia on inadequate grounds, quoting Mann *et al* (1986), who consider NMS to be a neuroleptic-induced iatrogenic form of lethal catatonia. A similar opinion has also been expressed by Lindesay (1986), who perceived NMS as a hybrid of iatrogenic disorder and mis-diagnosed lethal catatonia. Lindesay suggested that lethal catatonia represents an idiopathic form of the disorder, whereas NMS may represent an iatrogenic form.

While not rejecting these opinions outright, it has to be kept in mind that the so-called lethal catatonia has only face validity as a nosological entity. There have been no well-planned prospective studies on its descriptive validity, construct validity, or predictive validity. Whatever information is available at the moment on lethal catatonia is a compilation of anecdotal reports such as the reviews by Mann *et al* (1986) and Kellam. Moreover, lethal catatonia is not mentioned in the current diagnostic and classification systems (DSM-III, ICD–9), and most psychiatrists no longer diagnose Stauder's lethal catatonia. Indeed, the term 'catatonia' is obsolete and confusing and should be elimintPed from psychiatric terminology (*Lancet*, 1986), despite recent attempts to rehabilitate it (Barnes *et al*, 1986; Mann *et al*, 1986).

A superficial clinical resemblance between an iatrogenic syndrome and an entity of historical importance should not be grounds to contest the nosological status of NMS.

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Sir: I agree with Singh & Maguire. Possibly if a new name for the syndrome is required it should be simply descriptive, as the aetiology remains obscure. I would suggest 'pyrexial catatonia'.

I am grateful to Adityan Jee for directing me to the invaluable article by Barnes *et al.* I would agree that 'catatonia', and especially its 'acute lethal' sub-type, only have face validity as clinical entities. I am not sure that any of the ICD–9 or DSM–III categories have much more. If we were to restrict ourselves to those categories whose validity had been demonstrated by well-planned prospective studies on their descriptive validity, construct validity, and predictive validity we should have few diagnostic entities left to use, either clinically or for further study.

My impression remains that a syndrome marked by rigidity and abnormalities of movement (catatonia) has been observed by several generations of psychiatrists, probably since they started to record their observations systematically. It was most often associated with other symptoms of what we now call schizophrenia, but has become rare since the advent of neuroleptic drugs. Occasionally it was associated with a fulminating course and death in hyperpyrexia. If this syndrome were to occur now it would usually do so in a patient already on neuroleptics, which would account for the Parkinsonian features now commonly seen and thus be indistinguishable from the neuroleptic malignant syndrome. I am therefore prepared to question whether neuroleptics or the other well-documented changes in dopaminergic drive, such as stopping L-dopa, are always to blame for the syndrome. I am currently enquiring of Barnes *et al* whether their idiopathic cases were pyrexial, in the hope that they may have recorded neuroleptic malignant syndrome in a drug-free patient.

The syndrome is probably stuck with its current name. My thesis is that we need to remind ourselves that we are not certain of either the implied aetiology or outcome. Vigorous treatment is to be encouraged in view of the reduction in mortality which can apparently be expected.

Recent work by Addonizio *et al* (1986) suggested the presence of a partial NMS in eight patients, as well as the full syndrome in two patients, out of a series of 82. All showed pyrexia and extra-pyramidal rigidity or tremor, and in the partial cases the symptoms remitted without the neuroleptics being stopped. I hope to repeat this work on a larger series to see if a mild pyrexia is common in patients receiving neuroleptics and showing extra-pyramidal side-effects.

Finally, I should like to retract my statement that neuroleptic malignant syndrome is not mentioned in any British textbook (see O'Shea & Falvey, 1985).

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Edinburgh Post-natal Depression Scale

SIR: Cox *et al* (*Journal*, June 1987, **150**, 782–786) have produced a concise instrument for detecting post-natal depression. I would question the inclusion of question seven in its present format. Probably the majority of new mothers experience sleepless nights for many weeks, because neonates generally take some time learning to sleep through the night. For such people, it might prove difficult to answer this question, which implies that unhappiness is the only cause for their insomnia. Rephrasing the question as follows might help:

"I have been so unhappy that I have had difficulty sleeping, even when my baby has been quiet and the opportunity for sleep was there...".

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SIR: Vincenti has drawn attention to the central dilemma of the validity of established self-report mood questionnaires when administered to pregnant or puerperal women and to our attempt to overcome these difficulties.

The main problem which was discussed in our paper (*Journal*, June 1987, **150**, 782–786) is that certain somatic symptoms, such as weight loss or palpitations, may be caused by the physiological changes of childbearing as well as by a mood disturbance. The wording of the questionnaire items may also need to take into account the particular social circumstances of the mother and the incessant demands that may be made on her. The Edinburgh Post-natal Depression Scale (EPDS) was developed to reduce these ambiguities as far as possible.

Because many mothers experience sleepless nights for many weeks after childbirth, the sleep item (item 7), "I have been so unhappy that I have had difficulty sleeping", was worded to detect those mothers whose sleep difficulty was secondary to a mood disturbance and not caused *directly* by a noisy baby or by a restless partner.

The correlation matrix (available on request) between the 10 items on the EPDS confirmed that we had been successful in this endeavour. The sleep item had its highest correlations ($r = 0.52$) with item 3 ("I have blamed myself unnecessarily when things went wrong") and item 8 ("I have been feeling sad or miserable"), which would indicate that this item is detecting women whose sleep difficulties relate to their depression.

The modification suggested by Vincenti is, in our opinion, unlikely to be an improvement and might cause yet further problems; for example, what *are* the requirements for "the opportunity to sleep" – a comfortable bed, or perhaps a quiet partner?

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Predictions of Outcome in Depressive Illness

SIR: Eagles (*Journal*, May 1987, **150**, 715) appears to claim that we were unjustified in assuming that patients presenting for admission to hospital on clinical grounds were more likely to be depressed than those who remained in the community (*Journal*, January 1987, **150**, 43–48). We would like to hear the evidence supporting his rather idiosyncratic view.

We will answer Eagles' points seriatim. He observes that the sample was not "normally distributed" on the Newcastle Scale. However, elsewhere in the article, we clearly showed that there was evidence of discontinuity in the distribution.

He questioned the representative nature of the sample. However, we made no claim that the sample was representative of anything other than severely depressed patients. We made this clear on p. 46, second column: "we were probably not looking at a random sample of the general population, sources of potential bias being the mode of referral to hospital and the criteria of selection for the trials". Moreover, this was confirmed by the use of the Newcastle Scale worked out for use with similar in-patients.

We accept that there is a distinction between treatment response and outcome, and this may become important in the remoter outcome period. However, in the short term the two are virtually identical. Differences might arise in the longer term, and outcome measures could provide further criteria for determining whether there is a binary distribution of outcome or, whatever the follow-up measures employed, this had a unimodal distribution. However, this was outside the modest remit of our own study, although it could be looked at in the future. Thus the inference drawn by Eagles that "these findings provide no convincing evidence for the dualist theory of the classification of depressive illness' is clearly not supported by the evidence and is therefore inappropriate.

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The Hungerford Massacre and its Aftermath

Sir: The above hypereredic* episode has received world-wide publicity. The unanticipated consequences of such attention seem worthy of closer observation, as does the possibility of prevention in some instances of this type of explosive behaviour.

During the 1950s and 1960s I examined hypereredic individuals categorised in this area as 'amok runners'. They came from among peoples of three grades of technological development: the Melanesians of Papua New Guinea, the Chinese in Singapore, and the Europeans resident in Australasia (Burton-Bradley, 1968, 1972). Since that time further instances have surfaced in this area at the rate of approximately one every 18 months. Although culturally variable, there is a hard-core symptomatology in all of them, based on the self-defined unrelieved insult. There are four discrete periods: (a) a prodromal brooding, during which a resolution may be effected if circumstances permit; (b) explosive homicidal outburst; (c) continuing homicidal drive; and (d) a retrospective claim of amnesia by the ones who survive.

Case Reports: (i) A 35-year-old Caucasian public servant had received no promotion for 10 years. He wrote me a letter in 1967 outlining his claim of differential treatment. He stated that he had been continually overlooked, that colleagues with the same or lesser qualifications had been promoted, and that he had been railroaded into a dead-end position. He declared his empathy with, and total acceptance of, the solution adopted by a laboratory technician in remote Pennsylvania, USA, who had recently ran amok killing six people and wounding six others. This had been reported in great detail in the weekly newspaper. The victims were people in authority over him. I asked the public servant to come and see me, and during the course of interview I detected oversensitivity coupled with anxiety and anger. I formed the opinion that he had a diminished capacity to select and control alternative forms of behaviour. There was, I felt, a serious risk that he would act out the Pennsylvania model. I conferred with his superiors, and it transpired that his promotion was in fact being processed, but at a slow bureaucratic rate. This was speeded up, and the would-be amok runner informed, with dramatic effect in eliminating his brooding. When examined again two years later he was free of all symptoms.

(ii) Another Caucasian amok runner, who was killed during the period of his episode, left behind a letter showing how he had modelled his plans on a similar tragedy which took place in remote Canada one month earlier. He had read about it in the newspaper. He had been rated by his victims as a "humbug". He acted out the Canadian model.

These brief observations show that the physician, close acquaintance, or other person in contact with an adult male (usually living alone) who outlines his disaffections in the above fashion should be alerted. The proposed ban on certain classes of firearms, admirable as it may well be, is based on a response to public expectation and not necessarily on fact. It will do little to eliminate such episodes, and gives the impression of seeking a solution merely by closing the door after the horse has bolted. Also, it is unlikely that much help will, or could be, obtained from the media, but such bodies should be adequately informed. But is psychiatric expertise being fully utilised?

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Hypereredism: a morbid state of hostile tension, following provocation, sometimes leading to explosive behaviour out of proportion to the circumstances.

Behaviour Disorder in Childhood Re-assessed

Sir: We have completed a pilot study of one year's intake of 207 children as new out-patients to a hospital-based department of child and adolescent psychiatry. At follow-up, 16 months after the intake began, there were 67 cases closed; these were evaluated by two clinicians, mutually blind to each other's assessments. The assessments were made from recordings of the parents' overall description of their children's present state. Inter-observer disagreement occurred in 5 out of the 46 cases assessed by both.

The interesting result was that response to intervention was greatest with behaviour disorder (76%) and least with neurotic disorder (57%). This is opposite to the generally held view, supported by numerous studies (West & Farrington, 1977; Mulligan *et al*, 1963; Richman *et al*, 1982), that behaviour disorder is difficult to treat and has a poorer prognosis than childhood neurosis (Rutter, 1972; Kovaks *et al*, 1984).

Perhaps the introduction of a family therapy approach (which always involves the father in our clinic) changes the prognosis compared with more traditional therapeutic approaches. A three-year follow-up of all 207 cases will be completed shortly

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Indecent Exposure – A Report of a Female

SIR: Indecent exposure is a rare problem in women (O'Connor, 1987), and the few reported cases are generally associated with factors such as hypomanic episodes or alcohol use. We report an ex-nun with a history of inappropriate sexualisation as a young child, referred because of genital exposure.

Case Report: A 36-year-old sales assistant was referred because of feeling depressed and a six-year history of genital exposure, usually to authority figures: for example, the mother superior while living in a convent, and the priest running a therapy centre. She had also exposed in order to get attention when feeling miserable and lonely. The exposure was planned; she would enjoy a response of shock and anger and lie on the floor kicking her legs, talking in a babyish fashion, and hoping to be smacked. She would also make repeated telephone calls, and haunt the front door of her victims (usually women).

The patient's mother had no time for talking, playing, or cuddling with her four children (her husband was a violent man and a heavy drinker), and would masturbate her children to comfort them if they were upset; their only bodily contact was smacking. Her younger brother was impotent as an adult, and our patient had never had a sexual relationship, but enjoyed looking at other women's bodies.

We aimed to help the patient to make friends and to relate normally in a therapeutic community of patients with mainly long-term neuroses, while continuing at work. She managed to keep this job, but made no friends and continued to expose to senior nursing and medical staff.

This report illustrates the lasting damage of inappropriate sexualisation in a young child (Mzarek & Kempe, 1981). She had no experience of affectionate non-sexual handling, and attached erotic sensations to non-sexual infantile behaviour and so regressed in this way. This was sexual abuse from stressed parents who did not derive sexual gratification themselves but were in need of help with their family. Early intervention in such families is very important (Werner, 1985).

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Mania Following Head Injury

SIR: We read the report by Clarke & Davison, (*Journal*, June 1987, **150**, 841–844) and would like to report another example.

Case Report: A 38-year-old self-employed married businessman was admitted in May 1987 for investigation and assessment in respect of abnormal behaviour following severe head injury.

His personality type was of the overactive, ambitious, and aggressive type, with an above-average IQ – the epitome of the Type A personality. He ran two businesses simultaneously, and his hobby was competitive cycling. There was no past history or family history of psychiatric illness.

In December 1986 he fell from his push-bike and was found unconscious in the road. He showed signs of a right sub-dural haematoma, and was transferred to a neurosurgical department where CT scan confirmed this. An emergency craniotomy and evacuation of extra-dural and sub-dural clot was performed. At operation, laceration of the right temporal lobe was noted. His post-operative course was stormy – he was unconscious for several days, and then required sedation for restlessness and aggression. He was treated with phenytoin and phenobarbitone. He subsequently made a reasonable recovery physically, with only a persistent right-sided facial nerve palsy and hemianopia.

However, on his return home (three weeks after the injury) he developed markedly abnormal behaviour, with over-activity, outbursts of unprovoked aggression, disinhibition, extreme emotional lability, grandiosity, and irritability. The extent of these symptoms severely threatened his marriage and his business. At this time he refused tranquillisers or psychiatric treatment.

Some weeks later he accepted psychiatric assessment. On admission he presented as an intelligent man, mildly elated in mood, garrulous, and obsessed with his physical fitness. He had the fixed idea that his problems were entirely due to an abnormal glycogen metabolism which he could cure himself by strict dieting. He was physically overactive, but co-operative.

His EEG showed "abnormal asymmetrical activity and a persistent excess of slow activity over the right anterior to mid temporal region". CT brain scan was abnormal, showing "contusion and ischaemia at the right temporal and parietal regions and severe right temporal atrophy". Psychometric testing showed strong evidence of impairment of functioning at the anterior portion of the right temporal lobe (Rey Osterrieth) and also of the frontal lobe (verbal fluency and behaviour on the Wisconsin). It is likely that his frontal lobe pathology is *contre coup* injury, and the right temporal and parietal signs correlate well with this head injury.

He is now back at work, his mood has improved and stabilised, and he has developed some insight, although he continues to believe that his psychological problems have been due entirely to problems of glycogen metabolism. His wife says he is still "overdoing it", but he is no longer frankly hypomanic.

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Acute Psychotic Episode Caused by the Abuse of Phensedyl

Sir: Following the recent publications concerning ephedrine abuse and ephedrine psychosis (Whitehouse & Duncan, *Journal*, February 1987, **150**, 258–261; Jelley, *Journal*, September 1987, **151**, 418–419), we report a case of phensedyl abuse precipitating an acute psychotic episode.

Case Report: A 49-year-old asthmatic married woman was admitted to our psychiatric unit as an emergency in an agitated and excitable state. She was talking non-stop with some incoherence, was visually hallucinating, said that she was seeing "white spots", and was suspected of hearing voices. She thought that thoughts were being put into her mind and "made her do things", and was disoriented in time, getting the day, month, and year wrong, but was oriented to person and place. She had to be sedated because of her excitability, especially at night, and the psychotic symptoms and disorientation disappeared 48 hours later, at which point the medication was discontinued. Interview 24 hours later confirmed the visual hallucinations and thought insertion, but not the auditory hallucinations. In addition, she described experiencing something like thought broadcasting and passivity feelings. The patient was able to remember most of the period of her delirium with very minor gaps in detail. She stayed in hospital for the next five days and remained symptom-free with no medication.

Her history of phensedyl abuse dated back about 10 years. She remained vague about the amount she consumed, but on checking with her husband it appeared that she had been drinking 3–4 bottles per week. However, the week before her admission she had consumed a larger amount than usual. She also had a history of alcohol abuse, but no other psychiatric history.

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Compensation Psychosis

Sir: In describing a case of compensation psychosis, White *et al* (*Journal*, May 1987, **150**, 692–694) highlight a topic of growing importance. We report another case of psychosis in the context of compensation, also complicated by bereavement.

Case Report: Our patient was a 36-year-old plumber. Two years previously his wife had died unexpectedly while in hospital, leaving him with three young children. He soon began legal proceedings against the Health Authority concerned, and appeared unable to mourn his wife's death. Fifteen months later his solicitor sought a psychiatric opinion because of anxiety about his mental health. He presented with a two-month history of depressive symptoms with prominent biological features.

His mother killed herself when he was aged 7, and his elder brother had also committed suicide three years previously. A second brother and his cousin were both victims of homicide. The patient had suffered a major depressive illness three years before, following his brothers death, and this had responded to out-patient treatment with antidepressants. His premorbid personality was extroverted.

On examination he was severely depressed, perplexed, and held the delusional belief that he, and not the hospital, was responsible for his wife's death. He also believed that psychiatrists knew this and were in league with his wife's family to punish him. He was admitted and treated with amitriptyline and chlorpromazine. Suicidal ideas and impulses emerged, and he required intensive nursing for a month. Over the next six months his illness followed a protracted and fluctuating course. Although no longer severely depressed, he still believes that he will not be well until the Health Authority is successfully sued, thus absolving him of all blame.

We think he has had a morbid grief reaction complicated by a psychotic depression in which compensation could be a maintaining factor. We are not aware of any reports of such cases in the UK literature, but this may become an increasingly common phenomenon in the wake of tragedies such as Bradford and Zeebrugge. Our patient, like many survivors of those tragedies, has suffered multiple losses and is also involved in a compensation claim.

Rosenblatt (1983) suggests that lawyers involved with this client group should be aware of their special needs, and that "the recurrent review of the loss brought about by involvement in a suit may disrupt the normal detaching process, thus leading to a morbid grief reaction". Litigation may also be an increasingly fashionable style of response to such losses. While it is tempting to speculate further, there remains little systematic evidence on which to base important clinical decisions about management. This is an area which merits further study.

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Consent to Investigation

SIR: The philosophy of the Mental Health Act 1983 was to "strengthen the rights and safeguard the liberties of the mentally disordered" (Bluglass, 1984). This ethos emerged in the innovative consent-to-treatment area of the Act. However, the relevant sections are specific in nature, and thus situations could arise which are not dealt with by the Act. The following case report illustrates such a problem in organising investigations.

Case report: A 47-year-old housewife was admitted under Section 2 of the Mental Health Act 1983, suffering from her first psychiatric illness of an episode of typical agitated depression. After a four-week trial of amitriptyline and chlorpromazine there was no improvement, and ECT was prescribed. The patient refused consent to this treatment, and so Section 3 was applied. The Mental Health Commissioner agreed to a course of ECT, as the patient was unable to eat or drink. Two months later there was a little improvement, but a request for a second course of ECT was declined by the Commissioner on the grounds that even though the patient remained ill the situation was no longer life-threatening.

Throughout the hospital stay the patient refused investigations. Consequently, even though she was not responding to medication or ECT, an organic cause for her illness could not be excluded because of her non-cooperation. Enquiries were made to discover whether investigations could be performed without consent. The Mental Health Commission stated that the issue was not covered by the Act and a medical defence organisation advised us not to proceed, as so doing would probably constitute a battery.

Half-way through the duration of the patient's treatment under Section 3 the Commissioner returned to decide if the patient could be given medication against her consent. A treatment plan was provided suggesting a trial of lithium, in view of the failure to respond to antidepressants alone. However, if approved, the legality of forcing investigations to monitor serum lithium was not known. Further correspondence with a medical defence organisation revealed that they too were unsure. Fortunately, the Commissioner resolved the problem by agreeing to the treatment plan and to the investigation of the patient. Necessary blood tests were therefore taken, and an EEG was performed. The latter was reported as normal, and the patient responded well to the trial of lithium.

The requirement to consult Mental Health Commissioners in order to plan the treatment of detained patients is appropriate to safeguard their liberty. However, investigations are an important component of the management, but as they are not dealt with specifically by the Act they are covered by common law, which permits procedures only to be performed against or without consent if they are life-saving. Investigations are rarely life-saving, and so necessitate consent.

The importance of investigations are highlighted by the above patient, whose unresponsiveness to treatment may have been due to an organic cause. If this had been so, then the four-month delay before the investigations were carried out could have been detrimental. Investigation for an organic cause for mental illness is important to allow appropriate management. Just as patients do not understand or consent to treatment, they may also misunderstand or be frightened by investigations.

It is surprising that this point regarding investigations did not occur during the drafting of the Act or subsequently, but this patient and the difficulties and delays encountered exemplify the need for clarification on the point. Possibly the issue of investigations could be dealt with in the same manner as treatment presently is under the Act, and be administered by the nominated Commissioners.

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Reference

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CORRIGENDUM

Journal, August 1987, **151**, 160–165 (Wig *et al*). The names of Jørgen Achton Nielsen and Grethe Thestrup, from the Aarhus Psychiatric Hospital, Risskov, Denmark, should be added to the list of authors.

A HUNDRED YEARS AGO

Lunacy regulations in France

In consequence of certain irregularities revealed by the investigations into the case of Baron Seillières, who, it was alleged, was improperly detained in a lunatic asylum near Paris after he recovered his mental equilibrium, the Minister of the Interior, M. Fallières, has issued a circular to all prefects directing them rigidly to obey the enactment which requires them to make periodical visits to all asylums, public or private, within their jurisdiction. It is directed that these visits should be paid unexpectedly, and not less frequently than once a quarter; that patients who desire to leave the asylum should be interviewed, and in every doubtful case subsequently subjected to a special medical inquiry; that the use of the shower-bath as a punishment should be forbidden; that the period of observation should be reduced to the shortest possible time; and that frequent transfers from one establishment to another should be discouraged.

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Book Reviews

Editor: Sidney Crown

Psychology of the Self and the Treatment of Narcissism. By Richard D. Chessick. Northvale, NJ: Jason Aronson. 1985. 367 pp. $35.00.

'The psychology of the self' is the name now given to the theories and the approach to psychotherapy developed by Heinz Kohut and contained for the most part in his two books, *The Psychology of the Self* (1971) and *The Restoration of the Self* (1979). They have had an enormous influence on psychotherapists in the United States and to some extent elsewhere, and are clearly something all therapists should know about. It is not easy to read Kohut himself, and I have never been sure if it was worth taking the time and the trouble of getting to understand what he is talking about. This book will help readers to acquaint themselves with his ideas and form a personal judgement of them.

I have certainly found it fascinating to recognise how Kohut became preoccupied with narcissism and concepts of the self, which are the same issues which interest contemporary British psychoanalysts. His approach is totally different, however, and in its final version represents a turning away from psychoanalytic work.

Chessick seems to be experienced, not only as a psychiatrist and psychotherapist, but also as a philosopher. The bibliography lists 37 publications since his first in 1969, and no less than eight of these have been books. His chief preoccupation is with narcissistic disorders and borderline states, but his interests are wide and his erudition comes through clearly.

The book begins with an introduction to the concept of narcissism, via the Greek myth of Narcissus, and then quickly proceeds to review the psychoanalytic developments in this area, beginning with Freud. A fairly detailed account of Melanie Klein's views follows, and although Chessick is generally out of sympathy with Kleinian ideas he acknowledges them to be important and indeed suggests that they provide an alternative approach to some of the same issues which have occupied Kohut.

The largest and most important section of the book is the exposition of Kohut's theories, which is complicated by the fact that they changed as his ideas evolved. Chessick makes this relatively easy to follow by providing chapters on the first version, on Kohut in transition, and on the second version of the psychology of the self. These are preceeded by a discussion of the definition of the self and by a short account of the role of the self in the work of Fairbairn, Winnicott, Balint, and Laing.

It is not possible to give an account of Kohut's ideas in this short review, but the interested reader will be able to find out what Kohut means by a number of terms which are central to the understanding of his theories. Thus 'Narcissistic transference', 'empathy', 'selfobject', 'the grandiose self', 'the idealised parental image', 'the bipolar self', 'the varieties of mirror transference', 'narcissistic rage', and many other terms are clearly described and their importance discussed.

Kohut suggests that Freud presented us with a model of *guilty man*, so that psychoanalysis became preoccupied with conflict between love and hate and its consequences. The analyst becomes the representative of the patient's conscience, and instead of helping him, persecutes him. Kohut presents his model of the individual as *tragic man*. In his view the central issue facing the patient is the way he reacts to his failure to realise his ambitions and ideals. The cause of mental disorder is primarily put down to the failure of the parents to empathically understand and encourage the child's narcissism, and the therapist attempts to put this right by providing such understanding.

While most psychoanalysts consider that omnipotence, narcissism, and self-idealisation are defensive manoevres to deal with deeply disturbing experiences, Kohut emphasises the normal need for positive affirmation, which he considers leads to a proper sense of a cohesive self. He relates this to the glimmer in the mother's eye as she watches her baby with approval and admiration, and he teaches the therapist to allow similar idealising transferences to develop, in which the patient feels he can stimulate admiration and excitement in the therapist.

Further sections of this book present clinical material discussing some of Kohut's patients as well as those of the author, and these help the reader to apply the concepts clinically. There are also discussions of diagnosis and nosology, and a comparison with French views of the self as presented by Satre, Lacan, and Foucault. A special chapter on criticisms of Kohut is also useful, and although the author is clearly impressed with his work he is quite capable of discussing the problems it raises.

This is a very helpful book and a model of good exposition. Whether the reader will want to go on and study Kohut will depend on his approach to psychotherapy. Even those who prefer a psychoanalytic understanding will, however, find it useful and will be led to reconsider some of their own views as a result.

John Steiner, *Consultant Psychotherapist, Tavistock Clinic*

Drugs and Sexual Function: A Pharmacological Approach. By M. A. DAVIES and A. D'MELLO. Harpenden: Ridge Publications. 1986. 66 pp. £4.00.

This short booklet is intended to be read by a large group, including doctors, pharmacists, pharmacologists, research workers, medical teachers, and students. It is unlikely that any text could appeal to all of these equally, and I suspect that it will be read mostly by students, although they may well be put off by the price. The authors are extremely modest and do not indicate their place of work or qualifications. They give a good account of the physiology of sexual function in the first third of the book, followed by an account of the ways in which drugs can affect functioning in different ways. This is useful because it emphasises that it is not always easy to interpret the effects of drugs on sexual function. The final part of the book outlines the actual effects of drugs on sexual function, and this is summarised in tabular form at the end of the book. Much of the information was obtained from a computer search of the literature between 1979 and 1984 and is reasonably up to date.

I found the text easy to follow, although its style was a little journalistic. The clear diagrams aid understanding, and some of them are very good. There is little doubt that the authors have identified an important problem, and one which is not dealt with particularly well elsewhere. I hope the book will succeed in reaching a wide audience, and it is a pity that they have not been able to persuade their publishers, who appear to be new to the medical field, to market the book at a lower price to aid sales.

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Suicide in Adolescence. Edited by R. F. W. DIEKSTRA and K. HAWTON. Dordrecht, The Netherlands: Kluwer Academic Publishers. 1987. 196 pp. Dfl. 100.00, $43.50, £27.95.

This is a valuable book which will disabuse anyone who believes suicide is the prerogative of the elderly. The authors, each an authority on suicide in adolescence, have provided ten chapters organised into five themes: the epidemiology of suicide in the young; personal factors associated with the phenomenon (by means of an illuminating case history); the principles of assessment and treatment; the inter-relationship between their families and the young who kill themselves; and schemes for prevention.

There is a good balance, in terms of length and style, among the chapters, and each author writes lucidly and with authority. They are careful to differentiate between what are their own personal opinions and what are more widely established empirical findings.

The only disappointing features of the book are certain lapses in presentation. A few of the figures are a challenge to the eye. An incorrect figure has been presented in the text, although the publishers have provided the correct one in their 'Errata'. In the latter they have also corrected two typographical errors; unfortunately, there are others which seem to have gone undetected. There is also an unwelcome lack of correspondence on occasions between the references mentioned in the text and those in the reference lists. It is a pity about these matters, because generally the book is well presented.

In summary, this book will be useful for clinicians and researchers. It highlights an important and (sadly) a growing problem. The reader is not only challenged to take the matter seriously, but given useful information and practical advice about how to deal with this problem.

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The Uses of Countertransference. By MICHAEL GORKIN. Northvale, NJ: Jason Aronson. 1987. 312 pp. $27.50.

Gorkin is a psychologist and practising analyst, and his book comes very much from within the psychoanalytic tradition. He traces the development of the idea of countertransference from Freud's rather sketchy description onwards. In passing, he produces the clearest explanation I have seen of that most tricky concept, projective identification. The body of the book explores the nature of the countertransference experiences which can arise in a variety of clinical circumstances, and discusses whether and how such feelings should be made use of in therapy.

The writing is clear, very full references are given, and differences and inconsistencies in the work of the various authors are discussed. The conclusions drawn suffer from the common difficulty with psychoanalytic writings that they are not systematically researched (and are perhaps unresearchable). However, we all have to struggle with the feelings our patients induce in us, and even the most dedicated opponent of analysis could learn from the direct way in which Gorkin describes such feelings.

This book deserves to be widely read and should certainly appear on the shelves of psychiatric libraries.

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Mental Illness in Primary Care Settings. Edited by MICHAEL SHEPHERD, GREG WILKINSON and PAUL WILLIAMS. London: Tavistock. 1986. 296 pp. £19.95.

This book documents the proceedings of a conference held at the Institute of Psychiatry in 1984, the principal objective of which was to evaluate and learn from the link between research and policy in this field. The coverage is wide, ranging from classification, screening, and treatment differences on the one hand to professional team roles and patterns of collaboration on the other. In addition, there are a number of short contributions based on experience and aspirations in other countries which, by and large, fail to relate relevant insights to the English scene. It is, however, the omissions that stand out. There is an absence of any substantial discussion of primary prevention in this apparently ideal setting, perhaps emphasising how little of practical value is known about the aetiology of psychiatric conditions. Balint, too, is mentioned but once in passing, and interviewing skills hardly at all.

Given the primary care setting, the book adopts a prior assumption of the medical model and deviates little from this. Much emphasis is given to the team approach, but little empirical evidence is presented on what is actually done by whom and, in particular, the precise skills exhibited by the various professionals. This is epitomised by the fact that one contributor asserts that the most important aspect is that a person is on the spot at the right time. Indeed, in the book there is debate about whether collaboration within or between teams depends essentially on personalities, the discriminating characteristics of which are not identified. Except when considering drug treatment effectiveness, this book does not help to differentiate the specific from non-specific factors in this field.

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Parental Influences: In Health and Disease. Edited by James E. Anthony and George H. Pollock. Edinburgh: Churchill Livingstone. 1986. 531 pp. £25.70.

This book, edited by two distinguished psychoanalysts, consists of a series of psychoanalytically-orientated papers on the clinical aspects of ordinary and disturbed parenting. Topics include cross-generational influences, the parental experience, pathological parenting, and specific situations such as violence and psychosomatic disease. Much reference is made throughout to the works of Anna Freud, Winnicott, and Therese Benedek. It has the usual strengths and weaknesses of psychoanalytic writings – vivid and detailed case descriptions, but a turgid and often incomprehensible style of writing with an irritating lack of objectivity and criticism. This latter is particularly notable in the chapter by Sackin, ironically entitled "The parents of children with psychosomatic diseases: a critical review of the literature". Sackin criticises authors who espouse ideas with which he disagrees for reaching conclusions not adequately supported by their data. In contrast, he quotes in support of his arguments scores of authors, most of whom are subject to the same criticism, but the criticism is not forthcoming.

The psychoanalytically inclined will enjoy this book. With the exception of Wallerstein's excellent chapter on divorce, the rest of us would do well to look elsewhere if we are to learn more about parenting. Are we still expected to take seriously such statements as "the reason why such mothers (post-partum psychosis) are unable to recathect the baby after birth is because they are narcissistic and too fearful that 'a declaration of ownership of the baby' as a stolen penis would draw the anger and retaliation of those who regarded it as their own"?

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The Adolescent in Group and Family Therapy (2nd edn). Edited by Max Sugar. Chicago: The University of Chicago Press. 1975, 1986. 298 pp. £12.50.

This edition is a reprint, some ten years on, of the first, with an additional chapter by the editor on transference in adolescent group psychotherapy.

Each chapter describes a way of handling problems presented by adolescents according to the experience of the authors, whose expertise lies primarily within a psychoanalytical framework. The problems whose therapy is discussed range from those that present as personal problems to those seen as community based – e.g. school or ghetto problems – or related to membership of a problem family or to drug addiction.

For those to whom this book is unfamiliar, it contains timeless information about the adolescent process and the tasks of adolescence, which are presented, for the most part, in the form of clear descriptions of adolescent behaviour. Although the observations are usually presented from a psychoanalytical perspective, they are generally written in a way that would interest and inform those who do not embrace a psychoanalytical approach. The book can be recommended, provided it is read in the knowledge that the principles underlying both group and family therapy are subject to developmental change.

Dorothy H. Heard, *Consultant Psychotherapist, University of Leeds*

'Till Death Do Us Part: How Couples Stay Together. By Jeanette C. Lauer and Robert H. Lauer. New York: The Haworth Press. 1987. 192 pp. $24.95.

This book is based on questionnaire surveys of 351 marriages, some fulfilling, others not. The authors concentrate on what constitutes a successful marriage, and examine aspects such as commitment, individuality versus togetherness, conflict styles, intimacy, effective communication, "the meaning and course of true love", and managing change. Among issues raised are whether happiness really is the complete satisfaction of instinctual needs, and whether cohabitation is an adequate preparation for marriage. They conclude that in America, after experimentation with promiscuity, "marriage is back".

Research results include quotations from couples, and each section concludes with sentimental, often platitudinous, truisms such as, "Troubled as well as gratifying years may mark the course of a long-term satisfying marriage". Sub-sections called "Action guidelines" profer advice and suggestions on how to sustain a happy marriage. Much of this is schmaltz, apparently substantiated by research, presented in an ingenuous, moralising, 'Bible belt' style, using everyday language, and addressed to a "wide variety of professionals".

JAMES ATKINSON, *Consultant Psychotherapist, Cambridge*

Foundations of Contextual Therapy: Collected Papers of Ivan Boszormenyi-Nagy. By IVAN BOSZORMENYI-NAGY. New York: Brunner-Mazel. 1987. 337 pp. $35.00.

These papers, covering a period of 30 years, reflect the developing thought of one of the most original thinkers regarding the family context of mental illness.

Boszormenyi-Nagy's approach to psychotherapy is based on his appreciation of what he sees as the absolute existential reality of peoples' responsibilities and obligations to one another, especially within the family. The necessity of respecting these obligations, which the author refers to as 'relational ethics', is absolute; it cannot be ignored or postponed indefinitely without a heavy price in mental health. The individual is embedded in relationships, and, in particular, in a family matrix – the narcissist who attempts to deny this interdependence will be unfulfilled and may become ill. Arguing that a purely individualistic approach to therapy, or indeed to life itself, may be ultimately disastrous, Boszormenyi-Nagy encourages the therapist to take account of all persons involved in relations with the patient.

In working with marriages or families, the contextual therapist addresses the 'ethical' issues of fairness, trustworthiness, and entitlement. The assumption seems to be that people do have an intuitive sense of justice and fairness in personal transactions. 'Entitlement' is earned through the caring one gives to another. When this principle of justice is violated, the individual and his or her relationships become disturbed. The therapist helps all parties involved to reassess what each may expect of the other and also attempts to locate disguised forms of caring that are going on. The author talks also about 'destructive entitlement', a lack of remorse over destructive or exploitative behaviour, which results from being deprived or exploited. This is one of the main features described by Kernberg as characteristic of narcissistic disorders; interestingly, Boszormenyi-Nagy comments: "This freedom from remorse is not a psychological distortion. Tragically, it was actually earned through having been exploited. It is in consequence of a destructive reality". However, I am left wondering to what extent *unjustified* feelings of entitlement may be stoked up within certain personalities and grievances exploited by destructive parts of the mind – the book does not address this possibility.

The necessity for concern with posterity is emphasised. It is argued that it is only through a concern with future generations that one's legacy of caring received from one's parents can be repaid. The message is that an uncaring, exploitive attitude towards the world and its future is devastating to our mental health – a view which reminded me strongly of those of the psychoanalyst Donald Meltzer, expressed, for example, in his *Sexual States of Mind.*

Boszormenyi-Nagy sees his work as offering a perspective on therapeutic approaches, rather than a complete method in itself. His own earlier background was psychoanalytic, and indeed he regards an understanding of individual psychodynamics as essential. There are many aspects of mental life and relationships which these papers do not address – anxiety, for example, a basic focus of the analytic therapist, is not even listed in the index. While initially irritated by the prevalence of neologisms and unnecessarily dense writing in this collection, I was left feeling that my understanding of therapy and of human relationships had been enriched.

PHIL MOLLON, *Regional Psychologist in Psychotherapy (Top Grade), Dryden Road Hospital, Gateshead*

Therapeutic Trances: The Cooperation Principle in Ericksonian Hypnotherapy. By STEPHEN G. GILLIGAN. New York: Brunner/Mazel. 1987. 380 pp. $51.00.

Gilligan first reviews various approaches to hypnotherapy, clearly favouring the 'co-operative', or Ericksonian, approach. The basic premise is that therapy is a joint endeavour between therapist and client. Identification and use of the client's resources, rather than exploration of the past, are also central features. Symptoms may be used as the basis for trance induction.

The author reviews various theories of hypnotic trance and some of its characteristics. He then outlines the general approach of the Ericksonian hypnotherapist, and goes on to describe the 'co-operation strategies' whereby the therapist establishes rapport and prepares clients for the induction of trance. The remainder of the book describes various means of inducing trance and using it for therapeutic purposes. Gilligan quotes many clinical examples, and provides a full transcript of an induction. There is an interesting discussion of 'confusion' techniques for inducing trance.

This is a valuable account of how we may use Erickson's work in our psychotherapy. The newcomer to hypnotherapy will probably find it an insufficient guide to how to proceed, but as a supplement to practical instruction it should be invaluable. The more experienced hypnotherapist will find much that is stimulating and innovative. It is a valuable addition to the Ericksonian literature, if a little pricey.

PHILIP BARKER, *Professor of Psychiatry, University of Calgary, Canada*

Psychiatry in Primary Health Care. By S. SHARMA. Ranchi, India: Central Institute of Psychiatry. 1986. 150 pp.

It is increasingly recognised that untreated psychiatric illness constitutes a significant drain on the resources of developing countries. However, such is the magnitude of the problem that the specialised psychiatric services are quite inadequate to cope with that which is not already absorbed by traditional or religious healers. This textbook, aimed at the primary health care physician, is particularly written from an Indian perspective, although the content would be relevant in most developing countries. There is a concise but traditional exposition of the major illnesses, with some special chapters, for example on psychosocial aspects of fertility control, and some useful material on drugs, record keeping, and the organisation of mental health services. There is frequent use of flowcharts, and these are generally clear and easy to use. There is no index, which is a slight drawback. This is a useful benchbook for primary physicians in developing countries, but readers might be well advised to replace the plastic cover provided, which melted in my car in the relative cool of the current English summer.

STEPHEN W. BROWN, *Consultant, The David Lewis Centre for Epilepsy, Cheshire*

Making Contact: Uses of Language in Psychotherapy. By LESTON HAVENS. London: Harvard University Press. 1986. 201 pp. £15.75.

Havens, who is Professor of Psychiatry at the Cambridge Hospital, Harvard Medical School, discusses how he uses language in psychotherapy. His concern is to describe ways to be accurately empathic with patients. He is concerned to integrate descriptive psychiatry with psychoanalysis, social psychiatry, and existential psychiatry. He begins his book by first looking at the problem of establishing empathic contact with persons who for one reason or another are hidden from themselves and the therapist, and in consequence absent from the consultation. From this he formulates an empathic language to reach such persons, by means of various categories of statement: imitative, simple empathic, or complex empathic. He devotes much of the rest of the book to exploring issues of how to use language in the management of such persons.

He emerges as a warm and sensitive clinician whose preoccupations with the psychotherapy of more disturbed personalities have led him away from interpretation to a rather more active conversation. It is hard to know how transference issues can be dealt with in such a way of handling the patient.

Reading through his various categories of empathic statements, it seems as though he is offering the reader a series of prescriptions of language for the psychotherapeutic encounter. I wonder if empathic language can really be prescribed in this way? Surely it is something that can only be arrived at from the privacy of the psychotherapist's own experience of living and from the mutuality of each new therapeutic encounter with the patient.

P. J. SHOENBERG, *Consultant Psychotherapist, University College Hospital, London*

Psychosocial Treatment of Schizophrenia: Multidimensional Concepts, Psychological, Family, and Self-help Perspectives. Edited by JOHN S. STRAUSS, WOLFGANG BÖKER and HANS O. BRENNER. Ontario: Hans Huber. 1987. 220 pp. US$39.00.

Books based on the proceedings of conferences tend to be unsatisfactory for a variety of reasons, but this one is an exception because it contains several papers of unusual interest. In particular, Wing, Zubin, and Ciompi all contribute major pieces with important theoretical statements; all provide tabulated or numbered lists of propositions which could be valuable for teaching.

Wing specifies three conceptual levels underlying long-term management, as well as four categories of environmental influences on schizophrenic syndromes: the course of the illness, he points out, depends much on the social environment, and often requires walking a tightrope between too much and too little stimulation. However, factors that can affect the various manifestations of the disorder need to be translated into hypotheses about methods of care which can then be tested – a process that has scarcely yet begun. At the level of management he emphasises that this cannot be done successfully in the long term unless services are geographically responsible, comprehensive, and integrated. Relevant to the present Gadarene rush to extra-hospital facilities is his comment that "sheltered communities can have all the characteristics of open care, while a terraced house in an ordinary street can be like a small and isolated prison".

The veteran Zubin is determined to be optimistic and to reject chronic disability as being inherent in many cases, although this seems to strain the limits of acceptance. His vulnerability model regards the patient as being essentially well, although subject to one or more episodes of illness; the natural healing process may be impeded to varying degrees by "ecogenic forces", but enhanced by good social networks, ecological niches, or premorbid personality – which may mitigate the impact of stresses. Zubin says that outcome studies must use controls which are concordant for all important psychosocial variables, but it is far from clear how this could be done.

Ciompi lists no less than eight integrative models, and maintains that chronicity depends more on psychosocial than on genetic/biological factors, although the case for this is not strongly argued. He also proposes a three-phase model to establish connections between vulnerability and information-processing, based on Piagetian theory. It is elegantly argued, but largely translates well-known concepts into different terminology. There are also nine therapeutic principles for the psychosocial management of schizophrenia, including the suggestion that psychosocial measures may act as alternatives to medication, but the work of Leff and Hogarty does not support that view.

Other papers deal particularly with social skills training and with self-help by schizophrenics. The contributions which were originally in German show a number of translation problems, but overall this book stands out through its unusual quality.

HUGH FREEMAN, *Consultant Psychiatrist,*
Hope Hospital, Salford

A Primer of Human Behavioural Pharmacology. By ALAN POLING. New York: Plenum. 1986. 246 pp. $29.50.

The author, in his preface, writes that this book is intended to introduce principles of behavioural pharmacology to readers with little or no knowledge of the discipline but with an interest in how drugs affect human behaviour, but it is hard to know for whom the book is intended. The book is written in a lively style with each new concept being clearly defined, but once a reader's interest has been stimulated few references to more specialised textbooks are given (and where listed are, in at least one case, an out-of-date edition). The coverage of topics is varied. The section on basic pharmacology is very basic, while the chapter on clinical drug assessment touches on many topics, including the DSM–III diagnosis of schizophrenia and methods of measuring behavioural change by direct observation. Although the author graphically illustrates the measurement error inherent in different methods of observing behaviour, this can be of limited value to people embarking on clinical drug assessments when few references to methodology have been included.

This book is recommended as a stimulating introduction to the subject, but should not be regarded as a basic textbook in behavioural pharmacology, in spite of its title.

ELIZABETH GOODALL, *Research Fellow, Medical College of St Bartholomew's Hospital, London*

A Textbook of Clinical Neurophysiology. Edited by A. M. HALLIDAY, S. R. BUTLER and R. PAUL. Chichester: John Wiley. 1987. 730 pp. £32.50.

This book comprises a series of didactic lectures given at the International Congress of Electroencephalography and Clinical Neurophysiology, held in London in 1985. Distinguished clinicians and scientists were invited to lecture on specially chosen topics, so that the series formed a complete course covering all three branches of clinical neurophysiology: electroencephalography, evoked potentials, and electromyography. The speakers were asked to emphasise their views on clinical practice rather than research in progress, and texts based on those lectures have been published here. The result is a remarkable book in that it contains the views of so many international experts under one title. However, as might be expected of a volume compiled in this way, the chapters are very varied, particularly in the level of complexity addressed. They range from a section on signal averaging with instructing equations to a chapter describing the most elementary principles of nerve conduction studies. However, each contains interesting and useful information, as would be expected from this authorship.

The book has been produced and published promptly and is inexpensive (possibly to the slight detriment of one or two of the illustrations). As stated in the preface, it cannot really be regarded as a primer, and indeed there must be some question as to whether 'textbook' is the most appropriate title. It is, however, excellent value and mandatory reading for any modern clinical neurophysiologist. Presumably the sections on EEG and long latency evoked potentials would be of interest to psychiatrists.

CLARE J. FOWLER, *Consultant Clinical*
Neurophysiologist, The Middlesex Hospital and
St Bartholomew's Hospital, London

Community Clinical Psychology. Edited by HUGH C. H. KOCH. Beckenham: Croom Helm. 1986. 349 pp. £22.50.

This multi-author book has as its stated aim "To determine the main components of what clinical psychology has to offer within a community framework". It does not claim to be comprehensive overview, but covers a wide range of topics. There is an emphasis on "using psychologists' knowledge and skills that will help (other) staff". There is also an emphasis on the growing importance of prevention. The editor states that it is intended not only for those working with clients but also for managers, which is perhaps an interesting comment from a practitioner who became a manager shortly after completing this book.

The eighteen contributors all write from the basis of clinical practice and the majority from a British setting, with only four American contributions. Four of the chapters are concerned with particular lifestages ranging from childhood to old age, and emphasise those aspects of psychological practice the authors consider important. Two chapters are concerned with specific groups: the mentally handicapped and the chronically psychiatrically ill. Four chapters are concerned with very specific conditions: alcohol, drugs, smoking, and obesity. The chapter by Koch on anxiety and depression deals with management of these common conditions, not only in terms of therapy but also the location of services and the need for a preventative approach. The two remaining chapters deal with behavioural marital therapy and behavioural medicine.

This book provides a useful survey of the views of a number of active and experienced clinical psychologists and succeeds in providing an overview of much of the present scene. It inevitably raises questions as to the boundaries between the work of clinical psychologists and psychiatrists, as in places it seems to claim for clinical psychology that which, no doubt, many psychiatrists would see as falling within their own field. There is an emphasis on team work. This is a book that should be available to all psychiatrists and certainly should be included in every psychiatric and clinical psychological library. The print and format is not particularly attractive nor particularly easy to read, and it seems a pity that at this price it could not have been better. Alternatively, perhaps such books should be produced in paperback and therefore more likely to be purchased by individuals rather than mostly by libraries.

A. C. BROWN, *Consultant Senior Lecturer in Mental Health, University of Bristol*

Hypnogogia: The Unique State of Consciousness Between Wakefulness and Sleep. By ANDREAS MAVROMATIS. London: Routledge and Kegan Paul. 1987. 360 pp. £30.00.

This book has made a timely appearance. It examines hypnogogia at a time when there is a renewed interest in cognition and subjective experience within both psychiatry and psychology.

The first part of the book gives a comprehensive historical background to the phenomenon together with numerous descriptive accounts and pictorial illustrations. The second part relates hypnogogia to other states such as dreams, psychic experience, schizophrenia, and creativity. Finally, an attempt is made to determine its brain correlates and function.

According to the author, hypnogogia is a state, often initiated consciously and deliberately but frequently becoming automatic, in which original revelations of a psychic, artistic, or scientific nature may occur. It is usually pleasant and can be therapeutic.

"Hypnogogia" certainly makes fascinating reading to both the layman and those with some knowledge of the field. It also serves as an encouragement to the reader to initiate his own personal investigation of what is essentially a subjective experience.

ALYSON BOND, *Lecturer, Institute of Psychiatry, London*

Functional Psychological Testing: Principles and Instruments. Edited by RAYMOND B. CATTEL and RONALD C. JOHNSON. New York: Raven Press (distribution for Brunner/Mazel). 630 pp. $88.50.

The term 'functional' in the title of this book could mislead readers into expecting its contents to deal with the testing of practical, everyday functioning of human beings. In fact, the editors use the term in its most mathematical sense, the concern of the contributing authors being with exactness of measurement and principles of testing rather than with perhaps more fundamental questions concerning the ecology of testing. One of the editors states that functional psychological testing is 'based on quantitative personality theory', and readers who find this statement comfortable might find the book instructive and useful.

The book is in three sections: 'Psychometric principles in testing', 'Available structured tests for functional psychometry', and 'Art of testing in psychological practice'. These titles themselves are indicative of what I found to be the over-ambitious intentions of the editors, who have produced a book which is unnecessarily complicated. The section on psychometric principles is an example of intellectual overkill, which Anastasi (1982) avoids in her much more succinct coverage of the topic.

While I approve of Cattel's scorn for sloppy science and tests such as the Rorschach, I am not convinced that subjectivity does not enter the text of this book on a number of occasions. I was offended by the value judgements in chapters 8 and 20 on the ending of the Eleven Plus examination in Britain. On page 154, for example, the contributors write: "... in Britain, the labor (*sic*) government's abolition of selection by intelligence test for more advanced education ... was frightful." Statements of this kind – and there were others of similar ideology – should have been rooted out of this book by editors who pride themselves on their pursuit of objectivity.

I liked the historical accounts, particularly on industrial and vocational selection. There is a useful discussion on available attitude scales, and ideas on how to design such a scale. For me, the most interesting chapter was the one dealing with legal considerations for the psychologist. Overall, although I found the book useful on instruments, I found it almost unreadable on principles and kept wondering whether *my* education was at fault. I suspect that this American text will have a similar effect on other British readers who may not be familiar with quantitative personality theory.

BARBARA WILSON, *Senior Lecturer, University Department of Rehabilitation, Southampton*

Sexuality and Medicine. Vol. II: Ethical Viewpoints in Transition. Edited by EARL E. SHELP. Dordrecht, The Netherlands: Kluwer. 1987. 279 pp. Dfl. 130.00, $49.00, £38.95.

This is in the generally excellent *Philosophy and Medicine* series published by Reidel. It is the second of two volumes on aspects of sexuality; these can be read independently, and each is complete in itself.

This volume includes twelve essays by doctors, philosophers, and theologians whose concerns appear generally relevant to clinicians. I found three chapters particularly interesting. In 'An historical comment', John Duffy summarises the major changes in society's attitudes to sexuality since 1800, and notes how negative views which developed as 1900 approached have provided a legacy which remains. A century's presumed determinants and consequences of masturbation are a monument to medical, philosophical, and theological absurdity.

Chapters by R. Baker and N. Grose & E. Shelp include case material discussed in refreshing ways and cogently make the point that values and ethical questions are intrinsic to solving sexual problems. This is true for all helping activities, but is more pointedly so in the sexual field. Such issues should not be ignored, and this book is an interesting and cogent reminder of their importance.

J. P. WATSON, *Professor of Psychiatry, Guy's Hospital, London*

1987 Year Book of Psychiatry and Applied Mental Health. Edited by FREEDMAN, LOURIE, MELZER, NEMIAH, TALBOTT and WEINER. London: Wolfe Medical Publications. 1987. 499 pp. £36.50.

One of the most daunting tasks that faces clinicians is remaining in touch with advances in knowledge over an ever-widening area. The general psychiatrist may be aware of current thinking about the organic substrates of schizophrenia, on which this Year Book reviews many papers, but not perhaps equally aware of the confusing claims for the advantages of recent antidepressants, as well as, perhaps, objective measures of transference. This book is most valuable in addressing this problem, and gathers together summaries of papers from 97 journals.

Each of the 17 chapters is introduced by one of the editors, outlining the main directions of research and placing the articles usefully in a historical perspective. These introductions, and the editorial comments on papers, may be somewhat idiosyncratic and even dogmatic, but nevertheless are stimulating. For example, to state (page 456) that "violence may be as American as apple pie" without levels of probability is refreshing in a learned journal. A few readers will be irritated by the emphasis on American journals, but this, I think, has the advantage of drawing attention to problems which will spread rapidly across the Atlantic. The current American fashion in substance abuse, for example, on which there are a number of papers, is now becoming apparent in Great Britain. Equally, several papers on the plight of the chronic psychiatric patient who is homeless in an urban society should underline our need for caution in the closure of psychiatric facilities for chronic disorders. I found this book useful and easy to consult. It should be in all postgraduate libraries, where it will be useful to psychiatrists and trainees alike.

BERNARD ADAMS, *Consultant Psychiatrist, University College Hospital*

Insanity: the idea and its consequences. By THOMAS SZASZ. New York: John Wiley. 1986. 414 pp. $17.95.

Szasz first burst on the psychiatric scene as one of the stars of the anti-psychiatry movement in 1961 with the publication of *The Myth of Mental Illness*. Perhaps uncertain as to whether his message was clear, Szasz added a one-page summary of the main argument to the British paperback edition of 1972. This précis has always been useful, and students have frequently been referred to it. All praise to Szasz for brevity and clarity.

However, it would appear that Szasz has not been content to neglect the alternative strategy for selling his message: not brevity but prolixity, as book after book extolling the same basic theme has emerged in the past quarter century. The book under consideration here is no exception. "Insanity: the idea" might suggest something different – perhaps an historical look at how the word has changed its meaning and use over time; however, the nearest Szasz gets is to look the word up in a handbook of the history of ideas, and, finding it missing, to suspect another (psychiatric?) plot.

The first claim in Szasz's 1972 summary – and the basis of his work – is that illness can only occur in the body, not in the mind. The argument is rehearsed again here: diseases of the body occur in nature; diseases of the mind, because of their lack of biological correlates, only in metaphor. Szasz, as usual, never doubts that the difference between a physiological bodily process and a pathological bodily process might be other than written in tablets of stone. Because he fails to see that "pathological" as referring to the body is a social construction ("nature" has only variety, it is humans who evaluate), just as it is when referring to the mind, he is only half right. He is also half wrong, and this ultimately spoils a passionate analysis which has its moments of insight. In this case, as with the others, potential readers might first be directed to the yet-to-be beaten one-page summary.

DAVID ARMSTRONG, *Senior Lecturer, Unit of Sociology, United Medical and Dental Schools of Guy's and St Thomas's Hospitals*

Adolescence and Development Breakdown: A Psychoanalytic View. By MOSES LAUFER and M. EGLE LAUFER. New Haven and London: Yale University Press. 1984. 225 pp. £6.95.

This book has now been published in paperback form. It is the result of the authors' experience of treating seriously disturbed adolescents in psychoanalysis. With the help of illustrative clinical material, the authors describe the adolescent's developmental task in integrating sexuality and aggression. They examine the complex pathological solutions adopted when development goes awry, and discuss suitability for analytic treatment and management of acting-out during therapy.

The authors have many important things to say in this book that contribute greatly towards illuminating an area relatively neglected in the psychoanalytic literature. The style of the prose does not make for particularly easy reading, but despite this I am sure that all those who adopt a psychotherapeutic stance in their work with adolescents will find the book extremely helpful and thought-provoking. For them it should be considered to be required reading, and it should be classified as recommended reading for adult psychotherapists.

DENIS V. CARPY, *Consultant Psychotherapist, Adult Department, The Tavistock Clinic*

Helping Delinquents Change: A Treatment Manual of Social Learning Approaches. By JEROME S. STUMPHAUZER. New York: The Haworth Press. 1986. 213 pp. $24.95.

Stumphauzer is Clinical Professor of Psychiatry and the Behavioural Sciences at the University of Southern California School of Medicine. His career has focused on the behavioural treatment of delinquency, and he has published extensively on the subject. The book is not intended to be a highly academic textbook or research review; nevertheless, three key references are provided at the end of each chapter and there is, in addition, a considerable bibliography at the end of the book. It is the reports of follow-up studies that are in short supply, leaving one wondering whether the long-term effectiveness of the approaches used matches the self-evident enthusiasm of the author.

The intention of the author was to produce an easily-read, non-technical guide for practitioners such as counsellors, teachers, and probation officers working with delinquents and using a social learning approach. The book is very successful in meeting its aims, and is easy to read. There are four introductory chapters devoted to understanding delinquent behaviour and social learning theory; the remaining chapters describe particular techniques applicable in different settings, e.g. probation and community programmes, and occupational skills training.

Whether many psychiatrists, especially those working with psychologists who routinely use behavioural methods in their approach to assessment and treatment, would use this book I am uncertain. However, for those with a busy case-load, who work as consultants to "mediators in the natural environment", it could prove to be valuable.

CAROL SHELDRICK, *Consultant in Child and Adolescent Forensic Psychiatry, The Maudsley Hospital*

Caught in Crossfire: Children and the Northern Ireland Conflict. By ED CAIRNS. Belfast: The Appletree Press. 1987. 179 pp. £5.95 (pb), £11.95 (hb).

This is a book about the children of Northern Ireland and how they appear to have been affected by the social and political situation there and by the violence which has been a feature of life in that province in the last decade. A few studies of a clinical kind by psychiatrists and psychologists are mentioned, but the majority of the investigations referred to can best be described as falling into the categories of social psychology and 'scientific' sociology. It would be unfair to reveal what conclusions the author comes to concerning rates of psychiatric disorder, criminality, truancy, and educational handicaps of the children in Northern Ireland; suffice it to say that the book is a good source of this information. A graphic account of the cultural divide is provided, and some of the efforts being made to breach it are mentioned. This is a useful contribution to the study of young people exposed to circumstances of exceptional stress.

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